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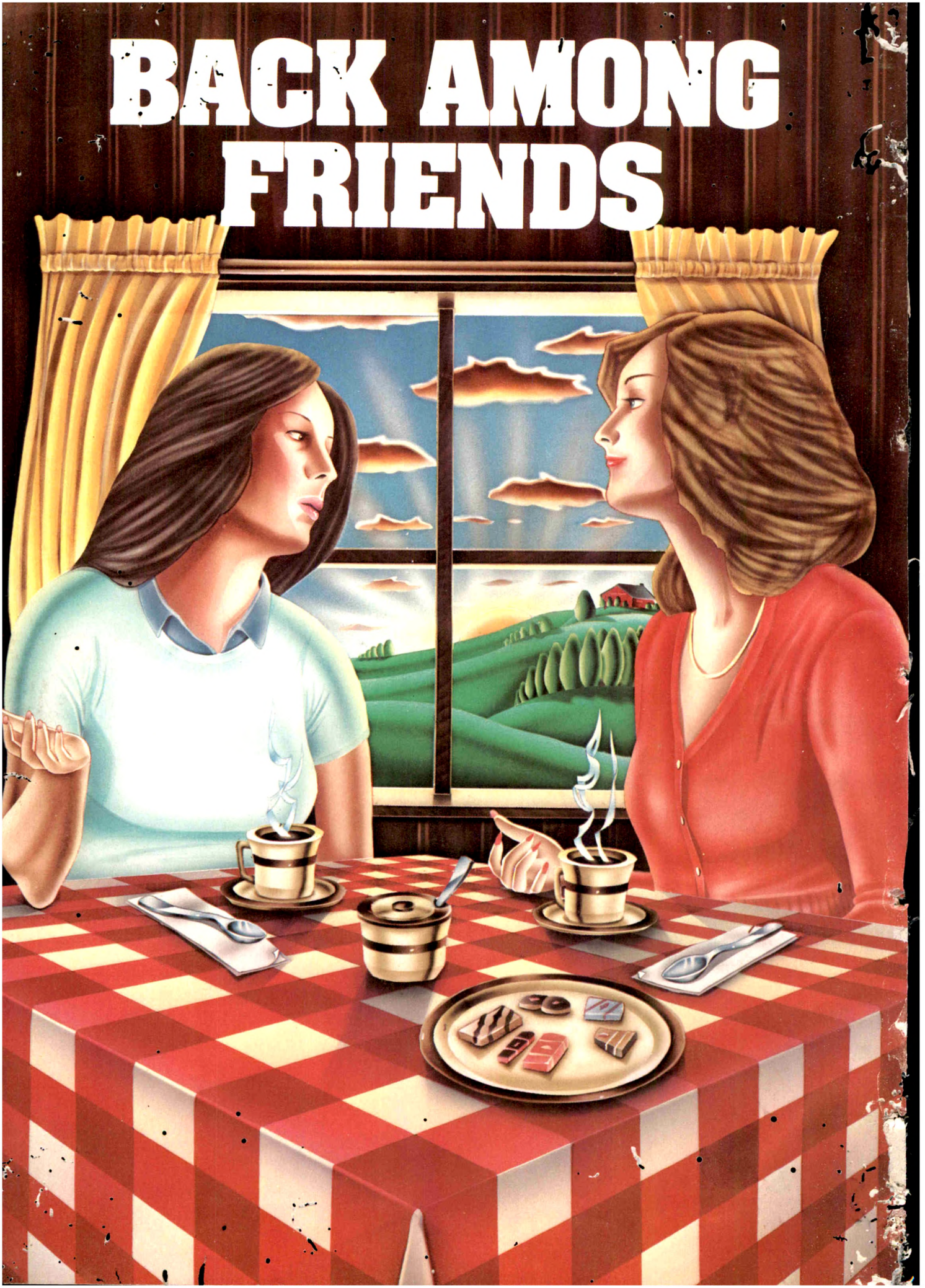
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Final classification of the less-than-effective indications requires further investigation.

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Use in pregnancy only when essential. There are reported instances of jaundice or prolonged extrapyramidal signs in newborn whose mothers had received chlorpromazine. Chlorpromazine is excreted in the breast milk of nursing mothers.

Precautions: Use cautiously in persons with cardiovascular, liver or chronic respiratory disease, or with acute respiratory infections. Patients with a history of hepatic encephalopathy due to cirrhosis have increased sensitivity to the C.N.S. effects of chlorpromazine. Due to cough reflex suppression, aspiration of vomitus is possible. May prolong or intensify the action of C.N.S. depressants, organophosphorus insecticides, heat, atropine and related drugs. (Reduce dosage of concomitant C.N.S. depressants.) Anticonvulsant action of barbiturates is not intensified. Special monitoring of adverse effects in mentally retarded patients may be necessary due to verbal deficits of these patients. High-dose and/or long-term therapy may reduce learning performance in retarded patients. Antiemetic effect may mask signs of toxic drug overdosage or obscure diagnosis of conditions such as intestinal obstruction, brain tumor, and Reye's syndrome (see Warnings). Discontinue high-dose, long-term therapy gradually. Patients on therapy for more than six months, especially high-dose therapy, should be evaluated periodically for possible adjustment or discontinuance of drug therapy. In severely disturbed retarded patients, periodic discontinuation should likewise be tried (once or twice a year) to assess the effects on behavioral problems. 'Thorazine' tablets contain FD&C Yellow #5 (tartrazine) which may cause allergic-type reactions (including bronchial asthma) in certain susceptible individuals. Although the overall incidence of FD&C Yellow #5 (tartrazine) sensitivity in the general population is low, it is frequently seen in patients who also have aspirin sensitivity. 'Thorazine' tablets are in the process of being reformulated to remove the FD&C Yellow #5. For specific information, contact Smith Kline &French Laboratories (outside Pa., call toll-free: 1-800-523-4835, ext. 4262; in Pa., call collect: 215-854-4262).







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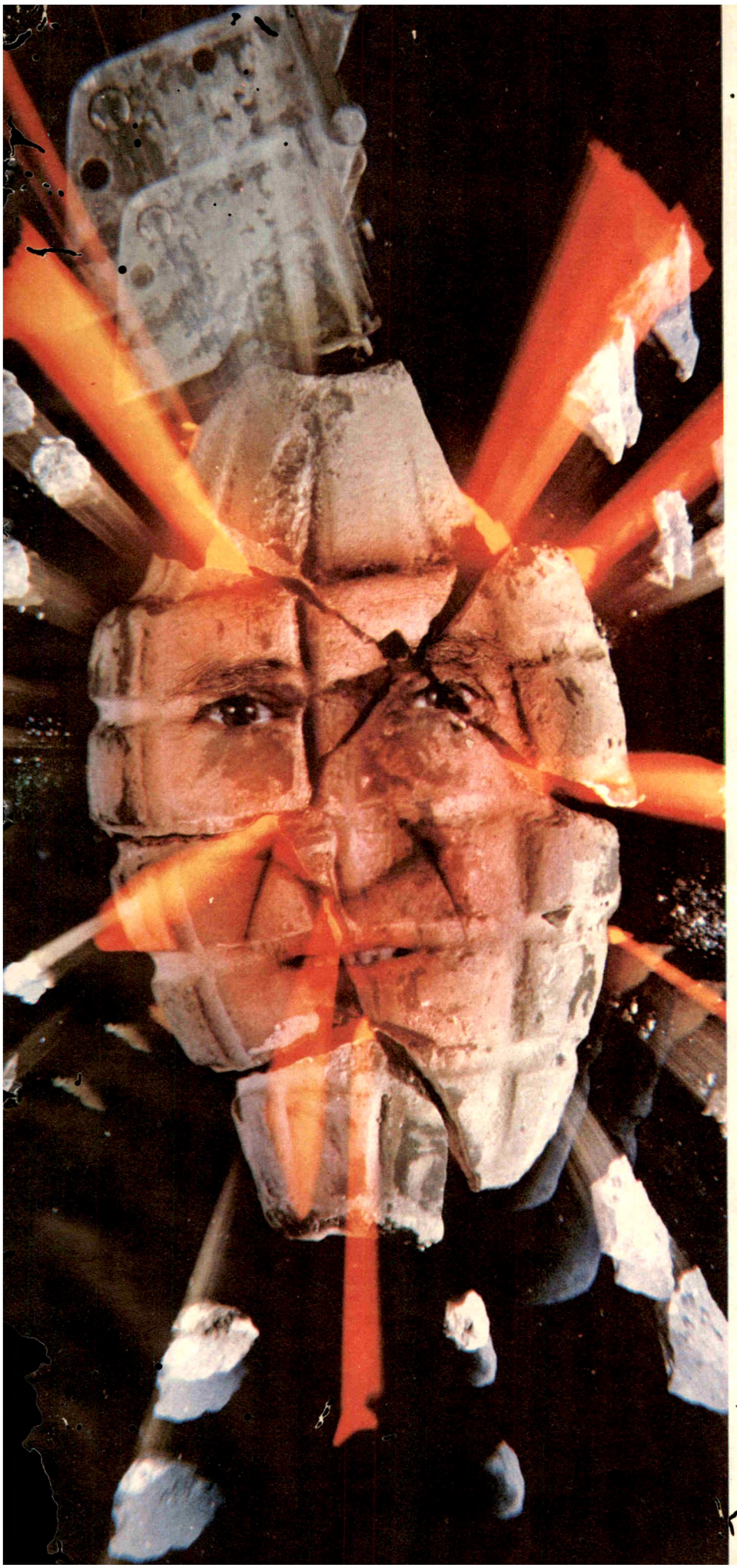
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Navane® (thiothixene)
Capsules: 1 mg, 2 mg, 5 mg, 10 mg, 20 mg
(thiothixene hydrochloride) Concentrate: 5 mg/ml,
Intramuscular: 2 mg/ml, 5 mg/ml

Contraindications. Navane (thiothixene) is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy*– Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration of Navane to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children– The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration– As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane (thiothixene). Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudo-parkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leukocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution; Navane For Injection– Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane (thiothixene) Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate– In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Stimulants that may cause convulsions (e.g. picrotoxin or pentylenetetrazol) should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg of thiothixene in bottles of 100, 1,000, and unit-dose pack of 100 (10 x 10's). Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100, 500, and unit-dose pack of 100 (10 x 10's).

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg, and in 30 ml (1 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, and 5 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10 vials. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

Navane (thiothixene hydrochloride) Intramuscular For Injection is available in amber glass vials in packages of 10 vials. When reconstituted with 2.2 ml of Sterile Water for Injection, each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene, and 59.6 mg of mannitol. The reconstituted solution of Navane Intramuscular For Injection may be stored for 48 hours at room temperature before discarding.

References: 1. Stotsky BA: Relative efficacy of parenteral haloperidol and thiothixene for the emergency treatment of acutely excited and agitated patients. *Dis Nerv Syst* 38:967-973, December 1977. 2. Brauzer B, Goldstein BJ: Comparative effects of intramuscular thiothixene and trifluoperazine in psychotic patients. *J Clin Pharmacol* 8:400-403, November-December 1968. 3. Gallant DM, Bishop MP, Bishop C, et al: Thiothixene: A controlled evaluation of the intramuscular antipsychotic preparation. *Curr Ther Res* 10:561-565, November 1968.

For additional information on Navane, please consult your Roerig representative or write to: Roerig Medical Department, 235 East 42nd Street, New York, NY 10017.

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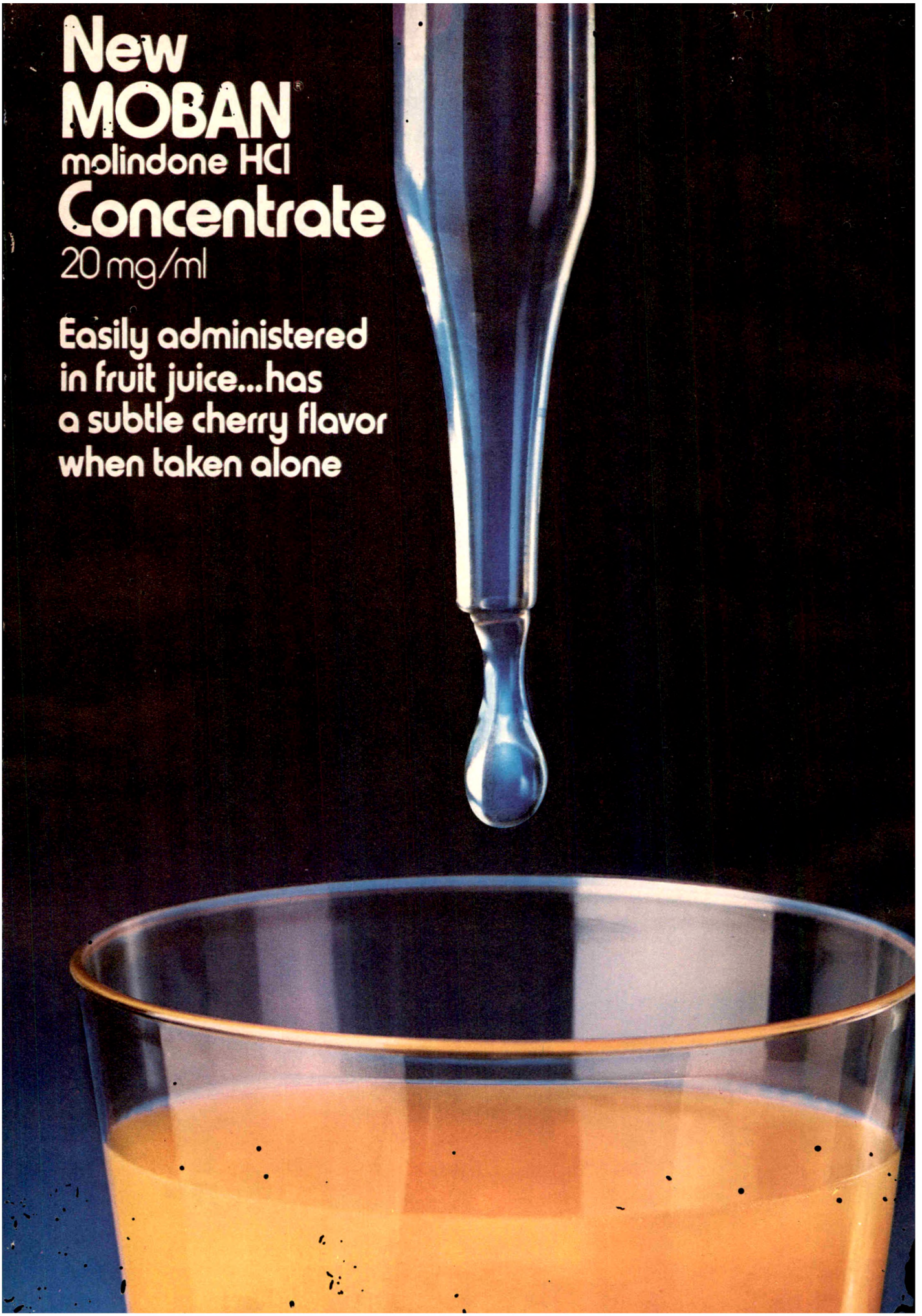
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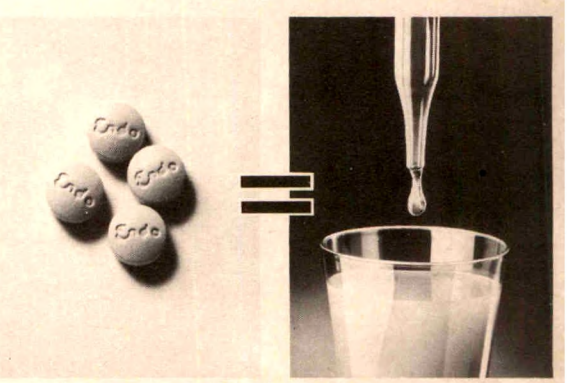
An effective antipsychotic agent with a favorable benefit/risk profile

NO incidence	excessive weight gain hepatotoxic changes ophthalmological changes persistent tardive dyskinesia skin pigmentation thyroid function changes tolerance development
LOW incidence	adverse drug interactions hematological changes hypotension nonpersistent symptoms of tardive dyskinesia sedation
MODERATE incidence	extrapyramidal reactions

This table compiled from clinical and field reports, according to the best available information.

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An effective antipsychotic agent

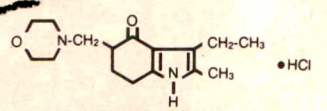
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continuing...Tablets 5 mg, 10 mg, 25 mg

DESCRIPTION MOBAN (molindone hydrochloride) is a dihydroindolone compound which is not structurally related to the phenothiazines, the butyrophenones or the thioxanthenes.

MOBAN is 3-ethyl-6, 7-dihydro-2-methyl-5-(morpholinomethyl) indol-4 (5H)-one hydrochloride. It is a white crystalline powder, freely soluble in water and alcohol and has a molecular weight of 312.8.



MOLINDONE HYDROCHLORIDE

ACTIONS MOBAN (molindone hydrochloride) has a pharmacological profile in laboratory animals which predominantly resembles that of major tranquilizers causing reduction of spontaneous locomotion and aggressiveness, suppression of a conditioned response and antagonism of the bizarre stereotyped behavior and hyperactivity induced by amphetamines. In addition, MOBAN antagonizes the depression caused by the tranquilizing agent tetrabenazine.

In human clinical studies tranquilization is achieved in the absence of muscle relaxing or incoordinating effects. Based on EEG studies, MOBAN exerts its effect on the ascending reticular activating system.

Human metabolic studies show MOBAN (molindone hydrochloride) to be rapidly absorbed and metabolized when given orally. Unmetabolized drug reached a peak blood level at 1.5 hours. Pharmacological effect from a single oral dose persists for 24-36 hours. There are 36 recognized metabolites with less than 2-3% unmetabolized MOBAN being excreted in urine and feces.

INDICATIONS MOBAN (molindone hydrochloride) is indicated in the management of the manifestations of schizophrenia.

CONTRAINDICATIONS MOBAN (molindone hydrochloride) is contraindicated in severe central nervous system depression (alcohol, barbiturates, narcotics, etc.) or comatose states, and in patients with known hypersensitivity to the drug.

WARNINGS Usage in Pregnancy: Studies in pregnant patients have not been carried out. Reproduction studies have been performed in the following animals:

Pregnant Rats oral dose— 20 mg/kg/day—10 days no adverse effect
40 mg/kg/day—10 days no adverse effect

Pregnant Mice oral dose— 20 mg/kg/day—10 days slight increase resorptions
40 mg/kg/day—10 days slight increase resorptions

Pregnant Rabbits oral dose— 5 mg/kg/day—12 days no adverse effect
10 mg/kg/day—12 days no adverse effect
20 mg/kg/day—12 days no adverse effect

Animal reproductive studies have not demonstrated a teratogenic potential. The anticipated benefits must be weighed against the unknown risks to the fetus if used in pregnant patients.

Nursing Mothers: Data are not available on the content of MOBAN (molindone hydrochloride) in the milk of nursing mothers.

Usage in Children: Use of MOBAN (molindone hydrochloride) in children below the age of twelve years is not recommended because safe and effective conditions for its usage have not been established.

MOBAN has not been shown effective in the management of behavioral complications in patients with mental retardation.

PRECAUTIONS Some patients receiving MOBAN (molindone hydrochloride) may note drowsiness initially and they should be advised against activities requiring mental alertness until their response to the drug has been established.

Increased activity has been noted in patients receiving MOBAN. Caution should be exercised where increased activity may be harmful.

MOBAN does not lower the seizure threshold in experimental animals to the degree noted with more sedating antipsychotic drugs. However, in humans convulsive seizures have been reported in a few instances.

EDO 854M580

The physician should be aware that this tablet preparation contains calcium sulfate as an excipient and that calcium ions may interfere with the absorption of preparations containing phenytoin sodium and tetracyclines.

MOBAN (molindone hydrochloride) has an antiemetic effect in animals. A similar effect may occur in humans and may obscure signs of intestinal obstruction or brain tumor.

ADVERSE REACTIONS—CNS EFFECTS The most frequently occurring effect is initial drowsiness that generally subsides with continued usage of the drug or lowering of the dose.

Noted less frequently were depression, hyperactivity and euphoria.

Neurological—Extrapyramidal Reactions Extrapyramidal reactions noted below may occur in susceptible individuals and are usually reversible with appropriate management.

Akathisia Motor restlessness may occur early.

Parkinson Syndrome Akinesia, characterized by rigidity, immobility and reduction of voluntary movements and tremor, have been observed. Occurrence is less frequent than akathisia.

Dystonic Syndrome Prolonged abnormal contractions of muscle groups occur infrequently. These symptoms may be managed by the addition of a synthetic antiparkinson agent (other than L-dopa), small doses of sedative drugs, and/or reduction in dosage.

Autonomic Nervous System Occasionally blurring of vision, tachycardia, nausea, dry mouth and salivation have been reported. Urinary retention and constipation may occur particularly if anticholinergic drugs are used to treat extrapyramidal symptoms.

Hematological There have been rare reports of leucopenia and leucocytosis. If such reactions occur, treatment with MOBAN may continue if clinical symptoms are absent. Alterations of blood glucose, liver function tests, B.U.N., and red blood cells have not been considered clinically significant.

Metabolic and Endocrine Effects Alteration of thyroid function has not been significant. Amenorrhea has been reported infrequently. Resumption of menses in previously amenorrheic women has been reported. Initially heavy menses may occur. Galactorrhea and gynecomastia have been reported infrequently. Increase in libido has been noted in some patients. Impotence has not been reported. Although both weight gain and weight loss have been in the direction of normal or ideal weight, excessive weight gain has not occurred with MOBAN.

Cardiovascular Rare, transient, non-specific T wave changes have been reported on E.K.G. Association with a clinical syndrome has not been established. Rarely has significant hypotension been reported.

Ophthalmological Lens opacities and pigmentary retinopathy have not been reported where patients have received MOBAN (molindone hydrochloride). In some patients, phenothiazine induced lenticular opacities have resolved following discontinuation of the phenothiazine while continuing therapy with MOBAN.

Skin Early, non-specific skin rash, probably of allergic origin, has occasionally been reported. Skin pigmentation has not been seen with MOBAN usage alone.

MOBAN (molindone hydrochloride) has certain pharmacological similarities to other antipsychotic agents. Because adverse reactions are often extensions of the pharmacological activity of a drug, all of the known pharmacological effects associated with other antipsychotic drugs should be kept in mind when MOBAN is used. Upon abrupt withdrawal after prolonged high dosage an abstinence syndrome has not been noted.

Tardive Dyskinesia Although rarely reported with MOBAN (molindone hydrochloride) symptoms were reversible upon discontinuation of therapy.

Tardive dyskinesia associated with other agents has appeared in some patients on long-term therapy and has also appeared after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). There may be involuntary movements of extremities.

There is no known effective treatment of tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time the syndrome may not develop.

DOSAGE AND ADMINISTRATION Initial and maintenance doses of MOBAN (molindone hydrochloride) should be individualized.

Initial Dosage Schedule The usual starting dosage is 50-75 mg/day.
- —Increase to 100 mg/day in 3 or 4 days.
- —Based on severity of symptomatology, dosage may be titrated up or down depending on individual patient response.
- —An increase to 225 mg/day may be required in patients with severe symptomatology.

Elderly and debilitated patients should be started on lower dosage.

Maintenance Dosage Schedule
1. Mild—5 mg-15 mg three or four times a day.
2. Moderate—10 mg-25 mg three or four times a day.
3. Severe—225 mg/day may be required.

DRUG INTERACTIONS Potentiation of drugs administered concurrently with MOBAN (molindone hydrochloride) has not been reported. Additionally, animal studies have not shown increased toxicity when MOBAN is given concurrently with representative members of three classes of drugs (i.e., barbiturates, chloral hydrate and antiparkinson drugs).

MANAGEMENT OF OVERDOSAGE Symptomatic, supportive therapy should be the rule.

Gastric lavage is indicated for the reduction of absorption of MOBAN (molindone hydrochloride) which is freely soluble in water.

Since the adsorption of MOBAN (molindone hydrochloride) by activated charcoal has not been determined, the use of this antidote must be considered of theoretical value.

Emesis in a comatose patient is contraindicated. Additionally, while the emetic effect of apomorphine is blocked by MOBAN in animals, this blocking effect has not been determined in humans.

A significant increase in the rate of removal of unmetabolized MOBAN (molindone hydrochloride) from the body by forced diuresis, peritoneal or renal dialysis would not be expected. (Only 2% of a single ingested dose of MOBAN is excreted unmetabolized in the urine.)

However, poor response of the patient may justify use of these procedures. While the use of laxatives or enemas might be based on general principles, the amount of unmetabolized MOBAN in feces is less than 1%. Extrapyramidal symptoms have responded to the use of diphenhydramine (Benadryl*) and the synthetic anticholinergic antiparkinson agents, (i.e., Artane*, Cogentin*, Akineton*).

HOW SUPPLIED As tablets in bottles with potencies and colors as follows:

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25 mg light green

As a concentrate containing 20 mg molindone hydrochloride per ml in 4 oz. (120ml) bottles.

*Benadryl—Trademark, Parke Davis and Co.
*Artane—Trademark, Lederle Laboratories
*Cogentin—Trademark, Merck Sharp & Dohme
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Rev. Nov. 1979 6102-5

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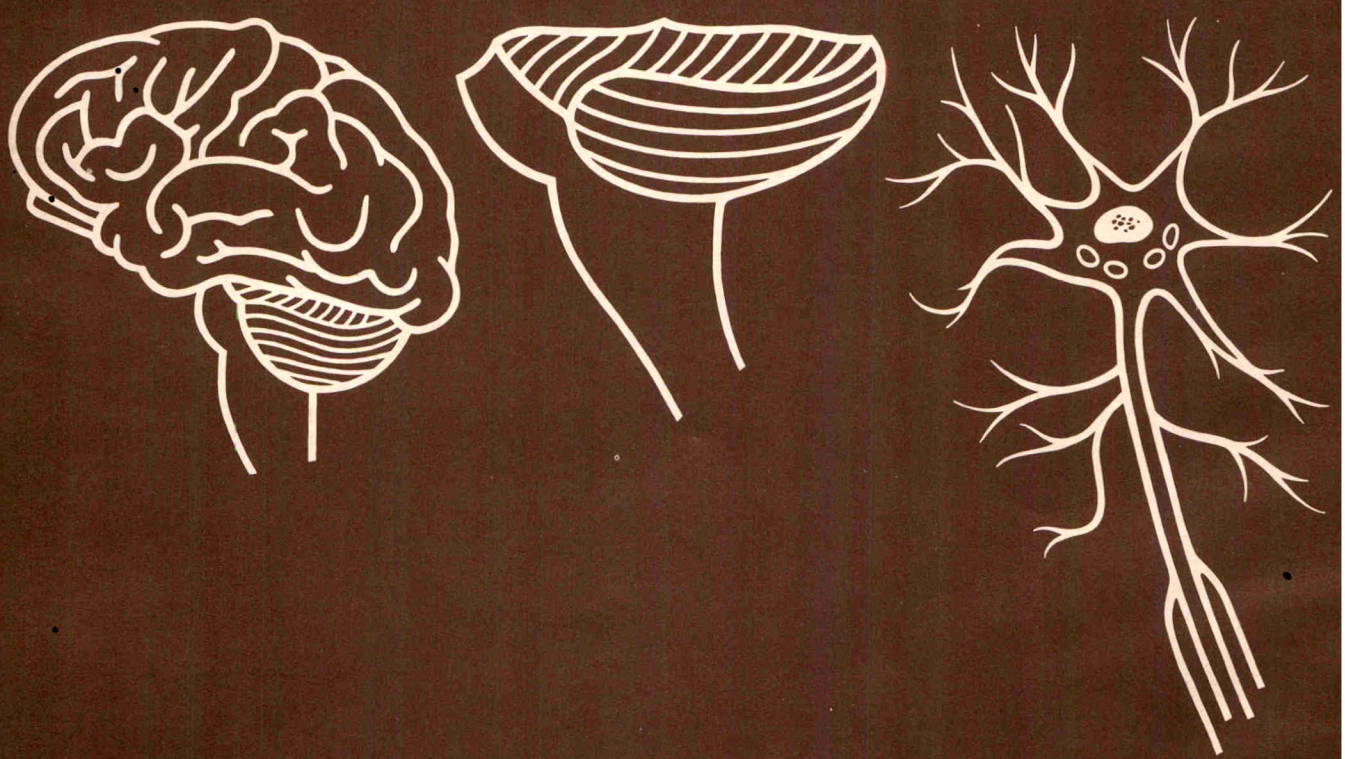
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AJPsych/7/80

A legacy of evolution



Increasing evidence links anxiety to phylogenetically ancient brain structures.[1]

Catecholamine-rich locus coeruleus has been proposed as one of a number of anatomic structures intricately involved in anxiety.[2]

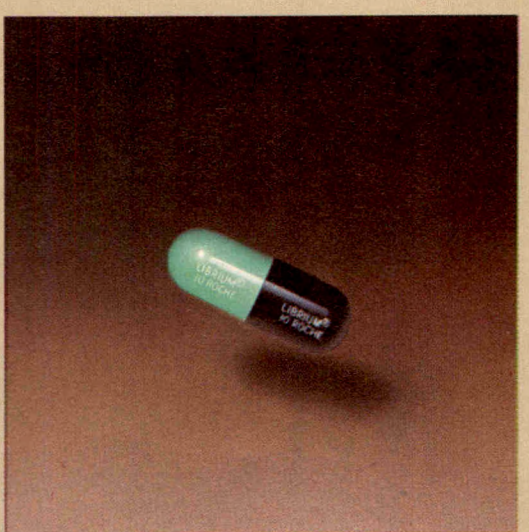
Specific brain binding sites for benzodiazepines appear to be evolutionary legacy shared by most vertebrate species studied.[3]

1. Betz BJ: *Am J Psychiatry 136*:1251-1256, Oct 1979

2. Redmond DE, Huang YH: *Life Sciences 25*:2149-2162, Dec 24, 1979

3. Bunney WE, Jr, Tallman J: *New Biological Research Relevant to Anxiety*, Presented at the meeting of the International Committee on Pharmacopsychiatry, Aug 29, 1979

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Indications: Relief of anxiety and tension occurring alone or accompanying various disease states. Efficacy beyond four months not established by systematic clinical studies. Periodic reassessment of therapy recommended.

Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Warn patients that mental and/or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

Usual Daily Dosage: Individualize for maximum beneficial effects. *Oral–Adults:* Mild and moderate anxiety and tension, 5 or 10 mg *t.i.d.* or *q.i.d.*; severe states, 20 or 25 mg *t.i.d.* or *q.i.d. Geriatric patients:* 5 mg *b.i.d.* to *q.i.d.* (See Precautions.)

Supplied: Librium® (chlordiazepoxide HCl) *Capsules*, 5 mg, 10 mg and 25 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available singly and in trays of 10. Libritabs® (chlordiazepoxide) *Tablets*, 5 mg, 10 mg and 25 mg—bottles of 100 and 500. With respect to clinical activity, capsules and tablets are indistinguishable.

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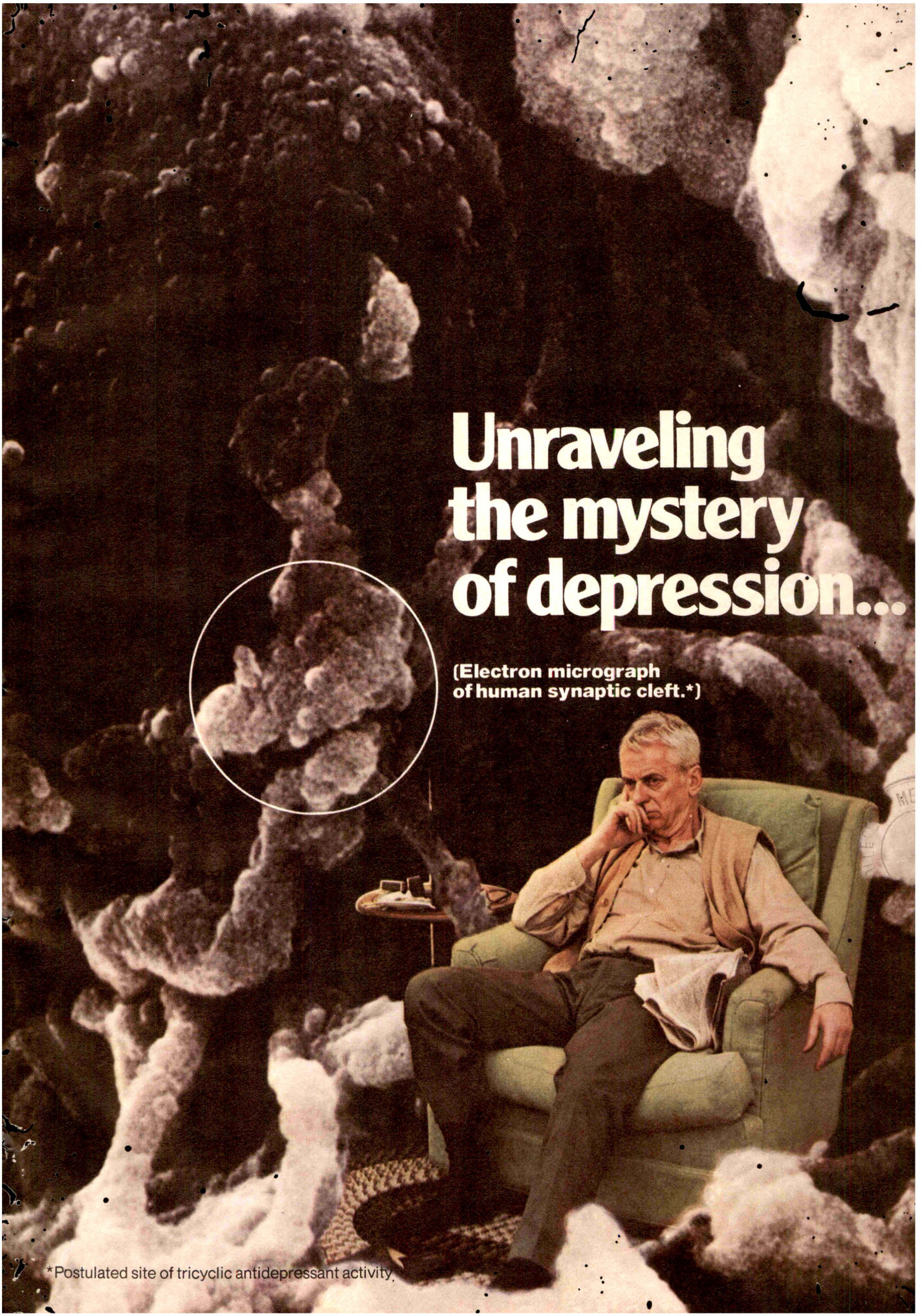
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Unraveling
the mystery
of depression...
(Electron micrograph
of human synaptic cleft.*)
*Postulated site of tricyclic antidepressant activity

Current evidence suggests that many depressions have a biochemical basis in the form of a relative deficiency of neurotransmitters, such as norepinephrine and serotonin.

Deficiency of norepinephrine is postulated to be a major factor in a number of depressions. Laboratory studies have shown that desipramine is a potent inhibitor of norepinephrine re-uptake,[1] a mechanism of action that is thought to increase norepinephrine at the synaptic cleft when norepinephrine deficiency exists. (See Figures 1 and 2.)

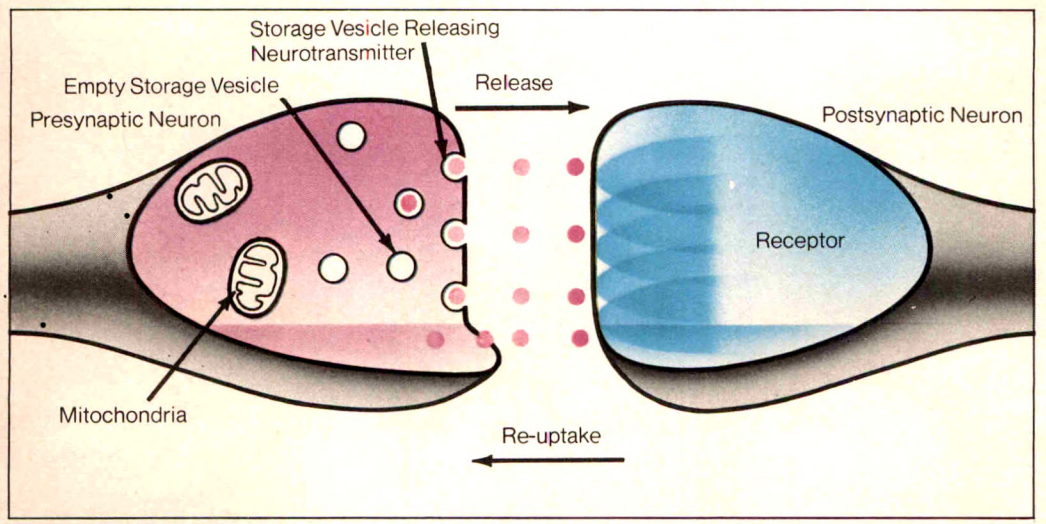


Figure 1. Diagrammatic representation of the *normal* process of neurotransmitter release, transfer across the synaptic cleft to the receptor site, and re-uptake. The supply of neurotransmitters is adequate in the normal state.

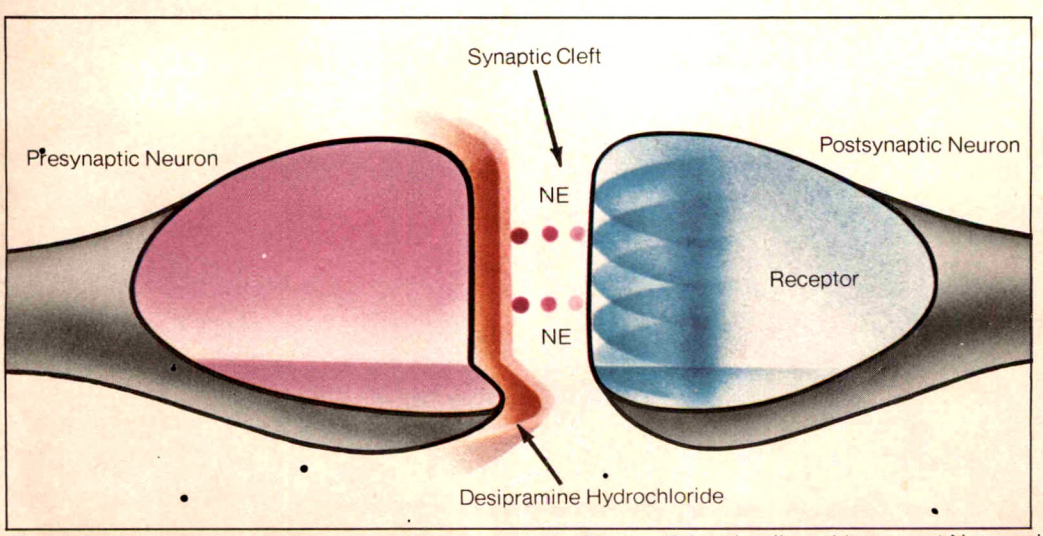


Figure 2. Diagrammatic representation of the mechanism of action of the tricyclic antidepressant Norpramin when there is a relative deficiency of the neurotransmitter norepinephrine (NE), as seen in some depressive states. Norpramin (desipramine hydrochloride) blocks the re-uptake of norepinephrine at the presynaptic nerve terminal (light brown band), ensuring increased cleft and receptor site neurotransmitter concentration.

Norpramin®
[desipramine hydrochloride tablets NF] 25,50,75,100,150 mg.

Potent blocker of norepinephrine re-uptake

According to current theory,[2,3] some depressions may respond to desipramine when other tricyclic antidepressants that block the re-uptake of a different neurotransmitter have been ineffective. Therefore, if your patient is unresponsive to treatment with one tricyclic, consider switching to a tricyclic that blocks the re-uptake of a different neurotransmitter.

Helps patient remain calm but active

Norpramin does not usually inhibit normal activity, although patients should be cautioned against driving or operating machinery if drowsiness occurs. (See Warnings, Precautions, and Adverse Reactions.)

Begins to improve sleep patterns within one week in some patients[4]

As the depression is relieved, difficulty in falling asleep, restlessness, and early morning awakening diminish.

Less anticholinergic activity

(See Warnings.)

Studies in the laboratory[5] and in normal human subjects[6,7] have shown that desipramine, with its single bond side chain, has less anticholinergic activity than amitriptyline or doxepin. This may mean:

- less dry mouth
- less blurred vision
- less urinary retention

Merrell

P2A, 356

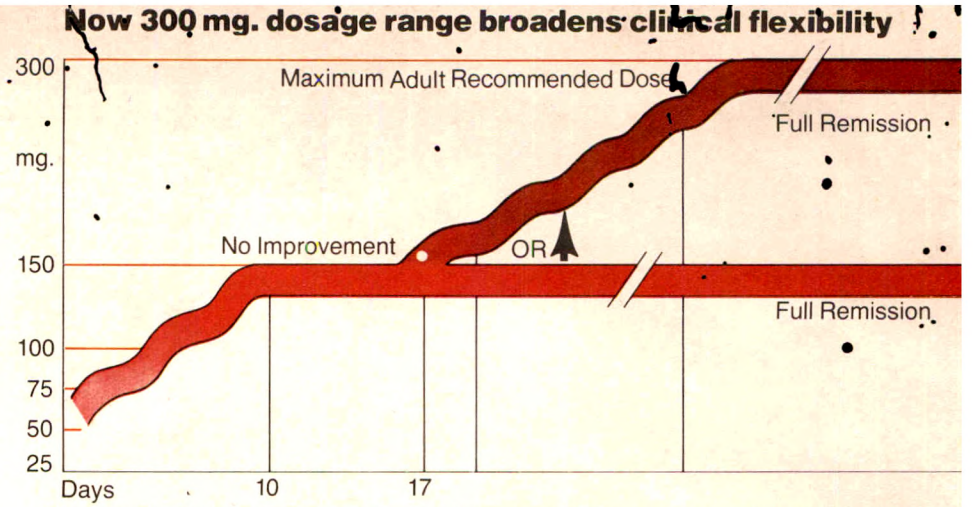
References: 1. Ross, S.B. and Renyi, A.L.: Tricyclic antidepressant agents. I. Comparison of the inhibition of the uptake of ^{3}H-noradrenaline and ^{14}C-5-hydroxytryptamine in slices and crude synaptosome preparations of the midbrain-hypothalamus region of the rat brain. Acta Pharmacol. (Kobenhavn) 36:382-394, 1975. 2. Maas, J.W.: Biogenic amines and depression. Biochemical and pharmacological separation of two types of depression. Arch. Gen. Psychiat. 32:1357-1361, 1975. 3. Davis, J.M.: Central biogenic amines and theories of depression and mania. In, Phenomenology and Treatment of Depression, W.E. Fann, I. Karacan, A.D. Pokorny, and R.L. Williams, Ed. New York, Spectrum Publications, Inc., 1977, pp. 17-32. 4. Zung, W.W.K.: The pharmacology of disordered sleep. A laboratory approach. J.A.M.A. 211:1532-1534, 1970. 5. Snyder, S.H. and Yamamura, H.I.: Antidepressants and the muscarinic acetylcholine receptor. Arch. Gen. Psychiat. 34:236-239, 1977. 6. Blackwell, B., Stefopoulos, A., Enders, P., Kuzma, R., and Adolphe, A.: Anticholinergic activity of two tricyclic antidepressants. Amer. J. Psychiat. 135:722-724, 1978. 7. Peterson, G.R., Blackwell, B., Hostetler, R.M., Kuzma, R., and Adolphe, A.B.: Anticholinergic activity of the tricyclic antidepressants despiramine [*sic*] and doxepin in nondepressed volunteers. Communic. Psychopharmacol. 2:145-150, 1978.

See Following Page for Brief Summary of Prescribing Information.

Norpramin®
(desipramine hydrochloride tablets NF)

AVAILABLE ONLY ON PRESCRIPTION

Brief Summary



A maximum dosage of 300 mg. a day, as a single or divided dose, may now be prescribed for Norpramin. Since inadequate dosage is thought to be one of the major causes of treatment failure, the new therapeutic range should prove clinically helpful. Doses above 150 mg./day are not recommended for geriatric and adolescent patients.

MECHANISM OF ACTION: Available evidence suggests that many depressions have a biochemical basis in the form of a relative deficiency of neurotransmitters such as norepinephrine and serotonin. Norepinephrine deficiency may be associated with relatively low urinary 3-methoxy-4-hydroxyphenyl glycol (MHPG) levels, while serotonin deficiencies may be associated with low spinal fluid levels of 5-hydroxyindolacetic acid.

While the precise mechanism of action of the tricyclic antidepressants is unknown, a leading theory suggests that they restore normal levels of neurotransmitters by blocking the re-uptake of these substances from the synapse in the central nervous system.

Evidence indicates that the secondary amine tricyclic antidepressants, including Norpramin, may have greater activity in blocking the re-uptake of norepinephrine. Tertiary amine tricyclic antidepressants, such as amitriptyline, may have greater effect on serotonin re-uptake.

Norpramin (desipramine hydrochloride) is not a monoamine oxidase (MAO) inhibitor and does not act primarily as a central nervous system stimulant. It has been found in some studies to have a more rapid onset of action than imipramine. Earliest therapeutic effects may occasionally be seen in 2 to 5 days, but full treatment benefit usually requires 2 to 3 weeks to obtain.

INDICATIONS: Norpramin (desipramine hydrochloride) is indicated for relief of symptoms in various depressive syndromes, especially endogenous depression.

CONTRAINDICATIONS: Desipramine hydrochloride should not be given in conjunction with, or within 2 weeks of, treatment with an MAO inhibitor drug; hyperpyretic crises, severe convulsions, and death have occurred in patients taking MAO inhibitors and tricyclic antidepressants. When Norpramin (desipramine hydrochloride) is substituted for an MAO inhibitor, at least 2 weeks should elapse between treatments. Norpramin should then be started cautiously and should be increased gradually.

The drug is contraindicated in the acute recovery period following myocardial infarction. It should not be used in those who have shown prior hypersensitivity to the drug. Cross sensitivity between this and other dibenzazepines is a possibility.

WARNINGS: 1. Extreme caution should be used when this drug is given in the following situations: a. In patients with cardiovascular disease, because of the possibility of conduction defects, arrhythmias, tachycardias, strokes, and acute myocardial infarction. b. In patients with a history of urinary retention or glaucoma, because of the anticholinergic properties of the drug. c. In patients with thyroid disease or those taking thyroid medication, because of the possibility of cardiovascular toxicity, including arrhythmias. d. In patients with a history of seizure disorder, because this drug has been shown to lower the seizure threshold. **2.** This drug is capable of blocking the antihypertensive effect of guanethidine and similarly acting compounds. **3.** USE IN PREGNANCY: Safe use of desipramine hydrochloride during pregnancy and lactation has not been established; therefore, if it is to be given to pregnant patients, nursing mothers, or women of childbearing potential, the possible benefits must be weighed against the possible hazards to mother and child. Animal reproductive studies have been inconclusive. **4.** USE IN CHILDREN: Norpramin (desipramine hydrochloride) is not recommended for use in children since safety and effectiveness in the pediatric age group have not been established. **5.** The patient should be cautioned that this drug may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery. **6.** In patients who may use alcohol excessively, it should be borne in mind that the potentiation may increase the danger inherent in any suicide attempt or overdosage.

PRECAUTIONS: 1. It is important that this drug be dispensed in the least possible quantities to depressed outpatients, since suicide has been accomplished with this class of drug. Ordinary prudence requires that children not have access to this drug or to potent drugs of any kind; if possible this drug should be dispensed in containers with child-resistant safety closures. Storage of this drug in the home must be supervised responsibly. **2.** If serious adverse effects occur, dosage should be reduced or treatment should be altered. **3.** Norpramin (desipramine hydrochloride) therapy in patients with manic-depressive illness may induce a hypomanic state after the depressive phase terminates. **4.** The drug may cause exacerbation of psychosis in schizophrenic patients. **5.** Close supervision and careful adjustment of dosage are required when this drug is given concomitantly with anticholinergic or sympathomimetic drugs. **6.** Patients should be warned that while taking this drug their response to alcoholic beverages may be exaggerated. **7.** Clinical experience in the concurrent administration of ECT and antidepressant drugs is limited. Thus, if such treatment is essential, the possibility of increased risk relative to benefits should be considered. **8.** The sedative effects of Norpramin and benzodiazepines (*e.g.*, chlordiazepoxide or diazepam) are additive. Both the sedative and anticholinergic effects of the major tranquilizers are additive to those of Norpramin. **9.** This drug should be discontinued as soon as possible prior to elective surgery because of the possible cardiovascular effects. Hypertensive episodes have been observed during surgery in patients taking desipramine hydrochloride. **10.** Both elevation and lowering of blood sugar levels have been reported. **11.** Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathologic neutrophil depression. **12.** Norpramin 25, 50, 75, and 100 mg. tablets contain FD&C Yellow No. 5 (tartrazine), which may cause allergic-type reactions (including bronchial asthma) in certain susceptible individuals. Although the overall incidence of FD&C Yellow No. 5 (tartrazine) sensitivity in the general population is low, it is frequently seen in patients who also have aspirin hypersensitivity.

ADVERSE REACTIONS: Note: Included in the following listing are a few adverse reactions that have not been reported with this specific drug. However, the pharmacologic similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when Norpramin (desipramine hydrochloride) is given.

Cardiovascular: hypotension, hypertension, tachycardia, palpitation, arrhythmias, heart block, myocardial infarction, stroke.

Psychiatric: confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurologic: numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures; alteration in EEG patterns; tinnitus.

Anticholinergic: dry mouth, and rarely associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis, increased intraocular pressure; constipation, paralytic ileus; urinary retention, delayed micturition, dilatation of urinary tract.

Allergic: skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight), edema (of face and tongue or general), drug fever, cross sensitivity with other tricyclic drugs.

Hematologic: bone marrow depressions including agranulocytosis, eosinophilia, purpura, thrombocytopenia.

Gastrointestinal: anorexia, nausea and vomiting, epigastric distress, peculiar taste, abdominal cramps, diarrhea, stomatitis, black tongue.

Endocrine: gynecomastia in the male, breast enlargement and galactorrhea in the female; increased or decreased libido, impotence, testicular swelling; elevation or depression of blood sugar levels.

Other: jaundice (simulating obstructive), altered liver function; weight gain or loss; perspiration, flushing; urinary frequency, nocturia; parotid swelling; drowsiness, dizziness, weakness and fatigue, headache; alopecia.

Withdrawal Symptoms: Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache, and malaise.

DOSAGE AND ADMINISTRATION: Not recommended for use in children. Lower dosages are recommended for elderly patients and adolescents. Lower dosages are also recommended for outpatients compared to hospitalized patients, who are closely supervised. Dosage should be initiated at a low level and increased according to clinical response and any evidence of intolerance. Following remission, maintenance medication may be required for a period of time and should be at the lowest dose that will maintain remission.

Usual Adult Dose: The usual adult dose is 100 to 200 mg. per day. In more severely ill patients, dosage may be further increased gradually to 300 mg./day if necessary. Dosages above 300 mg./day are not recommended.

Dosage should be initiated at a lower level and increased according to tolerance and clinical response.

Treatment of patients requiring as much as 300 mg. should generally be initiated in hospitals, where regular visits by the physician, skilled nursing care, and frequent electrocardiograms (ECG's) are available.

The best available evidence of impending toxicity from very high doses of Norpramin is prolongation of the QRS or QT intervals on the ECG. Prolongation of the PR interval is also significant, but less closely correlated with plasma levels. Clinical symptoms of intolerance, especially drowsiness, dizziness, and postural hypotension, should also alert the physician to the need for reduction in dosage. Plasma desipramine measurement would constitute the optimal guide to dosage monitoring.

Initial therapy may be administered in divided doses or a single daily dose.

Maintenance therapy may be given on a once-daily schedule for patient convenience and compliance.

Adolescent and Geriatric Dose: The usual adolescent and geriatric dose is 25 to 100 mg. daily.

Dosage should be initiated at a lower level and increased according to tolerance and clinical response to a usual maximum of 100 mg. daily. In more severely ill patients, dosage may be further increased to 150 mg./day. Doses above 150 mg./day are not recommended in these age groups.

Initial therapy may be administered in divided doses or a single daily dose.

Maintenance therapy may be given on a once-daily schedule for patient convenience and compliance.

OVERDOSAGE: See prescribing information for a discussion of symptoms and treatment of overdose.

Product Information as of July, 1979

Merrell

MERRELL-NATIONAL LABORATORIES
Division of Richardson-Merrell Inc.
Cincinnati, Ohio 45215, U.S.A.

MNQ-211 0-5934 (Y635B)

ROCHE

MEDICAL DIRECTOR'S PAGE

Bruce H. Medd, M.D., Director of Professional Services



Protecting your Prescriptions

Recently, physicians—and, particularly, those new in private practice—are being approached by persons exhibiting varying extremes of drug-seeking behavior. Identifying these patients can become a problem; however, there are several clues which can alert you to the possibility that a patient may be a drug misuser/abuser:

- when an unfamiliar patient tells you that a controlled product he wants from you was previously prescribed for him by another physician (either in private practice or in a hospital) who is currently unavailable
- when an unfamiliar patient states that he is the patient of another physician for whom you may occasionally cover and wishes a prescription for a controlled product renewed

When either of these situations occurs, it is a good idea to ask the patient for the name of the physician or hospital and attempt to verify the story. If the patient tends to avoid answering your questions, it generally indicates a problem. Often with this type of patient, your prescription blank is the prime target. The following are some suggested guidelines to follow to insure its safety and proper use.

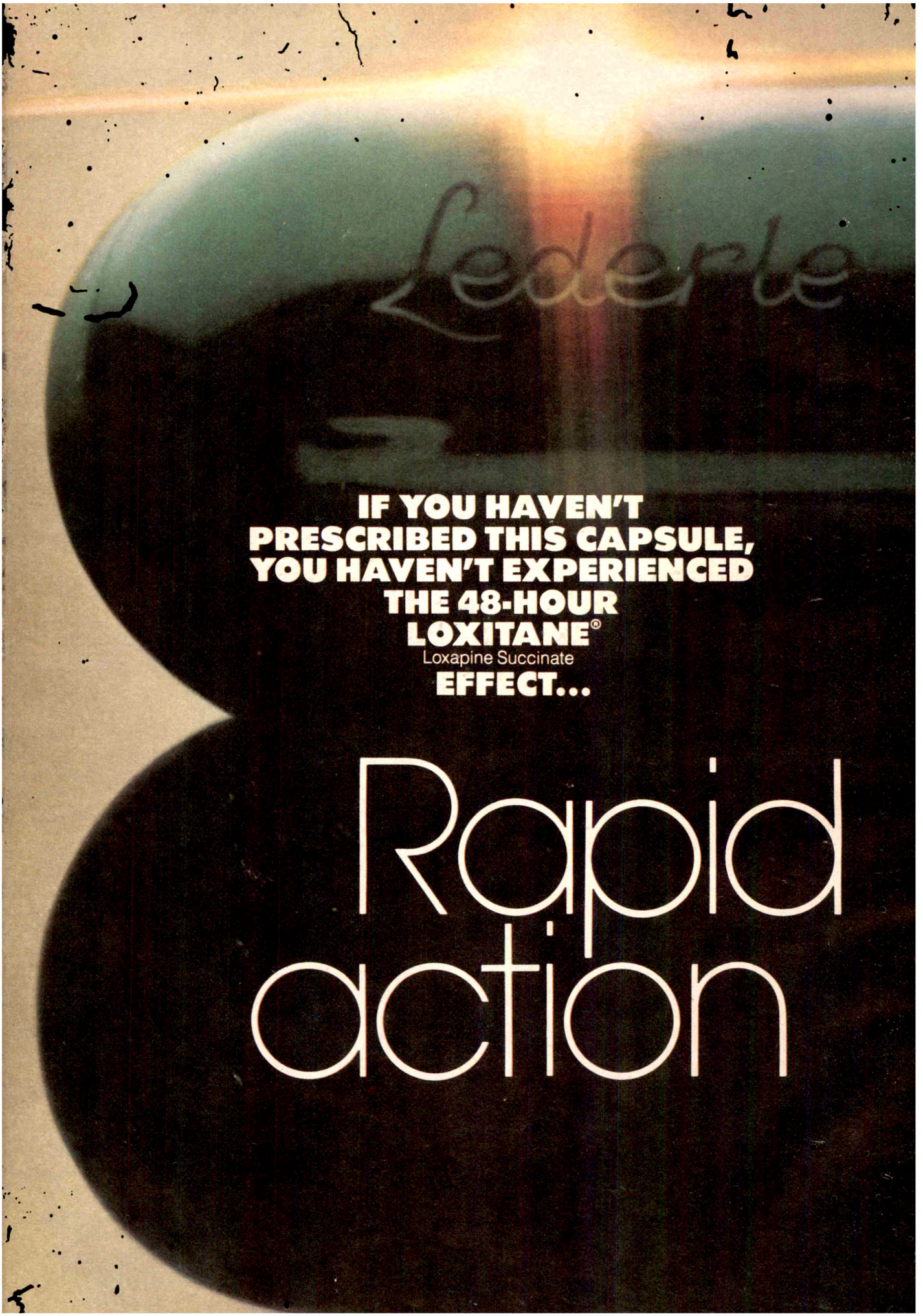
CUT OUT AND SAVE

The Well-Protected Prescription

GUIDELINES

1. Store all unused prescription pads in a safe place where they cannot be easily stolen
2. Minimize the number of pads in use at one time
3. Have prescription blanks numbered consecutively when printed so that you can tell if some sheets are missing
4. Never sign prescription blanks in advance
5. Write prescriptions in ink or indelible pencil to prevent changes
6. Write out the actual amount of medication prescribed in addition to using an Arabic or Roman numeral—this discourages alterations
7. Do not use your prescription blanks for writing notes or memos which can be erased and the blanks used again
8. Do not leave prescription pads in unattended examining rooms, office areas or in your bag or car where they can be easily picked up

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Division of Hoffmann-La Roche Inc.
Nutley, New Jersey 07110

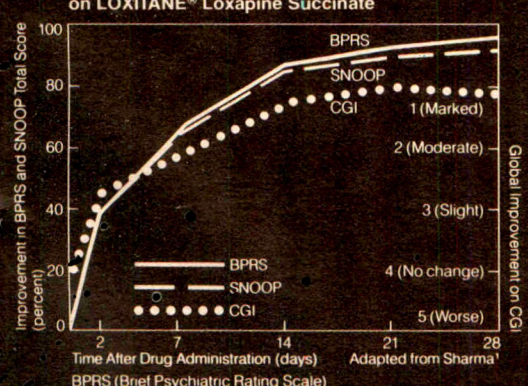

Lederle
IF YOU HAVEN'T
PRESCRIBED THIS CAPSULE,
YOU HAVEN'T EXPERIENCED
THE 48-HOUR
LOXITANE®
Loxapine Succinate
EFFECT...
Rapid
action

25mg.

Impressive improvement in the often critical first two days In a series of clinical studies[1-4] involving hospitalized and ambulatory schizophrenics, there was a particularly impressive decrease in key target symptoms such as conceptual disorganization, thinking disorder, hallucinatory behavior, and anxiety-depression during the first two days. Particularly notable was an almost 40% mean symptomatic improvement in BPRS reported in 18 acute schizophrenic patients after 48 hours of treatment.[1] (See chart)

Steady progress facilitates earlier resocialization Throughout the early weeks of therapy, the LOXITANE difference continues to be apparent. In clinical trials, near maximal control has been reported[1-4] by week four and behavior patterns often approached normal. Mean overall symptom improvement of almost 96% in BPRS has been observed within 28 days.[1] Prompt therapeutic response permits approximate psychotherapy for a rapid return to more normal activities.

Symptom improvement in 18 acute schizophrenics on LOXITANE® Loxapine Succinate



BPRS (Brief Psychiatric Rating Scale)
CGI (Clinical Global Impressions)

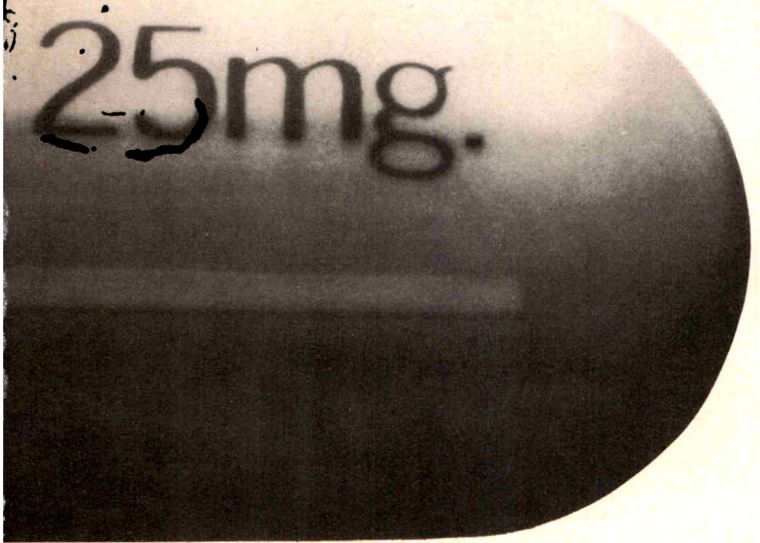
Loxitane®
LOXAPINE SUCCINATE
Lederle

5 mg, 10 mg, 25 mg, 50 mg capsules

See LOXITANE Brief Summary for indications, warnings and precautions and for more detailed information concerning side effects.

For the difference that begins with rapid action...

Loxitane®
LOXAPINE SUCCINATE
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Recommended Daily Dosage

Dosage	Initially: 20-50 mg During first 24 hours: up to 100 mg Usual therapeutic level: 150 mg/day Maintenance: 60-100 mg/day Maximum: 250 mg/day Dosage may be divided as necessary. Reduced dosage in geriatrics and adolescents should be considered.

Brief Summary
LOXITANE® Loxapine Succinate Capsules
LOXITANE® C Loxapine Hydrochloride Oral Concentrate
INDICATIONS: Manifestations of schizophrenia.
CONTRAINDICATIONS: Comatose or severely depressed states; hypersensitivity to the drug
WARNINGS: Safe use during pregnancy or lactation has not been established; weigh potential benefits to possible hazards. Not recommended for use in children under 16. May impair mental and/or physical abilities especially during early therapy; warn ambulatory patients about activities requiring alertness and concomitant use of alcohol or other CNS depressants. Not recommended for management of behavioral complications in mentally retarded patients.
PRECAUTIONS: Use with extreme caution in patients with a history of convulsive disorders; use with caution in patients with cardiovascular disease or in those with glaucoma or a tendency to urinary retention particularly when on concomitant anticholinergic medication. Loxapine has an antiemetic effect in animals which might occur in man masking signs of overdosage of toxic drugs and obscuring intestinal obstruction or brain tumor. Since possible ocular toxicity cannot be excluded, observe carefully for pigmentary retinopathy and lenticular pigmentation.
ADVERSE REACTIONS: *CNS effects*, other than extrapyramidal, infrequent. Mild drowsiness may occur at beginning of therapy or upon dosage increase, usually subsides with continued therapy. Sedation, dizziness, faintness, staggering gait, muscle twitching, weakness and confusional states have been reported. Extrapyramidal reactions often occur early in treatment manifested by Parkinson-like symptoms (tremor, rigidity, excessive salivation, masked facies, akathisia) controllable by dosage reduction or antiparkinson drugs at usual dosages. Dystonic and dyskinetic reactions, while less frequently occurring, may be more severe, requiring dosage reduction or temporary withdrawal plus appropriate counteractive drugs. *Persistent Tardive Dyskinesia* may appear during prolonged therapy or following discontinuance, the risk greater in the elderly, especially females, on high dosage. Symptoms, persistent and in some patients apparently irreversible, are characterized by rhythmical involuntary movement of the tongue, face, mouth and jaw sometimes accompanied by involuntary movement of extremities. Since there is no known effective treatment, discontinue all antipsychotic drugs if symptoms appear. Reinstitution of treatment, increased dosage, or switching to another agent may mask syndrome. The syndrome may not develop if medication is stopped when fine vermicular movements of the tongue first appear. *Cardiovascular Effects:* Tachycardia, hypotension, hypertension, lightheadedness and syncope. ECG changes, not known to be related to loxapine use, have been reported. *Skin:* Dermatitis, edema of face, pruritus, seborrhea. Possible photosensitivity and/or phototoxicity; skin rashes of unknown etiology seen in a few patients in hot summer months. *Anticholinergic:* Dry mouth, nasal congestion, constipation, blurred vision (more likely to occur with concomitant use of antiparkinson agents). *Other:* Nausea, vomiting, weight gain or loss, dyspnea, ptosis, hyperpyrexia, flushed facies, headache, paresthesia, polydipsia. Rarely, galactorrhea and menstrual irregularity of unknown etiology.

References: 1. Sharma T: Rapid response to loxapine in acute schizophrenia. *Curr Ther Res* 25:366-370, March 1979. **2.** Serban G: Loxapine in acute schizophrenic disorder, *Curr Ther Res* 25:139-143, January 1979. **3.** Zisook S et al.: Loxapine succinate (LOXITANE®) in the outpatient treatment of acutely ill schizophrenic patients, *Curr Ther Res* 24:415-426, August 1978. **4.** Thomas JL: Loxapine oral liquid concentrate in the treatment of young adult patients with acute schizophrenic symptoms, *Curr Ther Res* 25:371-377, 1979.

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Residential treatment has become highly specialized in the field of mental health. It is a specific treatment modality for those who need a totally planned and structured environment.

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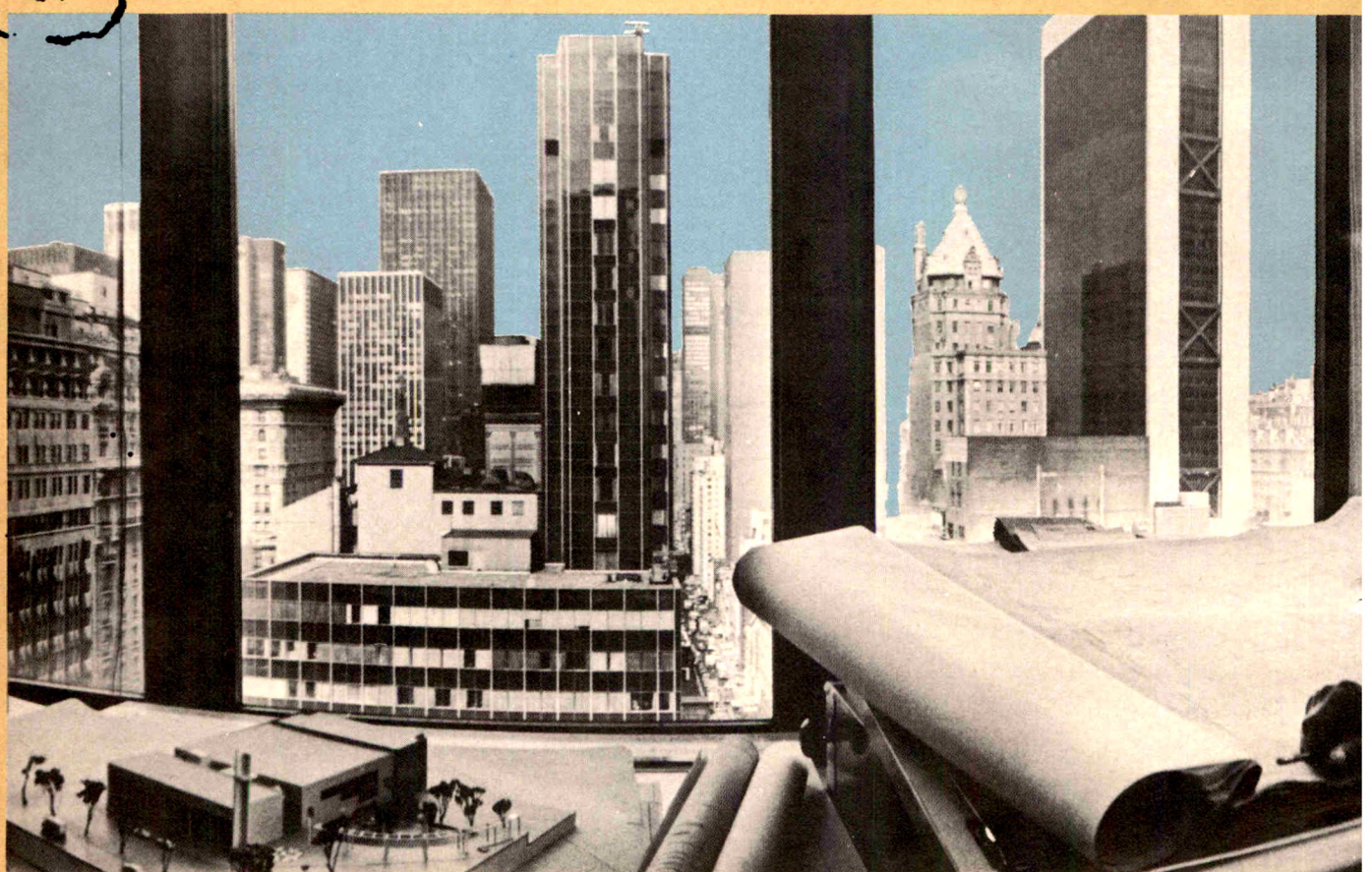
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7 OUT OF 10 NONPSYCHOTIC



The more logical choice for mixed depression and anxiety

☐ provides the well established antidepressant – amitriptyline – to relieve the depressive symptoms
☐ provides the action of Librium® (chlordiazepoxide HCl/ Roche)
- an antianxiety agent – not an antipsychotic – specific for the anxious symptoms
- a benzodiazepine with an outstanding safety record – not a phenothiazine

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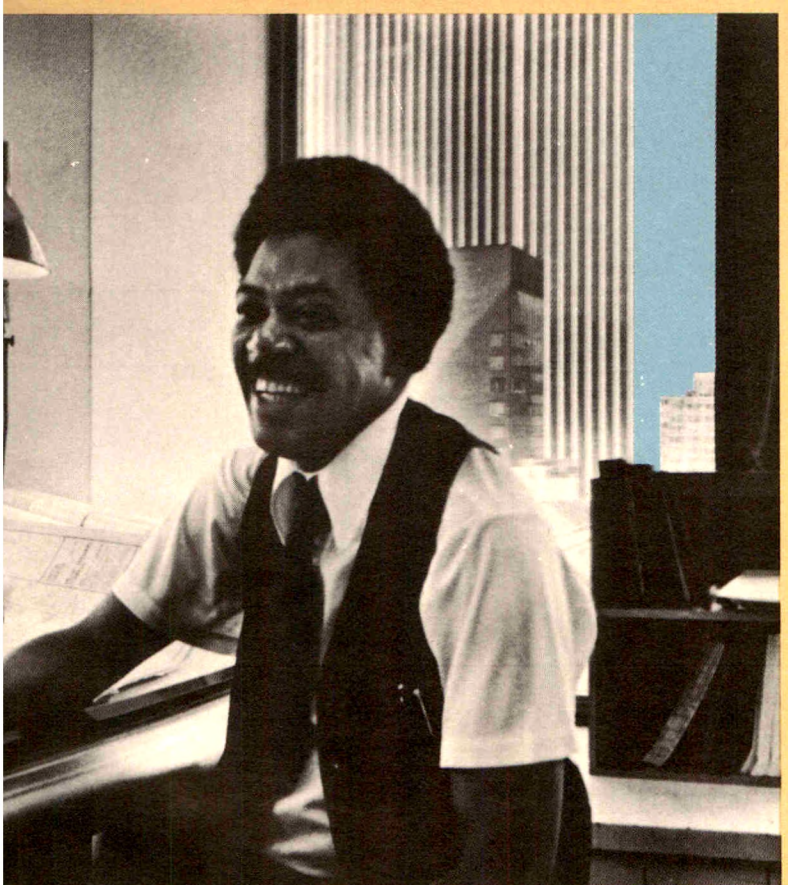
The choice without phenothiazine drawbacks

Minimizes risk of tardive dyskinesia and other extrapyramidal side effects. The causal relationship between the phenothiazines and extrapyramidal side effects, including tardive dyskinesia, is well established. In contrast, the reported incidence of these adverse reactions with Limbitrol or either of its components is rare.

For a complete listing of side effects reported with Limbitrol, consult full disclosure.

ROCHE®

DEPRESSED PATIENTS ARE ALSO ANXIOUS...



In moderate depression and anxiety

What better reason to switch from a phenothiazine combination and start prescribing

Limbitrol® CIV

Tablets 5-12.5 each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)
Tablets 10-25 each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt).



The nonphenothiazine formulation for the nonpsychotic patient

Please see summary of product information on following page.

LIMBITROL® TABLETS Tranquilizer—Antidepressant

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Relief of moderate to severe depression associated with moderate to severe anxiety.

Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants. Do not use with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Contraindicated during acute recovery phase following myocardial infarction.

Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs. Closely supervise cardiovascular patients. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses. Myocardial infarction and stroke reported with use of this class of drugs.) Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving).

Usage in Pregnancy: **Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage; withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline; symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).

Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Periodic liver function tests and blood counts are recommended during prolonged treatment. Amitriptyline component may block action of guanethidine or similar antihypertensives. Concomitant use with other psychotropic drugs has not been evaluated: sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy.

Limbitrol should not be taken during the nursing period. Not recommended in children under 12. In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects.

Adverse Reactions: Most frequently reported are those associated with either component alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline. Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely.

The following list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs:

Cardiovascular: Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric: Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.

Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns.

Anticholinergic: Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract.

Allergic: Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus.

Hematologic: Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia.

Gastrointestinal: Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.

Endocrine: Testicular swelling and gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female and elevation and lowering of blood sugar levels.

Other: Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.

Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. I.V. administration of 1 to 3 mg physostigmine salicylate has been reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.

Dosage: Individualize according to symptom severity and patient response. Reduce to smallest effective dosage when satisfactory response is obtained. Larger portion of daily dose may be taken at bedtime. Single h.s. dose may suffice for some patients. Lower dosages are recommended for the elderly.

Limbitrol 10-25, initial dosage of three to four tablets daily in divided doses, increased up to six tablets or decreased to two tablets daily as required. Limbitrol 5-12.5, initial dosage of three to four tablets daily in divided doses, for patients who do not tolerate higher doses.

How Supplied: White, film-coated tablets, each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) and blue, film-coated tablets, each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt) — bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50.

ROCHE®

ROCHE PRODUCTS INC.
Manati, Puerto Rico 00701

How to initiate and maintain therapy

Select dosage strength appropriate for each patient

- ☐ Limbitrol 5-12.5 is recommended to minimize drowsiness and for elderly patients
- ☐ Limbitrol 10-25 may be indicated for patients who tolerate medication without undue side effects

Specify daily dosage based on symptom severity

- ☐ An initial dosage of three tablets is recommended
- ☐ Dosage may be increased to six tablets or decreased to two tablets daily as necessary
- ☐ Once a satisfactory response is obtained, patients should be continued on the smallest dose required to maintain the desired effect

Utilize dosage options to best accommodate individual patient needs

- ☐ T.I.D. or Q.I.D., familiar regimens most suited for patients who tolerate medication without undue drowsiness
- ☐ Two tablets one hour before bedtime and one tablet midday may minimize daytime drowsiness and help relieve a common target symptom — insomnia
- ☐ Entire dosage h.s. to take maximum advantage of the sedative effect

Your guide to patient management... when you decide medication is needed

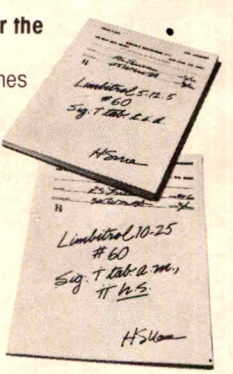
How to make each patient an informed patient

1. Discuss with patients the probability that they will experience drowsiness, especially during the first week.
2. Reassure your patients that drowsiness is one indication that the medication is working and that it may help alleviate their insomnia.
3. Encourage patients to report if drowsiness becomes troublesome so that, if necessary, dosage schedule can be adjusted.
4. Caution patients about the combined effects with alcohol or other CNS depressants. Let them know that the additive effects may produce a harmful level of sedation and CNS depression.
5. Caution patients about activities requiring complete mental alertness, such as operating machinery or driving a car.
6. Warn pregnant patients and patients of childbearing age that the safety of Limbitrol in pregnancy has not yet been established.

Please see complete product disclosure for other pertinent information.

Limbitrol should not be used under the following circumstances:

1. Hypersensitivity to benzodiazepines or tricyclic antidepressants.
2. Concomitantly with an MAO inhibitor. To replace an MAO inhibitor with Limbitrol, discontinue MAO inhibitor for a minimum of 14 days before cautiously initiating Limbitrol therapy.
3. During the acute recovery phase following myocardial infarction.



In moderate depression and anxiety

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Relief without a phenothiazine

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Introduces

An essential program for clinicians learning to incorporate and utilize DSM-III

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Personality Disorders
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Brief Summary of Prescribing Information

Indications and Usage: Symptomatic relief of anxiety, tension, agitation, irritability ar insomnia associated with anxiety neuroses and transient situational disturbances; anxie associated with depressive symptoms and as a treatment of symptoms of anxiety if such sym toms are a significant feature of functional or organic disorders, particularly gastrointestinal cardiovascular.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systen atic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses: As with all CNS acting drugs, warn patients on lorazepam not to operate machinery or motor vehicles, and diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barb turates and alcohol have occurred following abrupt discontinuance of benzodiazepine (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addic tion-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillanc when on benzodiazepines because of their predisposition to habituation and dependence Withdrawal symptoms have also been reported following abrupt discontinuance of benzod azepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid ove sedation.

Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result i symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occa sional convulsions.

Observe usual precautions with impaired renal or hepatic function.

Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepar has not been shown of significant benefit in treating gastrointestinal or cardiovascular compo nent.

Esophageal dilation occurred in rats treated with lorazepam for more than 1 year a 6mg/kg/day. No effect dose was 1.25mg/kg/day (approximately 6 times the maximum huma therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn withi 2 months of first observation. Clinical significance is unknown; but use of lorazepam for pro longed periods and in geriatric patients requires caution and frequent monitoring for symptom of upper G.I. disease.

Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have ha elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function test are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressan effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged i rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship t dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lowe doses. Clinical significance of these findings is not known. However, increased risk of congeni tal malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies Because use of these drugs is rarely a matter of urgency, use of lorazepam during this perioc should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become preg nant to communicate with their physician about desirability of discontinuing the drug.

In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Overdosage: In management of overdosage with any drug, bear in mind that multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring of vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

Ativan® (IV) for (lorazepam) Anxiety

Dosage: **Individualize for maximum beneficial effects. Increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; insomnia due to anxiety or transient situational stress, 2-4mg h.s.**

How Supplied: **0.5, 1.0 and 2.0mg tablets.**

Wyeth Laboratories
Philadelphia, PA 19101

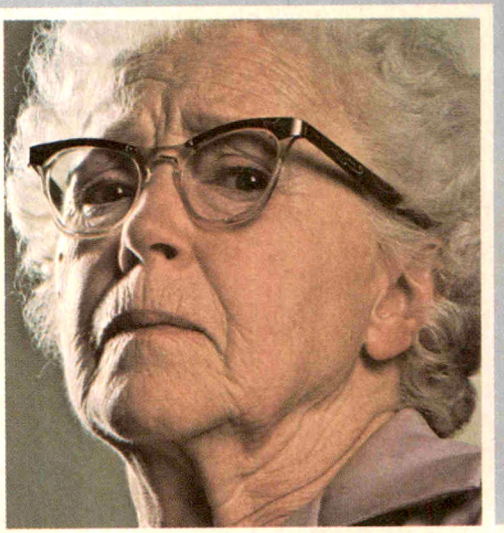
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An age for anxiety.

The aging process all too frequently is accompanied by anxiety states. When anxious older patients do not respond to your sympathetic counseling, a benzodiazepine may be the next logical step.

But which one? Here, more than in many other patient groups, the metabolites of the available compounds may make a decisive difference. Unlike diazepam, clorazepate and chlordiazepoxide, Ativan (lorazepam) has no long-acting metabolites, and its simple metabolism is less readily impaired with advancing age. Steady-state serum levels are rapidly achieved and disappear rapidly once you decide to discontinue therapy. For these reasons, residual effects are not likely to present problems. (The pharmacokinetic profile of a drug can define such characteristics as its absorption, distribution, metabolism and elimination but cannot, at present, be directly related to its therapeutic effectiveness.)

Proved effective in many nationwide, double-blind studies (including patients in older age groups), Ativan is compatible with a wide range of commonly prescribed drugs. (The benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.) Patients who have trouble swallowing solid medication appreciate the small Ativan tablet, which is tasteless and disintegrates within seconds in water or fruit juice.



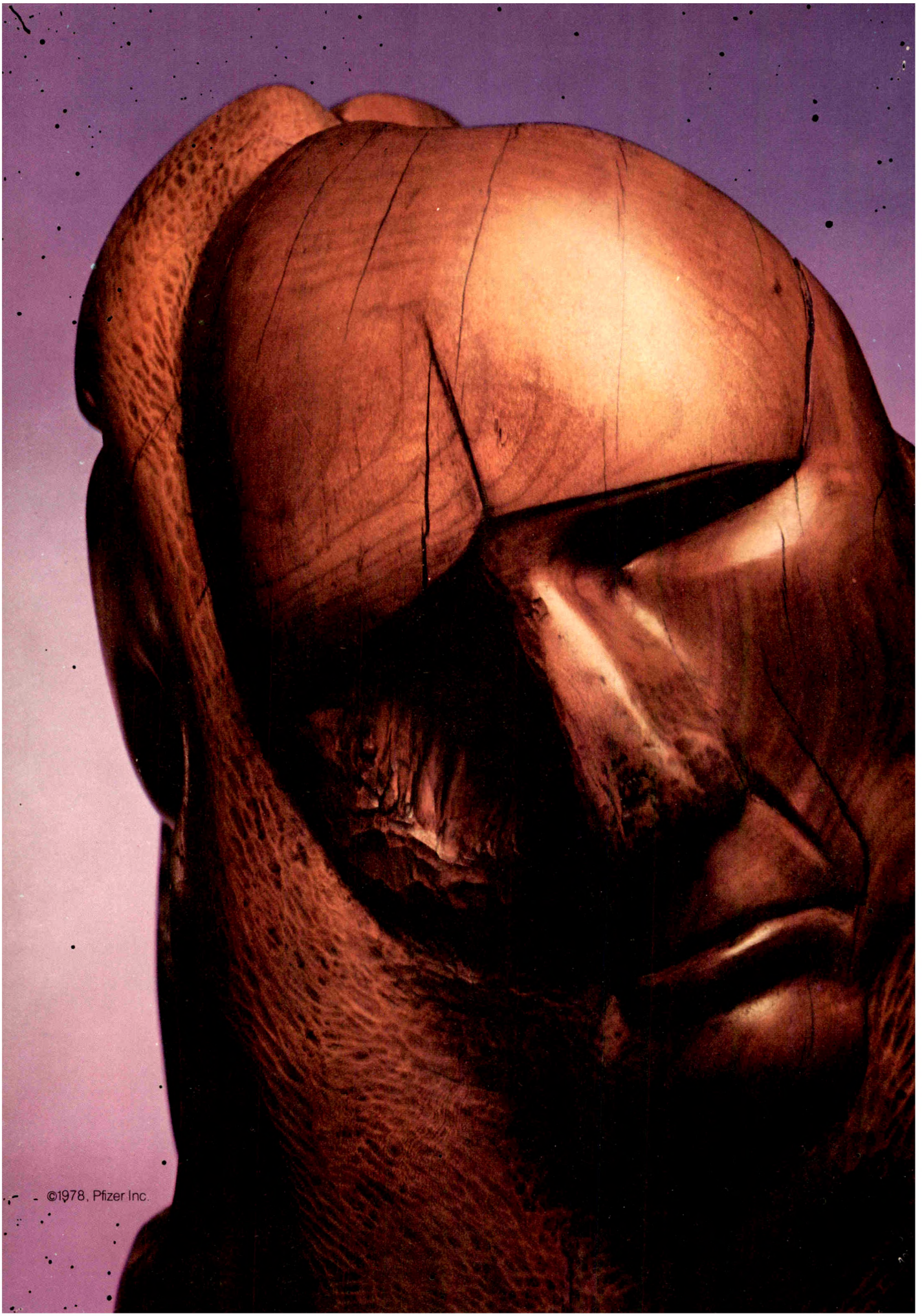
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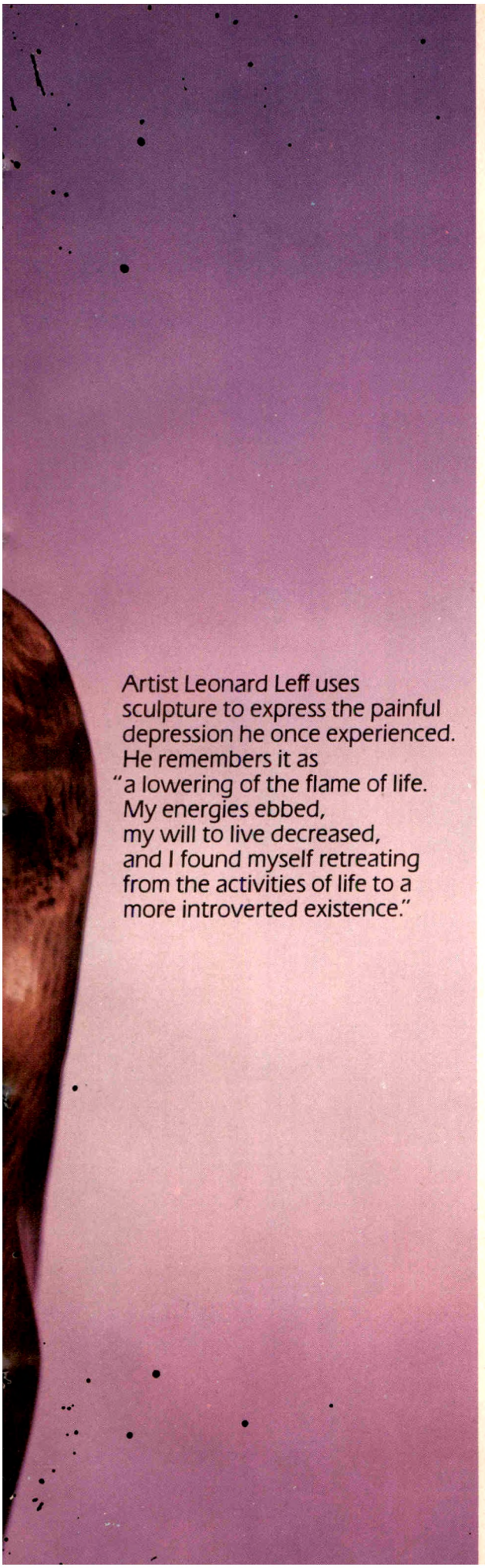
0.5 mg tablet



Ativan® (lorazepam) IV

for Anxiety


©1978, Pfizer Inc.



Artist Leonard Leff uses sculpture to express the painful depression he once experienced. He remembers it as "a lowering of the flame of life. My energies ebbed, my will to live decreased, and I found myself retreating from the activities of life to a more introverted existence."

WHEN DEPRESSION EXPRESSES ITSELF

SINEQUAN®
(DOXEPIN HCl)

ANTIDEPRESSANT EFFECTIVENESS with convenient once-a-day *h.s.* dosage*

150-MG CAPSULE†

Also available in:
100-mg, 75-mg, 50-mg, 25-mg, 10-mg CAPSULES and ORAL CONCENTRATE, 10 mg/ml, in 120-ml (4-oz) bottles

*The total daily dosage of Sinequan, up to 150 mg, may be administered on a once-a-day schedule without loss of effectiveness.

† The 150-mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.

See Brief Summary on next page for information on contraindications, warnings, precautions and adverse reactions.

ANTIDEPRESSANT EFFECTIVENESS

SINEQUAN®
(DOXEPIN HCl)

150-mg CAPSULE*

CONVENIENT ONCE-A-DAY *h.s.* DOSAGE

which may improve patient compliance. The total daily dosage, up to 150 mg per day, may be given on a once-a-day schedule without loss of effectiveness. Sinequan may also be given on a divided dosage schedule, up to 300 mg per day.

PROMINENT SEDATIVE EFFECT

which may help to relieve the difficulty in falling and staying asleep, and the early-morning awakening often associated with depression.

ESTABLISHED ANTIANXIETY ACTIVITY

to help alleviate the anxiety which often accompanies clinical depression.

USUALLY WELL TOLERATED

At doses up to 150 mg per day, Sinequan does not generally affect the antihypertensive activity of guanethidine and related compounds. Tachycardia and hypotension have been reported occasionally. Drowsiness is the most commonly observed side effect. Dry mouth, blurred vision, constipation and urinary retention have been reported.

EXTENDED RANGE OF DOSAGE STRENGTHS

for flexibility in individualizing therapy.

*The 150-mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.

BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate

Contraindications. Contraindicated in individuals who have shown hypersensitivity to the drug, and in patients with glaucoma or a tendency to urinary retention. These disorders should be ruled out, particularly in older patients. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind.

Warnings. The once-a-day dosage regimen of SINEQUAN (doxepin HCl) in patients with intercurrent illness or patients taking other medications should be carefully adjusted. This is especially important in patients receiving other medications with anticholinergic effects.

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies performed in animals have shown no evidence of harm to the animal fetus. Since there is no experience in pregnant women receiving this drug, safety in pregnancy has not been established. There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.

Usage in Children: Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors. Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug. The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered and the dosage involved.

Usage with Alcohol: It should be borne in mind that alcohol ingestion may increase the danger inherent in any intentional or unintentional SINEQUAN overdosage. This is especially important in patients who may use alcohol excessively.

Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug.

Patients should also be cautioned that their response to alcohol may be potentiated.

Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy. Prescriptions should be written for the smallest feasible amount.

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.

Adverse Reactions. NOTE: Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN.

Anticholinergic Effects: Dry mouth, blurred vision, constipation, and urinary retention have been reported. If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage.

Central Nervous System Effects: Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued. Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, numbness, paresthesias, ataxia, and extrapyramidal symptoms and seizures.

Cardiovascular: Cardiovascular effects including hypotension and tachycardia have been reported occasionally.

Allergic: Skin rash, edema, photosensitization, and pruritus have occasionally occurred.

Hematologic: Eosinophilia has been reported in a few patients. There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura.

Gastrointestinal: Nausea, vomiting, indigestion, taste disturbances, diarrhea, anorexia, and aphthous stomatitis have been reported. (See anticholinergic effects.)

Endocrine: Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.

Other: Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice, alopecia, and headache have been occasionally observed as adverse effects.

Dosage and Administration. For most patients with illness of mild to moderate severity, a starting daily dose of 75 mg is recommended. Dosage may subsequently be increased or decreased at appropriate intervals and according to individual response. The usual optimum dose range is 75 mg/day to 150 mg/day.

In more severely ill patients higher doses may be required with subsequent gradual increase to 300 mg/day if necessary. Additional therapeutic effect is rarely to be obtained by exceeding a dose of 300 mg/day.

In patients with very mild symptomatology or emotional symptoms accompanying organic disease, lower doses may suffice. Some of these patients have been controlled on doses as low as 25-50 mg/day.

The total daily dosage of SINEQUAN (doxepin HCl) may be given on a divided or once-a-day dosage schedule. If the once-a-day schedule is employed the maximum recommended dose is 150 mg/day. This dose may be given at bedtime. **The 150 mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.**

Antianxiety effect is apparent before the antidepressant effect. Optimal antidepressant effect may not be evident for two to three weeks.

Overdosage.

A. Signs and Symptoms

1. Mild: Drowsiness, stupor, blurred vision, excessive dryness of mouth.
2. Severe: Respiratory depression, hypotension, coma, convulsions, cardiac arrhythmias and tachycardias.

Also: urinary retention (bladder atony), decreased gastrointestinal motility (paralytic ileus), hyperthermia (or hypothermia), hypertension, dilated pupils, hyperactive reflexes.

B. Management and Treatment

1. Mild: Observation and supportive therapy is all that is usually necessary.
2. Severe: Medical management of severe SINEQUAN overdosage consists of aggressive supportive therapy. If the patient is conscious, gastric lavage, with appropriate precautions to prevent pulmonary aspiration, should be performed even though SINEQUAN is rapidly absorbed. The use of activated charcoal has been recommended, as has been continuous gastric lavage with saline for 24 hours or more. An adequate airway should be established in comatose patients and assisted ventilation used if necessary. EKG monitoring may be required for several days, since relapse after apparent recovery has been reported. Arrhythmias should be treated with the appropriate antiarrhythmic agent. It has been reported that many of the cardiovascular and CNS symptoms of tricyclic antidepressant poisoning in adults may be reversed by the slow intravenous administration of 1 mg to 3 mg of physostigmine salicylate. Because physostigmine is rapidly metabolized, the dosage should be repeated as required. Convulsions may respond to standard anticonvulsant therapy; however, barbiturates may potentiate any respiratory depression. Dialysis and forced diuresis generally are not of value in the management of overdosage due to high tissue and protein binding of SINEQUAN.

Supply. SINEQUAN is available as capsules containing doxepin HCl equivalent to: 10 mg, 75 mg, and 100 mg doxepin: bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's). 25 mg and 50 mg doxepin: bottles of 100, 1000, 5000, and unit-dose packages of 100 (10 x 10's). 150 mg doxepin: bottles of 50, 500, and unit-dose packages of 100 (10 x 10's). SINEQUAN Oral Concentrate (10 mg/ml) is available in 120 ml bottles with an accompanying dropper calibrated at 5 mg, 10 mg, 15 mg, 20 mg, and 25 mg. Each ml contains doxepin HCl equivalent to 10 mg doxepin. Just prior to administration, SINEQUAN Oral Concentrate should be diluted with approximately 120 ml of water, whole or skimmed milk, or orange, grapefruit, tomato, prune or pineapple juice. SINEQUAN Oral Concentrate is not physically compatible with a number of carbonated beverages. For those patients requiring antidepressant therapy who are on methadone maintenance, SINEQUAN Oral Concentrate and methadone syrup can be mixed together with Gatorade®, lemonade, orange juice, sugar water, Tang®, or water; but not with grape juice. Preparation and storage of bulk dilutions is not recommended.

More detailed professional information available on request.

Pfizer **LABORATORIES DIVISION**
PFIZER INC.

Before prescribing or administering, see Sandoz literature for full product information. The following is a brief summary.

Contraindications: Severe central nervous system depression, comatose states from any cause, hypertensive or hypotensive heart disease of extreme degree.

Warnings: Administer cautiously to patients who have previously exhibited a hypersensitivity reaction (e.g., blood dyscrasias, jaundice) to phenothiazines. Phenothiazines are capable of potentiating central nervous system depressants (e.g., anesthetics, opiates, alcohol, etc.) as well as atropine and phosphorus insecticides; carefully consider benefit versus risk in less severe disorders. During pregnancy, administer only when the potential benefits exceed the possible risks to mother and fetus.

Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy, observed primarily in patients receiving larger than recommended doses, is characterized by diminution of visual acuity, brownish coloring of vision, and impairment of night vision; the possibility of its occurrence may be reduced by remaining within recommended dosage limits. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving), and increase dosage gradually. Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension since phenothiazines may induce a reversed epinephrine effect on occasion. Daily doses in excess of 300 mg should be used only in severe neuropsychiatric conditions.

Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; rarely, nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—ECG changes (see *Cardiovascular Effects* below). *Other*—Rare cases described as parotid swelling.

It should be noted that efficacy, indications and untoward effects have varied with the different phenothiazines. It has been reported that old age lowers the tolerance for phenothiazines; the most common neurologic side effects are parkinsonism and akathisia, and the risk of agranulocytosis and leukopenia increases. The following reactions have occurred with phenothiazines and should be considered whenever one of these drugs is used. *Autonomic Reactions*—Miosis, obstipation, anorexia, paralytic ileus. *Cutaneous Reactions*—Erythema, exfoliative dermatitis, contact dermatitis. *Blood Dyscrasias*—Agranulocytosis, leukopenia, eosinophilia, thrombocytopenia, anemia, aplastic anemia, pancytopenia. *Allergic Reactions*—Fever, laryngeal edema, angioneurotic edema, asthma. *Hepatotoxicity*—Jaundice, biliary stasis. *Cardiovascular Effects*—Changes in terminal portion of electrocardiogram, including prolongation of Q-T interval, lowering and inversion of T-wave, and appearance of a wave tentatively identified as a bifid T or a U wave have been observed with phenothiazines, including Mellaril (thioridazine); these appear to be reversible and due to altered repolarization, not myocardial damage. While there is no evidence of a causal relationship between these changes and significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients showing characteristic electrocardiographic changes while taking the drug. While proposed, periodic electrocardiograms are not regarded as predictive. Hypotension, rarely resulting in cardiac arrest. *Extrapyramidal Symptoms*—Akathisia, agitation, motor restlessness, dystonic reactions, trismus, torticollis, opisthotonus, oculogyric crises, tremor, muscular rigidity, and akinesia. *Persistent Tardive Dyskinesia*—Persistent and sometimes irreversible tardive dyskinesia, characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and sometimes of extremities may occur on long-term therapy or after discontinuation of therapy, the risk being greater in elderly patients on high-dose therapy, especially females; if symptoms appear, discontinue all antipsychotic agents. Syndrome may be masked if treatment is reinstituted, dosage is increased, or antipsychotic agent is switched. Fine vermicular movements of tongue may be an early sign, and syndrome may not develop if medication is stopped at that time. *Endocrine Disturbances*—Menstrual irregularities, altered libido, gynecomastia, lactation, weight gain, edema, false positive pregnancy tests. *Urinary Disturbances*—Retention, incontinence. *Others*—Hyperpyrexia; behavioral effects suggestive of a paradoxical reaction, including excitement, bizarre dreams, aggravation of psychoses, and toxic confusional states; following long-term treatment, a peculiar skin-eye syndrome marked by progressive pigmentation of skin or conjunctiva and/or accompanied by discoloration of exposed sclera and cornea; stellate or irregular opacities of anterior lens and cornea; systemic lupus erythematosus-like syndrome.

Dosage: Dosage must be individualized according to the degree of mental and emotional disturbance, and the smallest effective dosage should be determined for each patient.

SANDOZ PHARMACEUTICALS. EAST HANOVER, NJ 07936

SANDOZ

SDZ 9-406R1

MELLARIL® IS KIND (THIORIDAZINE) TO MANY PATIENTS

- **minimal drug-induced akathisia, tremor, and other extrapyramidal side effects**
- **effective control of psychotic symptoms**



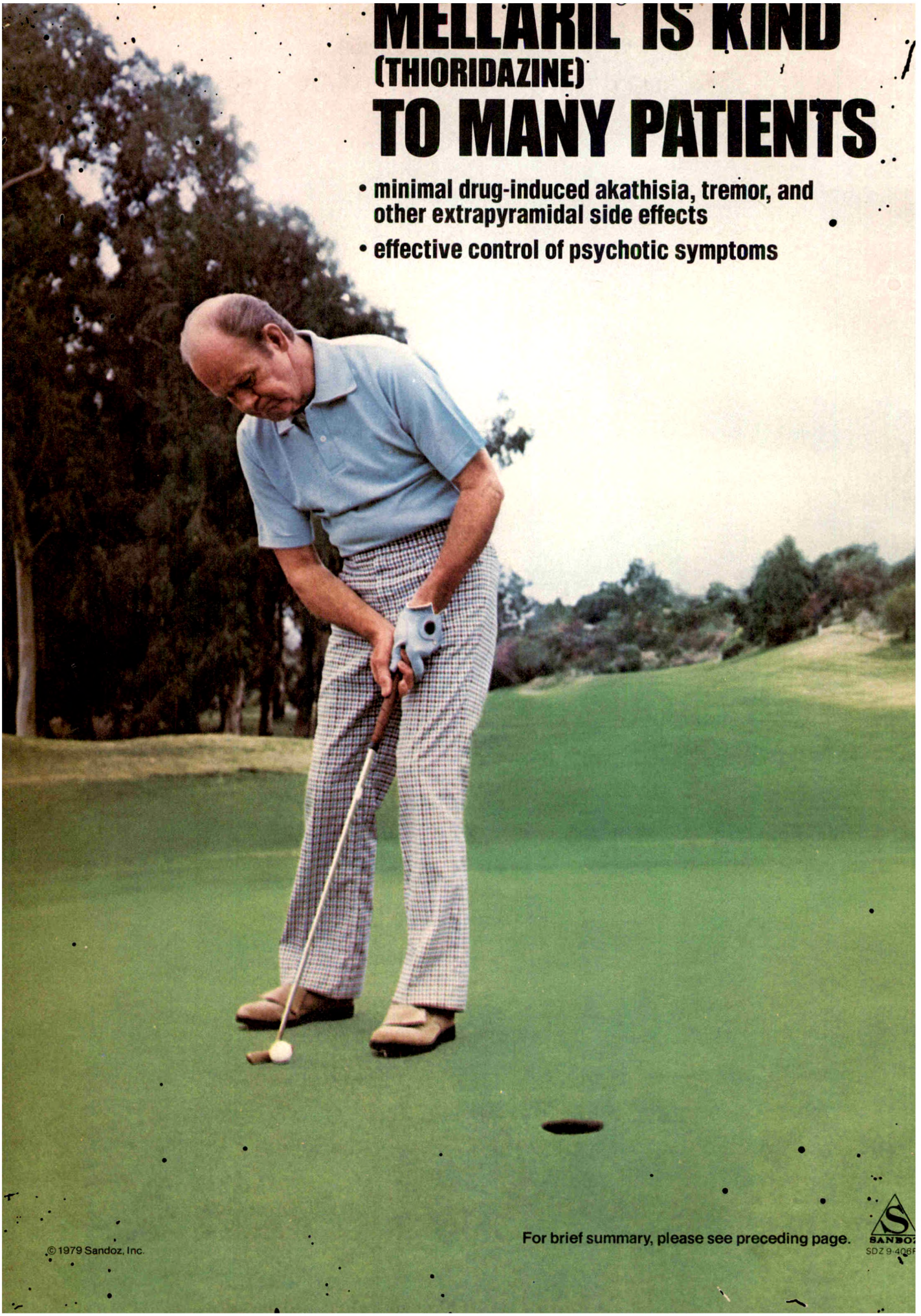
Mellaril® (thioridazine) is kind *two* ways to the patient with psychotic symptoms. It has an impressive record of relieving agitation, excitement, and hallucinations, while successfully helping the patient live and perform in the community. And, unlike other major tranquilizers, Mellaril (thioridazine) causes minimal akathisia or other extrapyramidal symptoms. Mellaril (thioridazine) is contraindicated in patients with hypertensive or hypotensive heart disease of extreme degree.

Effectiveness plus infrequent side effects—that's the kind of kindness the physician likes to see in a major tranquilizer!

MELLARIL® (THIORIDAZINE)

TABLETS: 50 mg, 100 mg, 150 mg, and 200 mg thioridazine HCl, USP

MELLARIL-S™ (thioridazine) SUSPENSION, per 5 ml (teaspoon): thioridazine base equivalent to 100 mg thioridazine HCl, USP



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THE AMERICAN JOURNAL OF PSYCHIATRY

Trust-Based Therapy: A Contextual Approach

BY IVAN BOSZORMENYI-NAGY, M.D., AND BARBARA R. KRASNER, PH.D.

Contextual therapy has emerged from the practice of both individual and classical (transactional-systemic) family therapy. Its basic rationale is that the key dynamic of relationships is the trustworthiness that emerges from mutual consideration of all family members. Its contract maintains a simultaneous, multiple consideration of all the people affected by therapeutic intervention. Contextual therapy is not confined to any one model of practice, although it is founded on specific principles and methods. Its implications are obvious when applied to relationships, yet they are also evident in the aspects of individual approaches that are concerned with the therapist's liability and accountability. Although it encompasses the viable contributions of all schools of psychotherapy, contextual therapy operates out of its specific, ethically founded guidelines and strategies.

Contextual therapy has emerged out of our 45 combined years of psychotherapeutic experience, which includes intensive work with psychotic individuals, extensive experience with children, including the very young, and attempts at healing human suffering across all classes and layers of society (1). The contextual approach incorporates tenets of both individual therapy and classical (conjoint) family therapy, but its major contribution lies in the discovery that the key dynamic of relationships is merited trust. Acknowledging the concept of merited trust leads to a fundamentally new perspective of therapy: simultaneous multilateral consideration of more than one human being. Concern, trustworthiness, adversariness, exploitation, and fair mutuality between family members are heretofore neglected facets of therapy. This radical resource-oriented departure from the traditional (medical or psychological) individual perspective is hard to overemphasize.

For long-range effectiveness, we propose that the foremost therapeutic leverage lies in the processes of trust investments within the family and between family members and the therapist. Because family members are bound to each other by the ethical dimension of mutual trust, or lack of it, we also propose that a sufficiently responsible therapeutic contract is obliged to take account of all persons potentially affected by professional intervention (multidirectedness). Multidirectional accountability is also the key ingredient of therapeutic efficacy. It establishes the therapist's own trustworthiness and provides him or her with a rationale to help avoid the pitfalls of unilateral siding and of manipulation through countertransference. In our view, contextual therapy offers a strategic guideline that holds implications for all forms of psychotherapy.

The term "context" was chosen to indicate the dynamic connectedness of a person with his or her significant relationships. That is, while an individual is a discrete and unique biological entity, dynamically each person's life derives meaning through reference to a social context. Thus the word "context" is used to describe the long-term relational involvement of people, both in its systemic and multi-individual aspects, and includes four key dimensions: the factual, the psychological, the transactional, and the ethical (2). The ethical dimension may be illuminated most simply by the example of a mother who reliably cares for her baby. Her active concern becomes the source of her infant's trust, which, of course, eventually provides the rela-

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tional and psychological foundations of his or her psychosocial development (3). By being caring, available, and accessible, functionally she helps her infant survive, develop, and function in the world. Her trustworthiness is also an ethical contribution. Whatever its comparative limitations, her mothering is a trustable reality based on personal investment and accumulates merit for her in the unique context of the parent-child relationship. The personal uniqueness of both parent and child is just as much a concern in the contextual view as are their transactional "systemic" roles.

In addition to its key dynamic of merited trust or "merit ledger" (reference 1, pp. 78, 371), context also refers to a person's relatedness to his or her multigenerational roots, with their specific racial, religious, and ethnic facets. The dynamic of merited trust was originally understood as interindividual in nature. In addition, however, having roots in common is transpersonal and can convey a legacy that is the result of expectations handed down from generation to generation. More than a theory, therapy based on trust resources is a pragmatic strategy that helps contextual therapists seize on evident signs of mistrust as potential resources for trust building.

A desired commodity, trust reduces the emotional cost of relational investment. This is illustrated by situations in which people cannot tolerate close relationships despite their desperate hunger for closeness. Trust is required as an initial investment in relationships, after which a person can take further steps toward trust building. It is also a necessity for being able to accept periods of transitory unfairness, a situation that is unavoidable in any relationship. A fluctuation of temporarily unilateral dependence, imposition, or even exploitation characterizes close relationships. Balance in relationships is kept through long-range multilaterality, which bridges and equilibrates short-term periods of unfairness and loss of trustworthiness.

ROOTS AND RELATIONSHIPS

The therapeutic significance of the life-long implications of legacy-bound (parent-child) relationships has been grossly disregarded by the traditions of both classical individual and classical family therapy, not to mention many symptom-oriented therapeutic fads. People stay tied to their families of origin long after it appears that family members have ended their connections with each other either by choice or perforce. Who lived for them, who wanted them, who was available to them, and who made material and relational investments in them are fundamental factors in their attitudes toward the world. Having roots and legacies in common is a nonsubstitutive bond among people that not only outlasts physical and geographical separations from families of origin but influences the degree to which offspring can be free to commit themselves to relationships outside of their original ties, including marriage and parenthood of their own. The long-term legacies of parental accountability and filial loyalty are inescapably weighty. A lack of interaction, negative affect, and even indifferent attitudes and abusive behavior may affect parent-child linkages but per se cannot devalue or terminate their importance. Even strong rebuke or revenge is usually meant to test a parent's or child's willingness to respond rather than to end the relationship. Procreation and roots-in-common invariably supersede the quality of the relation as the stimulus for continuity and mutual concern.

As prior investments in past and future generations, relational legacies are not only a part of a person's factual destiny but are an ethical claim. That is, receiving people are obliged to give. Expecting people are obliged to meet expectations. Over-available or over-performing relatives, for example, are entitled to acknowledgment of their investments and merits. In the first place, common roots, legacies, and linkages to racial, religious, ethnic, and familial contexts are nonsubstitutable configurations that contribute to an individual's uniqueness. In the second place, common bonds comprise a demand for balance in relationships. Merited trust born of mutual caring is the mainstay of such a balance.

Broader Implications of Intergenerational Legacies

Legacy-based therapeutic strategies are fundamentally different from psychological or transactional designs. As basic contextual determinants of relationships, legacies are created neither by emotional states nor by power alignments. If therapists are unable to base their convictions about the significance of legacies on their own relational experiences, they may well fall into the trap of the typical contemporary observer of human relationships: in this view repayment of obligations emanating from procreation is optional and depends on the participants' feelings and a variety of power manipulations. In subscribing to such a view, therapists surrender the major dimension of their therapeutic leverage.

Without entering into a debate on what constitutes deep versus superficial therapy, it can be said that an overt concern with relational responsibility almost instantaneously leads the therapist into depth dimensions of relationships. The question of relational responsibility is a private aspect of human life and is seldom faced even between partners themselves. Typically, discussion of the issue is either avoided and leads to isolation, to increasing relational (ethical) stagnation, pathogenicity, and untenability of relationships, or to painful and threatening confrontations. The contextual therapist is thus obliged to learn how to guide people through the most heated, direct, and controversial aspects of their relationship, that is, toward responsibility for trustworthiness. Contrary to its sur-

face appearance, the contextual approach is often short-term rather than long-term and direct rather than intellectualized or rationalistic.

TRUSTWORTHINESS AS A PROPERTY OF RELATIONSHIPS

The result of mutual consideration and actual exchange, trustworthiness is always a relational property of at least two people and cannot be reduced to the psychological universe of either one of them. Although it interlocks with the concept of basic trust as a psychological characteristic (3), trustworthiness always results from a mutilateral investment of relating partners on behalf of their mutual welfare and life interests. Trustworthiness is a characteristic of mature, nonexploitative (object) relations of any kind and, for example, determines whether an exciting love affair can be converted into an enduring marriage. It connects and channels the vicissitudes of the opposing needs of two or more persons in a relationship. Moreover, trustworthiness enables ego strength to be invested in controlling one's tendencies toward an exploitative misuse of close relationships and ultimately serves self-interest through maintaining the relational resource. Furthermore, caring for another person's needs can enhance personal satisfaction through empathy and love.

Retaining a trustworthy relationship is in the reality interest of all participants. A capacity for consideration and responsibility toward another person is thus to be viewed as neither an outgrowth of traditional guilt-based morality nor as a psychodynamic motive "located" in the superego. In practice, concentration on questions associated with the trustworthiness of close relationships incorporates and uses the therapeutic dimensions of individual-oriented psychotherapy like ego strength, reality testing, projections, and insight into distortions. Yet it also goes beyond them in pursuit of a balanced give and take in relationships. From the perspective of contextual therapy, the balance of give and take in relationships is a more sensitive and more accurate measure of reality distortions than is the insight-oriented self-reflection of one individual.

RESIDUAL TRUST AND FAMILY LEGACIES

In therapy the implications of transgenerational legacies are most readily visible in the "accounts" that exist in the reproductive aspects of parent-child relationships. The legacy of parental accountability originates from the existential fact of reproduction. Having given life to a dependent infant, parents owe it nurturance. Conversely, the legacy of filial loyalty originates from the fact of conception, early human survival, and growth. Children are linked to their parents by the event of birth; they are recipients of biological life, psychological endowment, nurturant care, and techniques for staying alive. Receiving life, benefits, skills, and guidance from their roots, the offspring are behooved to transmit them in kind to future generations. Moreover, it should be noted that in modified form the legacy of filial loyalty exists even when children have been given up or abandoned very early in life. Whatever the degree of parental responsibility or behavior after a child's birth, the child's relationship to the parents who gave him or her life is a unique, nonsubstitutable, and unreworkable fact of his or her existence. Even in cases of adoption, paradoxically perhaps, regard for the adoptive child's entitlement to his or her roots tends to improve the relationship with the adoptive parents.

Specifics of a given legacy shape the nature of both parental expectations and filial response to their expectations. For example, the sacrifice of a martyred family member can intensify the heirs' obligation to excel, or, marred by shame, the reputation of a family member can demand exoneration for generations to come. A case in point is the situation of the daughter of an alcoholic father. Bearing the weight of her father's former behavior and apparent failure, she may press her son toward perfection to make up for her filial shame. Overburdened by unfair legacy expectations, her son is faced with the unacceptable choices of abandoning his mother physically or emotionally or of submitting to her impossible demands. More realistically, contextual therapy offers him still another choice: he can help free himself and his mother for a new balance of trust and fairness in their relationship. Overcoming the mistrust, resentment, and stagnation that have accrued from his mother's undue substitutive and compensatory expectations of him, her son can find ways to discover aspects of his grandfather's behavior which serve to exonerate his life. Through active strategic work he can also determine and implement his own terms for accountably fulfilling his legacy rather than becoming entrapped into deferring to his mother's terms or simply learning to evade them.

MULTIDIRECTED PARTIALITY: GUIDELINE TO CONTEXTUAL THERAPY

Multidirected partiality (4), contextual therapy's basic methodology, is used to help the therapist guide clients toward a fresh assessment of legacy expectations and toward more interindividual fairness and trust. It assumes that sooner or later contextual therapists must be prepared to side with all family members, present or absent. It requires a therapist to hear and give courage to each family member as he or she struggles to bring to the surface his or her side. It is a process of consecutive siding with now one and now

the other relating partner in order to help family members state their respective terms with some confidence that opposing views can be heard and tolerated. Multidirected partiality is also meant to provide a paradigm that eventually can enable family members to listen and respond to one another's terms.

Multidirected partiality differs from the therapeutic stances of nonpartial neutrality on the one hand and stagnant unilateral partiality on the other. In our view, nonpartial neutrality is probably humanly impossible to attain, and traditional, individual-oriented therapy typically encourages fixed one-sidedness. Conversely, effectively employed, multidirected partiality begins a constructive process of trust building. It also requires that the therapist have the courage to side temporarily against family members as well as with them. It should be noted that consecutive siding with relating partners is a protection against the dangers of countertransference reactions.

Here a distinction should be made between the strategy of multidirected partiality and relational multilaterality, which is an inherently basic aspect of relationships. Multilaterality means that any action or transaction has at least two sides. In ethical terms it means that usually each of the two partners tends to approach the relationship with a self-centered viewpoint. The degree of fairness in a relationship is determined by the relative consideration each partner accords the other. Since neither only one party nor only an outsider can explore the relative merit accounts between them, the partners should be helped to explore and define their own mutual terms for fairness. For example, in case of adoption the adoptive parents should determine the terms of their own vulnerabilities and requirements for consideration but in the long run it should be the adopted child who determines his own terms of needs and entitlements. The therapist's role is to elicit the family members' active search for a definition and then assertion of their respective terms rather than to inject his or her own moral or behavorial value preferences. By offering consecutive multidirected partiality the therapist encourages family members to explore their own multilaterality. However, the therapist is not a contextually rooted participant in the family members' multilateral relationships. He or she is a natural partner only to the multilaterality of his or her own relationships, e.g., with his or her spouse, children, parents, and others of significance.

Identifying Trust in Stagnant Relationships

The following excerpt demonstrates a contextual process examination of trustworthiness among three family members who are stuck in their relationships with each other (stagnation). What begins as an initial diagnosis of an adolescent turns into a relational exploration of multi-individual diagnoses. The clinical material was obtained by way of a multidirected contract through which the therapist became accountable to all family members who in turn were helped to be accountable to each other. The multidirected therapeutic contract would have been in force whether or not all family members were present at the session.

Bob, a 16-year-old adopted, only son of a middle-class couple, was hospitalized after his second suicide attempt. In the evaluation interview Bob's father took a long time enumerating Bob's many doings: school failure, drug involvements, disrespectful behavior, and so forth. As a first attempt at developing trust resources, without disconfirming the validity of the father's concerns, the interviewer asked whether the parents could recall any instance in which Bob offered a helpful, concerned, or caring attitude to them. The mother denied that this had ever happened. The absurd intensity of her response could be taken as evidence of pathological scapegoating on the mother's part. However, the contextual therapist began to wonder about the possible merits on her side. How could he find aspects of her that would justify his contracting with her with genuine partiality? At this moment she was probably close to a point where she could describe the difficulties of her own childhood. The therapist envisaged that she might be manifesting a desperate overreaction to the fact that Bob's growth would soon lead to her loss of his regular companionship. Consequently, the therapist tried to elicit trust through exploring other aspects of Bob's merit, partly to be able to contract with him, partly to give the parents an opportunity to acknowledge his victimization, and ultimately to find new ways for Bob to display filial loyalty.

At another level of concern, the therapist asked Bob about his feelings concerning adoption. Bob responded that he would like to meet his natural mother but could not conceive of doing something that "brutal" to her. By this time the therapist had factual evidence of Bob's capacity for consideration, especially when Bob added that he did not want to hurt his adoptive parents, either. The merit of the victim becomes immediately obvious: he holds responsibility for not hurting the adult world that betrayed him at the beginning of his life.

From here on engagement in issues of trustworthiness became unavoidable and led directly into the therapist's curiosity about the parents' victimization in their own childhood. The movement from stagnation toward trust building had begun.

In this evaluation and assessment, the contextual therapist initially aimed at exploring the relational context of Bob's motivation to commit suicide. In fact, Bob spontaneously responded to the therapist's questioning by linking his self-destructive inclinations to his adoptive mother's habitual and overt disconfirmation of all his accrued merit as a loving son. In response to Bob's disclosures, the therapist pointed out the young man's concerns for both his natural and adoptive parents. Furthermore, he pointed out that it behooved Bob's parents to acknowledge their son's caring and consideration, which reflected favorably on them as well. At this moment Bob was able to say how much he loved his adoptive parents. With this, his mother started to cry and walked out of the room. Sub-

sequently his father was able to reveal that when he was away on business trips, Bob offered his mother comfort in her sadness and anxiety. This acknowledgment gave the therapist an opportunity to be partial to Bob while also endorsing the father's fairness. Obviously, blame was directed at the mother at this point, and even when she returned to the room she still was unable to acknowledge Bob's personal availability. This instance of extreme mistrust presented an opportunity to encourage her to describe the pain of her own childhood experiences. This, in turn, enabled the therapist to be genuinely partial to her as a victim of the past who presently appeared to be a victimizer.

The father's acknowledgment of Bob's active investment in his parents' well-being marked the beginning of strongly directive therapeutic explorations whose goals included 1) beginning to face intermember fairness and legacies, 2) reducing unfair legacy expectations by acknowledging them and redesigning repayment, 3) correcting one-sided, sometimes distorted, always unaddressed views of each other among family members, and 4) rebalancing the give and take among family members. In a brief time all of the family members had taken initial steps toward personal accountability for responsible action. As trust and trustworthiness began their upward spiral, new chances for positive collaboration and balanced relational growth among family members were likely to occur.

By the end of the initial session the family's concern for each other's well-being began to materialize in the midst of resentment, fears, and accusations. By being prodded to consider the merit as well as the pathology in his or her relationships, in some measure each person could begin to rethink his or her options. The therapist's concern about multidirectionality encouraged each of them to raise his or her side within the hearing of the others, and because they could be heard on their own terms each person was more willing to overcome his or her resistance to listening to the terms of others. After they were helped to voice both satisfactions and disappointments over the quality of their relationships, they could become somewhat freer to dwell on their own parts in contributing to unfair relating. By grasping the notion of joint responsibility for the modes of behavior among them each person was more able to examine interindividual sources of chronic mistrust. All the family members began to comprehend that each of them had made an investment in the welfare of the others and that to be productive these investments needed to be recognized and affirmed rather than discredited or negated. Moreover, no one had been designated as a scapegoat for the situation, and the session had taken place without condemning Bob's parents for the sake of his therapy.

Achieving Autonomy in the Family Context

Given a contractual concern for all family members, the therapist challenged some of the intrinsic unfairness in the family members' ways of treating each other. For example, the parents were charged with the task of learning how to acknowledge Bob's actual commitment and investment in their well-being. They were also asked to rethink on their own the implications of their endless list of demands. Was it actually Bob of whom they had a right to expect so much, or was it unfinished business with their families of origin that had subtly shifted their own filial legacies onto their son (see "the revolving slate," reference 1, pp. 65–67).

Over time Bob has been helped to realign the degree to which he was entitled to live his own life with the degree to which he was required to meet some of his parents' expectations of him. Diminishing the pseudo-adversity between Bob and his parents has established a new sense of security in the family. Fresh confidence about the continuing existence of mutual concern and loyalty among family members has freed Bob to mature in a number of ways. By assuring his parents of his fundamental concern for them, he became likely to win more freedom to commit himself to peer relationships. He was also likely to find more room in which to develop his sexual role and even to explore his biological origins. Eventually he will be helped to achieve this goal through learning how to repay his filial indebtedness in age-appropriate terms. In turn, his parents will be helped to define for themselves specifically what they require of Bob. They will be helped to learn how to state their expectations directly rather than by innuendo and to accept and acknowledge Bob's payments of his loyalty obligations in reasonable "installments." Moreover, they will be helped to learn that he cannot be used indefinitely to compensate his parents for what they may have lost in their own childhoods.

If possible, Bob's parents will be helped to revise their expectations of their son by reconsidering how they themselves have or have not met the terms of their own filial legacies. In clinical descriptions it is customary to ascribe inflexible parental clinging almost exclusively in terms of possessiveness and control through power measures. Conversely, we propose that parents who are overburdened with unmet legacy requirements, whether self-imposed or imposed by their own parents, will inevitably overburden their children. Thus recognizing and reworking long-standing unfairness in their own family legacies will not only free parents from a fruitless expenditure of energy in defensive and retributive behavior but will free their offspring from a similar fate. For example, if his parents were to find sources of trust and appreciation within their original relationships, Bob would become more able to "leave" them and invest his energy in his own welfare and development. Becoming less central to his parents' well-being, because of the investments of other people in their lives, he would feel less helpless. Undefined and boundless obligations invariably lead to destructive parentification of children (1).

In future sessions Bob will continue to be a recipient of therapeutic consideration, but the multidirected contextual approach will incorporate his parents as full partners in the therapeutic effort. As therapy progresses the substitutive function of Bob's symptoms is likely to decrease, and signs of his parents' difficulties are likely to surface with increasing clarity. The parents' childhood struggles will emerge, along with long-standing conflicts over their own invisible filial loyalties. Their marital problems will also come to the fore. From Bob's side, age-appropriate, peer-related problems will be raised and considered. Suicide will be viewed as an attempt to claim entitlement in the context of his family's unfulfillable, excessive expectations. Children who are locked into the endless spiral of expectation of perfection on the one hand and their parents' withholding of recognition on the other are candidates for self-destructive patterns. We view the threat of suicide as an eminent example of the pathogenicity of mistrust and the value of the preventive use of a family's trust resources, as opposed to pathology within individuals, which is the preoccupation of traditional, individual-oriented therapy.

By extending his efforts beyond the intrapsychic dynamics of one individual, the therapist avoided the trap of psychological reductionism. Instead, he identified the family's resources for helping Bob rather than choosing either to ignore or foreclose them. He drew family members toward responsible individuation instead of simply reinforcing the isolation, emotional distance, and mistrust that already existed among them. He began the multilateral process of identifying and mobilizing the residual trust that resided in the family, even in the midst of long-standing, acrimonious relating.

ELEMENTS OF CONTEXTUAL THERAPY

Contextual Assessment

In a contextual evaluation indications of injured trustworthiness between parents and offspring are regarded as signals for potential trust-building. Fundamental to the evaluation is the therapist's capacity to assess some acknowledgeable merit in every family member. Merit in relationships emerges from prior investments of trustworthiness that are capable of yielding interest and returns in trustworthy relating. Of course, all relational accounts are composed of both assets and debits. Assets are the sum total of past and present investments of care, concern, consideration, and devotion. Debits are the sum total of inconsideration, lack of acknowledgment, neglect, abuse, emotional or physical abandonment, or any combination of acts amounting to a failure of commitment and resulting in accrued mistrust. In any case, acknowledgment of merit is a first step on the way to fair return. In fact, even the process of taking an inventory of assets and debits can serve to readdress ethically stagnant relating. The weight of therapy is thereby redistributed from an emphasis on the removal, interruption, relabelling, or restructuring of pathological behavior to a concentration on resources inherent in the "merit ledgers" or balances of accounts among people involved in a relationship. (1)

Therapy

Conducting therapy requires specific training in the contextual approach and skill with a broad spectrum of psychotherapeutic methods. The more comprehensive the therapist's competence, the more he or she is able to assess the facets of a client's context and to integrate the four dimensions of contextual therapy into therapeutic planning. Factual circumstances, communication and transactions, psychology, and merit are elements that combine to determine the outcomes of therapy. In Bob's case, some of his family's factual circumstances include his parents' medical reasons for not having a natural child, the age at which Bob was adopted, and information about his natural parents. The fact of adoption also has a built-in legacy requirement of its own. Despite implicit suggestions of irresponsibility customarily ascribed to people who give their children up for adoption, no adoptive child, or anyone else for that matter, can accept the notion that his natural parent is worthless. Consequently, understanding the reasons why their natural parents gave them up is always a task for adopted children.

In addition to factual circumstances, systemic patterns and role structures associated with a family's communication and transactions are of obvious importance to the therapeutic process. These patterns, sequences, and structures represent the overt, symptomatic level of relational exchanges and are richly characterized in the literature of classical family therapy.

A knowledge of psychology is the third dimension of the contextual approach, which presumes familiarity with intrapsychic development and dynamics. The ego strengths and defenses of each family member, for example, provide the therapist with practical knowledge. In the case illustration Bob's mother may be diagnosed as having a rigidly paranoid type of personality. Her consistent projective inclinations suggest limitations that are to be considered in designing therapeutic strategies. However, initially it is the adolescent's suicidal inclination rather than his mother's psychodynamic configurations that should be the crucial therapeutic and preventive concern. The individually focused question of bypassing the mother's intrapsychic priorities rather than working them through must be subordinated temporarily to the prospect of danger to Bob's life. Once the immediate reality of this priority has been addressed satisfactorily, his mother's entitlement to make claims in her relationships will also be considered. In any case, from a contextual perspective multilateral considerations of entitlement and valid in-

debtedness should not be guidelines and strategies geared simply to each person's intrapsychic dynamics.

The ethical or trust-building dimension of contextual therapy is its guiding consideration. Given his concern for fairness among relating partners, the contextual therapist presumes that the ethic of filial accountability originates from relational reality rather than from guilt anchored in Bob's superego. Therefore he will be guided by each family member's obligation to recognize the impact of legacy expectations and to acknowledge the merit of each person's contributions to the quality of his or her relationships. This guideline is predicated on the fact that the improved quality of family relationships is a shared interest of all family members.

The multilateral contract marks a transition from diagnosing immediately noticeable intrapsychic symptomatology toward gradually uncovering the facets of relational circumstances. By eliciting trust from all family members the therapist can help each of them turn to him in an atmosphere of growing receptivity to help. Furthermore, the trust elicited by the therapist serves to reveal how Bob's inclinations toward suicide intersect with his mother's relationship with her family of origin.

Trust Building and Therapeutic Conviction

Trust building is both the ideological foundation and the primary tool of contextual therapy. It is the process through which residual trust resources among family members or otherwise significant relationships are identified, elicited, mobilized, and used. It is built on the characteristics that provide balance in life: 1) respect for equitability on every member's own terms, 2) integrated give and take in relationships, 3) mutual consideration and "use" of one another, and 4) redistribution of returns from the joint accounts of trust investments.

Clearly there are other powerful forces at play in determining the visible behavior of relating partners. People are also motivated by needs for affection, dependence, and sexuality. People are inclined to use each other unilaterally as objects for transference, displacement, and substitutive retribution. Contextual therapists integrate these factors and others like them but nevertheless proceed from the central conviction that evolving interindividual trustworthiness is the basic fiber of durable relationships and the shortest route to eradicating skewed and distorted behavior.

Knowledge, skill, and personal conviction are all elements in a therapist's capacity to channel and use trust. As a qualified helping professional, the therapist presents himself as worthy of his clients' trust. Over time he will become privy to private aspects of his clients' lives and help them investigate the loyalties in their families of origin, their marriages, and their capacity to parent. As various family members turn to him for help he himself becomes a major if interim recipient of their trust investments, in part because they sense his conviction about multilateral equitability. Trust between therapist and client can flow in a variety of ways:

1. From the outset the therapist receives trust through the client's act of contracting for help. This initial and usually tentative line of trust may be strengthened through the client's response to the therapist's elicitory measures aimed at encouraging people to take initiative on their own behalf.

2. Once clients have returned trust offered to them by a therapeutic investment in their situation the therapist has his own choice to make in terms of how he will use their trust. The therapist can accept the clients' trust, channel it into his own personal relationships, grow and mature from it, and use it to secure his commitment to a responsible performance of his difficult work, or he can convert his clients' trust into a reservoir that supplies his narcissistic desires of superiority and offers him a power-based notion of therapy. He may even use it as unilateral leverage for refueling his own personal relationships.

3. In the tradition of individually oriented therapy, trust can be channelled into a one-to-one relationship between therapist and client. This mode of using trust can create escalating demands in managing an intensification of transference and countertransference attitudes and needs.

4. Rechannelling trust into the process of strengthening relationships between family members is an alternative to the exclusivity of trust development between client and therapist. Using this option the therapist can inject his own regard for accountability and integrity in relationships. He can help family members reveal one another's unacknowledged consideration and contributions. Through his multidirected trust-building efforts, the therapist can elicit responsible attitudes that may lead to a more genuine dialogue (5) among family members.

Using multidirected trust building as a therapeutic methodology requires a knowledge of legacy expectations, a degree of personal maturity, and a set of convictions that can be brought into dialectic exchange.

The skilled contextual therapist operates with a set of convictions that emerge from both his personal and professional experience:

1. Trustworthiness is a decisive relational factor that cannot be reduced to the individual psychologies of the family members.

2. Trustworthiness follows the regulatory forces of earned merit rather than of power. For example, when a person is "successful" in unilaterally exploiting his partner's trust, he inevitably loses ground in maintaining trustworthiness (balance of accounts).

3. Multilateral investments of trust and consideration among family members enhance each person's entitlement to more satisfactory living (multi-

laterality). At least in part this conviction must emerge through the initiative, personal, and familial experience of the therapist.

4. The therapist's task is to elicit multilateral investments in relationships and to identify healing resources for all relating parties (multidirected partiality). This task is to be differentiated from any tendencies on the part of a therapist to impose his own value judgments.

5. A person's inherited legacy expectations cannot be ignored without an increase in the emotional costliness of living (legacies as present here-and-now relational contexts).

6. Children should not be forced to guarantee age-inappropriate nurturance to their parents or be used as unilateral, captive investors in their parents' depleted accounts of trustworthiness. This conviction is based on the further understanding that both parents and children benefit from balanced parent-child accounts (equitability of interests between parents and children).

The capacity to apply contextual concepts and convictions lies in a variety of skills:

1. Using multidirectionality to elicit and catalyze the active participation of all family members in discovering and mobilizing pockets of residual trust resources (1, chapter 5).

2. Helping people develop appropriate terms for repaying realistic legacy expectations within their specific contexts.

3. Helping unreceptive parents accept their children's attempts to repay them in "installments."

4. Eliciting multilateral investments from participating members of long-term significant relationships without the therapist becoming depleted through the effort. That is, the contextual therapist cannot function indefinitely as a primary source of trustworthiness for his clients or he becomes a captive to expectations that he supply hope, trust, dependent support, limitless empathy, and one-sided partiality to family members.

Many signs indicate that our era has reached a dangerous extreme of relational disintegration. Belief in linear progress toward individuation has resulted in a cost to relational responsibility. It has led to a frightening fragmentation of even nuclear families and to widespread grossly inadequate, often dehumanized child-rearing patterns. Moreover, the disintegration of both small and large communities raises the frightening possibility of a generalized downward spiral of trustworthiness, a condition that leads to pathogenic social patterns.

SUMMARY

The contextual approach stresses relational factors as resources for individual health and strength. Its focus on interindividual trust in family life and legacies introduces a fundamentally new perspective to therapy: the simultaneous consideration of more than one human being as the valid subject of therapeutic intervention. The approach retains an interest in both the singularity of individuals and the transactional-communicational systemic discoveries of classical family therapy.

Therapeutic focus on actively facing and planning for balanced relational accounts leads to a resource-based orientation to therapy. Contextual therapy takes care not to negate signals that indicate pathology but interprets them as evolving from a condition of stagnation between two or more people. Stagnation is usually the result of a lack of trustworthiness in relationships.

Work with families evokes the importance of trust and trustworthiness more sharply than individual-oriented therapy. Close and continuous observation of many families leads the therapist to rediscover the significance of trust over and beyond its worth for early childhood development. If basic trust is essential to emotional development in early childhood, merited trust is a fundamental need of adults. Born of multilateral investments of care and consideration among family members, merited trust is the primary principle of contextual therapy, and trust building is its primary strategy. Initially, merited trust is governed mainly by basic legacies. Parental accountability and filial responsibility are basic legacies that leave an impact, whether or not the participants' behavior seems to justify an investment in each other's welfare. Real or prospective legacy sharing is a residual trust resource that can compensate for transitory unfairness.

Contextual therapy is a complex scheme of interlocking concepts and methodological strategies. Its implications and skillful application require extensive study, training, and experience. While trust building is its guiding principle and its primary goal, at base the contextual approach is eclectic and noncompetitive. It underlies and incorporates all viable therapeutic orientations and strategies. The contractual implications of multidirected consideration of all persons affected by the intervention is a guiding principle for psychotherapy as a whole, and in our view this contract is bound to affect all forms of future professional practice, including individual therapy or counseling. In addition, contextual therapy's approach to relationships may have far-reaching implications for a fresh and creative reconsideration of disintegrative social patterns.

Finally, it should be noted that the multilateral perspective of contextual therapy should lead to an ethic of relationships that requires family members to define or redefine the balance of their relationships. In no way should this process be confused with judging or moralizing. Instead, it is a caring responsibility for the future, i.e., prevention in a genuine sense. The study of early development provides evidence for the relevance of present family behavior to the life options of the dependent offspring. At a more advanced state of

evaluation, the value of any psychotherapy will have to be assessed in terms of both curative and preventive impacts. Its multidirected accountability does not only render contextual therapy ethically sound and legally secure, it also provides it with a built-in preventive relevance.

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Notice to Clinical Investigators

Under program support from the Foundations' Fund for Research in Psychiatry, three research laboratories have been designated as resource groups for clinical investigators doing blood level research with antianxiety, antidepressant, and antipsychotic drugs. Investigators who seek to establish the reliability of their methods or those who wish to participate in a proficiency testing protocol should contact the appropriate group listed below.

Benzodiazepine anxiolytics: Drs. Greenblatt/Shader, telephone (617) 956-6997

Tricyclic antidepressants: Dr. Perel, (412) 624-2198

Phenothiazine antipsychotics: Dr. Davis, (312) 996-1065

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Overview: Public Health Issues in Tardive Dyskinesia

BY GEORGE GARDOS, M.D., AND JONATHAN O. COLE, M.D.

Public health concern over tardive dyskinesia has been rising, but the magnitude of the problem has been undetermined. The incidence of tardive dyskinesia is unknown, and prevalence rates yield conflicting and possibly misleading estimates. The natural course of tardive dyskinesia is highly variable: in some patients (probably many fewer than previously believed) it is irreversible. No currently available therapeutic agent satisfies the criteria of safety, marked effectiveness, and prolonged efficacy in the treatment of tardive dyskinesia. Primary prevention involves reducing antipsychotic drug exposure, secondary prevention involves early diagnosis and prompt intervention, and tertiary prevention involves clinical measures to reduce disability and to treat severe cases vigorously. Educational methods that disseminate knowledge and influence prescribing habits need to be identified and used more widely.

Tardive dyskinesia, an involuntary movement disorder characterized by choreoathetoid movements, tics, grimaces, and dystonia, has aroused great concern in psychiatry. One source of this concern is the possibly iatrogenic nature of the disorder and the likelihood of increasing litigation. Perhaps the major reason for worry is that no suitable alternatives to antipsychotic drug treatment are on the horizon; consequently, tardive dyskinesia is not likely to diminish and may in fact become more widespread. A quarter of a century into the neuroleptic era, drug-induced tardive dyskinesia has emerged as a significant public health problem. The response of the profession has shifted from curiosity and mild concern to panic, but there is no clear agreement on the magnitude of the problem. In this article we will approach key issues relating to tardive dyskinesia from a public health viewpoint. We will present relevant data from the literature as well as from our own research. We will offer promising avenues toward resolving problems wherever possible.

INCIDENCE OF TARDIVE DYSKINESIA

Incidence (as distinct from prevalence) refers to the number of new cases emerging in a well-defined population during a given time period. The true incidence of tardive dyskinesia is unknown. Prevalence rates (usually expressed as the proportion of patients with tardive dyskinesia at a particular time in a treatment facility) are often confused with incidence. The reported prevalence of tardive dyskinesia ranges from .5% to 40% in chronic drug-treated patients (1). Baldessarini (2) obtained a mean prevalence of 15% from 25 publications up to 1972 covering 28,000 patients. More recent reports, however, have placed the prevalence of tardive dyskinesia close to 50% in both in- and outpatient populations (3–5).

It is premature to draw alarming conclusions from such high and seemingly increasing prevalence rates. The methodological problems inherent in survey research are formidable and include the following: 1) arbitrary definitions of tardive dyskinesia, 2) measurement problems, particularly questionable reliability of rating scales (6), 3) generalizability (study populations often consist of elderly hospitalized patients, and the findings do not apply to drug-treated schizophrenic patients in the community), and 4) the confounding effect of concurrent antipsychotic treatment, which may influence the level of observed dyskinesia.

In view of the problems with survey research, it is most unfortunate that systematic data on the incidence of new cases of tardive dyskinesia are unavailable. Preliminary findings, however, have been obtained recently by one of us (J. O. C.), who found 15–20 inpatients with dyskinetic movements of recent (3–6 months) origin during consultation work at McLean Hospital over a 12-month period. Approximately 400 schizophrenic patients are admitted or readmitted to McLean each year; thus the incidence figure is roughly 4%–5%. The risk for a schizophrenic inpatient at McLean Hospital to develop tardive dyskinesia in 1 year is 4%–5%. Assuming that the risk remains the same during a second, third, and subsequent years, the risk for such a patient of developing tardive dyskinesia

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might be 20%–25% over a 5-year period. It should be noted that many of the patients at McLean had prolonged histories of drug therapy and multiple hospitalizations, although some developed dyskinesia after only a few months of drug exposure.

The major public health concern is the real possibility that with each passing year of widespread antipsychotic use, increasing numbers of patients will develop tardive dyskinesia. Gathering reliable incidence figures from a wide range of populations at risk over a long time period appears to be the only direct method of clarifying this issue.

NATURAL HISTORY OF TARDIVE DYSKINESIA

Natural history refers to the long-term course of tardive dyskinesia in patients in whom no specific interventions are carried out to influence the movement disorder. The public health issue is whether tardive dyskinesia is progressive and, if so, whether severe and disabling forms of the disorder develop with any great frequency.

Few studies have followed patients for more than a few weeks. A study by Heinrich and associates (7) showed that 40% of a sample of patients underwent spontaneous remission of tardive dyskinesia at 15-month follow-up on continued fixed-dose antipsychotic treatment. Degkwitz and associates (4) re-examined a cohort of 70 chronic drug-treated patients after 2 years. In 26% of the patients tardive dyskinesia increased in severity, in 23% there was no change, and in 10% the movements lessened. Fifteen percent of the patients revealed tardive dyskinesia for the first time at the second evaluation. Chien and Cole (8) reported no statistically significant changes in the dyskinetic movements of 24 patients with tardive dyskinesia after 18 months. In two studies Crane (1, 9) reported considerable increases in the number of patients with tardive dyskinesia at 1-year and 4½-year follow-up, respectively. The demonstration of marked spontaneous fluctuations of tardive dyskinesia ratings in longitudinal studies (10) may partially explain the different findings between studies.

At this time we can reasonably conclude that the meager research evidence available does not point to a progressive disorder in the majority of affected patients.

SEVERE TARDIVE DYSKINESIA

Although most patients show mild degrees of dyskinesia, quite severe cases of tardive dyskinesia have been clearly documented. Moreover, many patients, particularly younger individuals, are distressed by even mild dyskinesia and may become socially withdrawn as a result.

Marked dystonias tend to be seen in patients under 50 years of age and may lead to a disturbing degree of grimacing, contortions of the body, instability of posture, and gait disturbances (11), occasionally resulting in falls and injuries (12). Marked oral dyskinesia may result in dental problems: ill-fitting dentures, bruxism, broken teeth, tongue biting, and oral ulceration (13–15). Chewing and swallowing may also become impaired (14, 16).

In 1979 Ayd (17) described the following respiratory abnormalities induced by severe tardive dyskinesia: grunting, puffing, snorting, and disturbances of rate, rhythm, and depth of respiration. Dyspnea and chest pain (18), as well as life-threatening respiratory and gastrointestinal complications, have been reported in single cases (19). Speech impediments induced by tardive dyskinesia have also been noted (12). Marked weight loss has been described in patients with severe generalized dyskinesia (12, 20, 21). Despondency over the apparently irreversible movement disorder may induce suicidal potential (20). A 1978 report (22) suggested that life expectancy may be reduced in patients afflicted with tardive dyskinesia in comparison with matched controls.

The above description of serious complications is based on case reports. No reliable incidence figures are available for severe and disabling tardive dyskinesia; fortunately, however, the incidence appears to be extremely low. In our 12-year follow-up study of extensively drug-treated chronic hospitalized schizophrenic patients (the report is in preparation), not a single case of disabling tardive dyskinesia emerged in 89 patients. According to data we collected in an informal survey of 20 experienced investigators of the disorder, we estimated that the prevalence of functional impairment persisting after drug withdrawal was less than 10% of patients with dyskinesia.

Overall, we may state that although tardive dyskinesia can become an extremely serious and disabling condition, the more severe forms are rare and are not likely to reach epidemic proportions in the foreseeable future. The public health importance of severe tardive dyskinesia has to be evaluated in the context of the benefits that antipsychotic drug treatment provides for a tremendous number of psychotic patients.

IRREVERSIBILITY OF TARDIVE DYSKINESIA

Prognosis is a key public health issue. If tardive dyskinesia is reversible in most cases, then the major goal might be seen as the optimal management of the disorder, i.e., reversing the condition with the least risk of psychotic relapse. On the other hand, if a substantial number of cases of tardive dyskinesia proved to be irreversible, the paramount public health task would be prevention.

We discussed the natural history and likely outcome

TABLE 1
Summary of Studies of Effects of Antipsychotic Drug Withdrawal on Tardive Dyskinesia

Study	Number of Patients[a]	Age Range (years)	Population Studied	Duration of Observation (months)	Effect of Withdrawal on Tardive Dyskinesia
Uhrbrand and Faurbye (24)	17	52–80	Chronic psychotic inpatients	7–23	Persisted in 10 patients (59%)
Hunter and associates (25)	13	56–84	Chronic female inpatients with dementia	8–36	Unchanged in all patients
Paulson (26)	33	30–80	Inpatients in a state hospital	3	"Most movements failed to subside"
Degwitz (27)	10	20–80	Chronic schizophrenic inpatients	9	Persisted or intensified in all patients
Edwards (28)	20	45–84	Chronic female inpatients	10–12	Persisted in 18 patients; 5 patients became worse
Crane (29)	37	35–57	Chronic schizophrenic inpatients	6–24	Persisted in 36 patients; 10 patients became worse
Hershon and associates (30)[b]	23		Chronic schizophrenic inpatients	4	Oral dyskinesia unchanged
Yagi and associates (31)	19	23–72	Chronic psychotic inpatients	24	Disappeared in 10 patients; persisted in 6 patients; 3 patients regressed
Quitkin and associates (32)	11	24–55	Patients in an aftercare clinic	2–12	Disappeared in 10 patients; persisted in 1 patient
Jeste and associates (33)[c]	21		Schizophrenic and manic-depressive inpatients		Disappeared in 12 patients; persisted in 9 patients

[a]Only patients showing tardive dyskinesia before drug withdrawal were included.
[b]The age range for the patients in this study was not given; the mean age of the patients was 56.5 years.
[c]The age range for the patients in this study was not given; the mean age of the patients was 62 years. The number of months the study lasted was also not given; the mean length of the period of observation was 13 months.

when no specific treatment for tardive dyskinesia is given in the section above. The clinically obvious and most common intervention for tardive dyskinesia is to attempt to reverse the condition by discontinuing the drug or reducing its dose. Withdrawal of antipsychotic drugs and occasionally of antidepressants may result in an increase in the severity of tardive dyskinesia or in the appearance of new dyskinetic movements. The prevalence of withdrawal-emergent dyskinesia has been estimated to be 5%–40% (1). Many of these cases turn out to be withdrawal dyskinesias that are self-limited and usually disappear within 6 weeks (23). If the dyskinesia persists beyond 6 weeks, the likelihood is that the tardive dyskinesia was previously suppressed by drugs ("covert dyskinesia").

The long-term prognosis in patients with tardive dyskinesia who are no longer receiving antipsychotic medication is variable. Case reports and research studies provide examples of all possible results—from complete disappearance of abnormal movement to treatment-refractory, progressively worsening cases.

Table 1 summarizes studies of patients with tardive dyskinesia whose antipsychotic medication was discontinued and who were followed for several months. The data in the table tend to support the view that chronically institutionalized elderly patients with prolonged exposure to antipsychotic drugs are most likely to show irreversible tardive dyskinesia.

The 3 most recent studies (31–33) showed that patients who had received drug treament for a relatively shorter time were more likely to develop reversible dyskinesias. Despite the apparent statistical association between prolonged pharmacotherapy or older age and irreversible tardive dyskinesia, the prediction of prognosis for the individual patient remains problematical. The unpredictability of changes in tardive dyskinesia following drug withdrawal is highlighted by the results of the questionnaire we sent to 20 investigators of tardive dyskinesia. The data we gathered revealed amazing variability of opinions and experiences regarding outcome. The reported frequency of tardive dyskinesia persisting in patients whose drug treatment had been withdrawn for 3, 6, 12, and 24 months ranged from 0% to 100%.

Dose reduction is often preferred to abrupt drug discontinuation because it provides better protection against psychotic relapse. Changes in the status of tardive dyskinesia following dose reduction show the same variability of outcome as they do after drug withdrawal, but the former changes tend to be less dramatic.

From the neuropharmacological perspective, it is tempting to assume that reversible dyskinesias reflect reversible and permanent tardive dyskinesia reflects irreversible neurochemical changes at the synapses of the basal ganglia. Using dopamine agonists, Friedhoff and associates (34) demonstrated receptor sensitivity modification in patients with tardive dyskinesia. This raises the hope that, in some patients hitherto regarded as showing irreversible tardive dyskinesia, the neurochemical changes might be altered and the prognosis may not necessarily be bleak.

The clinical concern and public health focus on the possibly irreversible cases of tardive dyskinesia lose

sight of the fact that prolonged use of antipsychotic drugs may produce a whole range of adverse effects on the CNS. In a scholarly review, Crane (35) described three types of "neuroleptic induced encephalopathies": 1) reversible, 2) irreversible or slowly reversible, and 3) juvenile reversible symptoms. The irreversible category includes hypotonia, dystonias, and Parkinson-like syndromes in addition to the well-recognized manifestations of tardive dyskinesia. To underscore Crane's view, a number of surveys that have focused primarily on tardive dyskinesia have also shown an even higher prevalence of parkinsonian manifestations in chronic drug-treated patients, often despite usual anti-Parkinson drug treatment (3, 36). Not only are there apparently treatment-resistant cases of drug-induced Parkinson-like syndromes in numbers comparable to those of tardive dyskinesia, the functional impairment resulting from akinesia, rigidity, and tremor may well equal if not exceed the usually minimal functional disturbance of tardive dyskinesia. The need for a more comprehensive approach to the problem of the long-term side effects of antipsychotics in place of a narrow focus on tardive dyskinesia alone cannot be overemphasized. The key public health issue remains the risk-benefit ratio when the risk represents the aggregate of all the hazards of antipsychotic drug treatment versus the psychosocial benefits accruing from effective drug treatment.

DRUG TREATMENT OF TARDIVE DYSKINESIA

The following questions need to be answered when assessing the public health impact of a therapeutic agent: 1) What proportion of afflicted patients respond? 2) How effective is the treatment in severe cases? 3) Are the benefits evancescent or long-lasting? and 4) What is the risk-benefit ratio of the proposed treatment?

The major classes of drugs used in the treatment of tardive dyskinesia include dopamine antagonists, cholinergic drugs, and γ-aminobutyric acid (GABA) agonists (37). No drug or therapeutic method has emerged so far that has proved satisfactory in terms of the criteria raised above. Although many clinical drug trials demonstrate improvement in a sizable proportion of patients, few patients in any given trial have shown more than 50% improvement. Long-term clinical trials in tardive dyskinesia are conspicuous by their absence.

Side effects may severely limit the therapeutic potential of otherwise highly promising drugs, such as clozapine or choline. Severe and functionally impaired cases are often effectively treated with large doses of dopamine blockers (e.g., haloperidol). However, in such instances, the possibility of other side effects and the potential "breakthrough" of movements may raise the risk-benefit ratio as a legitimate concern.

From a public health view, the treament of tardive dyskinesia is unsatisfactory. Safe, effective, and specific treatment methods are lacking. Nonspecific measures such as dose reduction may be thought of as belated efforts at prevention with no certainty of success in the individual patient. Therefore, the search for newer and better therapeutic agents needs to be pursued relentlessly, and currently available drugs should be evaluated in long-term studies.

PREVENTION

The public health import of the prevention of tardive dyskinesia hardly needs stressing. Borrowing from community psychiatry, we can conceptualize the problem in terms of primary, secondary, and tertiary prevention.

Primary Prevention

Primary prevention of tardive dyskinesia involves identifying and eliminating etiological factors. The two major variables are antipsychotic drug therapy and age: tardive dyskinesia is virtually unknown in young people who have had no previous exposure to antipsychotic drugs. Because antipsychotic compounds remain the cornerstone of the treatment of acute and chronic schizophrenia, these drugs cannot be replaced. While the search continues for new antipsychotics with little or no extrapyramidal side effects, efforts have also been directed at better delineating the risk factors associated with neuroleptic drug exposure. To date, research focusing on the interrelationship between tardive dyskinesia and drug variables such as dosage, type of drug, anti-Parkinson drugs, treatment interruptions, and duration and amount of drug exposure has yielded conflicting and confusing results (6). Nevertheless, the goal of minimizing exposure to antipsychotic drugs as a primary preventive measure appears to have face validity and may be accomplished as follows:

1. Avoidance of antipsychotics whenever suitable alternatives are available (e.g., lithium or antidepressants for patients with affective illness, antianxiety agents for neurotic anxiety).
2. Optimal use of antipsychotic compounds. Many chronic schizophrenic patients receive antipsychotic doses greatly in excess of what is needed (9, 38, 39). Furthermore, patients who have had one or two psychotic episodes are not necessarily candidates for indefinite drug therapy (40).

Secondary Prevention

Secondary prevention of tardive dyskinesia concerns early case finding and prompt intervention and is based on the following assumptions: 1) tardive dyskinesia has a better prognosis if recognized early and 2)

intervention in early tardive dyskinesia is less harmful in the long run than allowing the disorder to take its natural course.

The above statements appear so obviously true that it becomes tempting to ignore the fact that little solid data exist to substantiate them. Very little research has been conducted on the course of early dyskinesia. It is generally believed that in most patients tardive dyskinesia develops insidiously, early signs being fine vermicular movements or restlessness of the tongue, mild choreiform finger or toe movements, and facial tics or frequent eye blinks. Clinical recognition of early tardive dyskinesia involves systematic observation of patients in a search for the above-mentioned signs. Crane (41) recommended examination of drug-maintained patients every 3 months for signs of tardive dyskinesia. A more aggressive method of detecting the disorder may involve drug holidays for a few days or weeks. A drug-free period of less than 3–4 weeks is usually too short to precipitate psychotic relapse but long enough to uncover dyskinesia. Other, so far experimental methods of detecting early or clinically undetectable dyskinesias include the administration of dopaminergic or anticholinergic challenge doses (42) and electrophysiological techniques (43, 44).

Therapeutic intervention for early tardive dyskinesia may involve a series of diagnostic and therapeutic measures. First, remediable neurological causes of dyskinesia (e.g., Wilson's disease) should be excluded by appropriate tests and by neurological consultation in doubtful cases. The indications for continued antipsychotic therapy should be reviewed and suitable alternatives selected (e.g., lithium carbonate for patients with affective disorder). In patients who are known to require antipsychotic drugs, attempts should be made to find the minimal effective dose level, although no standard method of titrating antipsychotic dosage has been established (45).

Jus and associates (38) have recently shown that stepwise reduction of antipsychotic dosage together with low doses of haloperidol or reserpine was effective in substantially reducing moderate and severe tardive dyskinesia in chronic schizophrenic patients over a four-year period. In patients who have never received an adequate trial without antipsychotics, drug withdrawal could be considered (46), but this recommendation is not generally accepted because of the substantial risk of psychotic relapse (40). Other suggestions to ameliorate or arrest tardive dyskinesia include drug holidays, drug-free weekends, withdrawal of anti-Parkinson drugs, and switching to an antipsychotic with less extrapyramidal effects; however, the research evidence in support of these recommendations remains unconvincing.

In summary, the effectiveness of intervention in arresting or reversing the course of early dyskinesia appears to be promising but remains to be established in longitudinal studies.

Tertiary Prevention

The public health goal in tertiary prevention is to limit and, as far as possible, ameliorate the disability resulting from already established tardive dyskinesia. The procedure is more or less synonymous with the clinical management of the disorder. The specific steps are identical with the intervention in early dyskinesia. In severe cases, the addition of specific drugs to reduce dyskinetic movements may become imperative.

Education

A further important issue in the area of prevention is the efficiency of educational methods such as the continuing medical education (CME) program. Specifically, it is not known how readily expert opinion and established research findings influence the prescribing behavior of the practicing psychiatrist. A disquieting fact emerged from a report by Crane (9) suggesting that presentation of relevant facts in lecture form may lead to no measurable changes in drug prescribing habits, a finding in accord with the experience of most teachers of psychopharmacology. Perhaps other educational methods, such as journal articles, symposia, courses, and audiovisual materials, may be superior techniques for disseminating information and inducing desirable changes in treatment practices. Nevertheless, as the recent history of the slow and spotty implementation of birth control methods in overpopulated areas of the world clearly indicates, there is a long and arduous process from knowledge of preventive techniques to improved public health.

CONCLUSIONS

More effective prevention of tardive dyskinesia is dependent on progress in the isolation of etiological factors and on the arrival of more effective therapeutic agents. Meanwhile, promising avenues include reducing neuroleptic drug exposure, early diagnosis of tardive dyskinesia, and rational clinical management of established cases.

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Carbamazepine in Manic-Depressive Illness: A New Treatment

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The authors evaluated carbamazepine (Tegretol), a drug of choice for treatment of temporal lobe epilepsy, in a double-blind placebo-controlled trial in patients with manic-depressive illness. Seven of 9 manic patients had a partial to marked response; several also showed relapses when placebo was substituted and improvement when carbamazepine was reinstituted. Five of 13 depressed patients showed significant improvement in depression ratings; 3 additional patients experienced partial relapse when placebo was substituted. Carbamazepine might also have prophylactic as well as acute efficacy in patients with both phases of manic-depressive illness, including some patients who do not respond to lithium. Therapeutic effects were achieved with 600–1600 mg/day at blood levels of 8–12 μg/ml with relatively few side effects. Carbamazepine may prove to be a useful additional treatment for affective illness.

Carbamazepine (Tegretol), an iminodibenzyl derivative, has many current therapeutic uses, including the treatment of trigeminal neuralgia (1), pain syndromes (2, 3), diabetes insipidus (4, 5), and dystonic disorders (6), and is one of the treatments of choice for temporal lobe epilepsy (7). We initially became interested in carbamazepine as a potential new agent in the treatment of manic-depressive illness because of our theoretical interest in its temporal/limbic anticonvulsant activity in patients with temporal lobe epilepsy, particularly in the light of its major psychotropic effects in these epileptic patients.

It has been found in 40 reports that approximately 50% of 2,500 patients with epilepsy improved psychiatrically when given carbamazepine (8). Most of these reports were of uncontrolled clinical trials; however, 7 double-blind placebo-controlled trials (9–15) also generally supported the finding that carbamazepine has a significant psychotropic effect. Other studies (1, 8, 10, 16–30) have demonstrated that the interictal personality difficulties associated with chronic epilepsy improve in both children and adults given carbamazepine. Patients describe feeling brighter and more alert and show improvement in initiative and social behavior as well as in the affective symptoms of anxiety, irritability, and depression. In addition, the phasic disorders of epilepsy that resemble functional psychiatric syndromes, particularly manic-depressive illness and schizophreniform psychoses (31–35), often improve in a dramatic and lasting manner with carbamazepine. A large percentage (33%–90%) of these syndromes improve, at times without concomitant reduction in the frequency of seizures (8). In addition, studies have demonstrated that patients who have nonspecific EEG abnormalities without overt epilepsy but who have behavioral problems, often including gross psychopathology and violent behavior, improve on carbamazepine (26, 27, 36–40).

We have postulated a "kindling" model of affective illness in which cumulative bioelectrical changes, particularly in the limbic area, secondary to repeated biochemical or psychological stresses, could result in abnormal limbic neuronal sensitization and major psychiatric disturbances (41–46). Carbamazepine's unique neurophysiological properties as a limbic anticonvulsant and antikindling agent (see the Discussion section) lead us to feel that the drug might be able to usefully modify this type of postulated disorder. Because of these converging theoretical, clinical, and neurophysiological considerations, we began a double-blind, placebo-controlled trial with carbamazepine in manic-depressive illness (44, 45). To our knowledge, these studies in affective illness represent the first clinical trials outside of Japan and the first double-blind placebo-controlled report of carbamazepine's efficacy.

Open clinical trials with carbamazepine in affective illness in Japan (47, 48) suggested that carbamazepine might be effective in manic-depressive illness. Okuma and associates (48) reported that carbamazepine was effective in about 50% of 64 acutely manic patients and in 3 of 9 depressed patients. There was a higher incidence of prophylactic effects in recurrent mania (71%) and depression (64%) in a sample of 31 patients. These positive effects were observed in some patients who did not respond to lithium. In these studies (48) carbamazepine was added to existing drug treatments, often including neuroleptics, lithium, or antidepressants,

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and double-blind placebo controls were not used. However, Okuma and Kishimoto (49) compared carbamazepine and chlorpromazine in acute mania in a double-blind trial. Placebo controls were not used, but both drugs were effective in acute mania; no significant differences were found between the drugs, although there were fewer in the carbamazepine-treated patients.

We are reporting our 3-year experience with the first 24 manic-depressive patients to complete the clinical trial. We have observed that carbamazepine has antimanic as well as antidepressant and prophylactic effects in affective illness. We have published preliminary results for the first 10 patients studied in a case report format (44, 45).

METHOD

Patients were hospitalized in a metabolic research ward at the National Institute of Mental Health in Bethesda, Md. They were diagnosed after an extended period of observation according to the Research Diagnostic Criteria (RDC) of Spitzer and associates (50). Twenty-two patients were diagnosed as having primary affective disorder and 2 as having schizoaffective psychosis. Oral and written informed consent was obtained for the clinical trial and all procedures. Patients with hematologic, cardiac, hepatic, or renal disease were excluded.

It should be emphasized that medical histories, neurological examinations, and routine EEGs were negative for any evidence of seizure disorder or other neurological difficulties. Brain computerized axial tomography (CT scans) and clinical lumbar punctures were also within normal limits for all patients. One patient received many EEGs, including sleep recordings and an EEG with nasopharyngeal leads during an episode of activated psychosis; all of these were within normal limits.

Trials with carbamazepine were accomplished in a double-blind fashion and were preceded by a 2-week placebo period and followed by a placebo period of several days to weeks. When relapse occurred following placebo substitution, carbamazepine was reintroduced for a second time whenever clinically possible. Carbamazepine was generally started at 200 mg given twice daily with meals and increased, as tolerated, in 200-mg increments to a maximum dose ranging between 600 and 1600 mg/day. Serum samples were obtained before the morning dose and were analyzed approximately twice weekly for carbamazepine levels; we attempted to maintain serum levels between 8.0 and 12.0 μg/ml. Blood samples were obtained twice a week for white blood cell count, hemoglobin, platelet count, liver function tests, and blood urea nitrogen for the first 6 weeks and then weekly thereafter.

Clinical response to carbamazepine was primarily assessed using global ratings of depression, mania, psychosis, anxiety, and anger rated twice daily using a 15-point Bunney-Hamburg modified rating scale (51). The ratings were done by consensus of experienced nurse raters who were blind to the drug trial. Self-ratings of mood and side effects were also obtained twice daily, and Brief Psychiatric Rating Scales (BPRS) were rated by nurses three times a week. Clinical response in mania was defined as follows: marked improvement represented a decrease in nurse ratings of 4 or greater or the absence of mania, moderate improvement represented decrease of 2–4 points, partial improvement represented a decrease of between 1 and 2 points, and a poor response represented a reduction of less than 1 point (see table 1). Response in depression (see table 2) was defined as good if the nurses' ratings fell more than 2 points, moderate if the ratings declined more than 1.5, partial if the reduction was between 1.0 and 1.5, and poor if the reduction was less than 1 point. This range of antidepressant improvements is the range we have observed on our unit with tricyclic antidepressants.

Six of the 9 manic patients developed their manic episodes after several weeks of hospitalization; these were often so severe that clinical management required rapid treatment and carbamazepine administration was started. Although each of the 9 manic patients had been in the hospital and receiving placebo for at least 2 weeks and in the hospital for a mean of 60±20 days before the trial, treatment with carbamazepine was started after 2–3 days of moderate to severe mania in some cases. The baseline ratings were taken from the 2 days (or 3 when the data were available) preceding the trial. The baseline ratings in the depressed patients were based on the week before the trial. Selected patients who responded favorably, particularly if they had previously been unresponsive to lithium, were given carbamazepine as a prophylactic agent following discharge.

RESULTS

Antimanic Responses

Table 1 summarizes the results for the 9 manic patients. Four patients had marked overall clinical responses, 1 patient had a moderate response, and 2 others had partial responses. Four of the patients had major clinical relapses when placebo was substituted for carbamazepine. Five of the 7 patients who experienced at least a partial positive response or better had been previously unresponsive to lithium. In table 1, the mean of the twice-daily global ratings made by nurses from the baseline period are compared with ratings from week 4 by group t test for each individual (52). Five trials were statistically significant. When t values from these individual trials were combined according to the method described by Winer (53), there

TABLE 1
Summary of Carbamazepine Trials in 9 Manic Patients

Hospital Number	Age (years)	Sex	Trial (days)	Mean Peak Dose (mg/day)	Blood Level (μl/ml)	Mania Ratings: Baseline Mean	Baseline SE	Last Week Mean	Last Week SE	Significance	Relapse on Placebo	Previous Lithium Response	Overall Clinical Response
95	41	M	37	2000	9.0	7.3	1.0	2.1	0.2	p<.001	Yes[a]	Equivocal	Marked
105	53	F	56	1400	10.0	6.3	0.5	2.0	0.4	p<.001	Yes[b]	Poor	Marked[c]
131	39	F	48	1400	8.7	6.2	0.5	1.7	0.2	p<.001	Yes[a]	Poor	Marked
100[d]	24	M	24	1000	12.6	1.5	0.1	1.0	0.0		No	Poor	Marked
152	24	F	11[e]	1000	15.5	5.7	0.9	3.6	0.3	p<.001	No	Good	Moderate
153	28	F	23	1400	10.0	5.2	0.4	3.8	0.5	p<.10	Yes	Poor	Partial
81	23	F	54	1600	7.5	3.8	0.2	2.2	0.2	p<.05	Equivocal	Equivocal	Partial
150	29	M	14	1400	12.0	8.3	0.3	9.2	0.4	n.s.	No	Poor	Poor
142[f]	35	F	13	1600	11.2	6.8	1.7	6.6	0.5	n.s.	No		Poor

[a]This patient responded well to blind reinstitution of carbamazepine.
[b]Manic episode on placebo was self-limited.
[c]There is evidence that carbamazepine had a prophylactic effect on this patient's mania during a 1½-year follow-up.
[d]This patient was hypomanic; the t test was not applicable because there was no variance in mania ratings; however, this patient's hypomania completely resolved when he was treated with carbamazepine.
[e]The trial was discontinued when the patient developed a skin rash.
[f]This patient had not received previous lithium treatment.

TABLE 2
Summary of Carbamazepine Trial in 13 Depressed Patients

Hospital Number	Age (years)	Sex	Diagnosis[a]	Trial (days)	Mean Dose (mg/day)	Blood Level (μg/ml)	Depression Ratings: Baseline Mean	Baseline SE	Last Week Mean	Last Week SE	Significance	Relapse on Placebo	Previous Lithium Response	Overall Clinical Response
112	63	M	BP	64	1400	8.0	8.7	.6	3.3	.3	p<.001	No		Good
150	29	M	BP	53	800	10.9	5.7	.3	3.1	.5	p<.01	No	Equivocal	Good
161	43	F	BP	87	1400	7.9	11.1	.4	6.4	.4	p<.001	Yes	Poor	Good[b]
105	53	F	BP	114	1600	8.9	5.7	.2	1.7	.4	p<.001		Poor	Good[c]
120	40	F	UP	43	1400	8.2	9.3	.2	7.5	.1	p<.001	Equivocal	Poor	Moderate
137	33	M	BP	43	1200	8.7	7.5	.2	7.4	.2	n.s.	Yes	Equivocal	Partial
103	49	F	BP, SA	35	1200	6.4	7.2	.4	6.8	.4	n.s.	Yes[d]	Poor	Partial[c]
111	27	M	UP	45	1000	10.9	5.3	.3	5.7	.6	n.s.	Equivocal		Poor
99	36	F	UP	47	600	10.6	8.8	.2	8.8	.3	n.s.	No		Poor
116	39	M	BP	84	1200	9.5	5.4	.4	4.4	.5	n.s.	Equivocal	Equivocal	Poor
159	55	F	BP	41	800	11.8	6.7	.3	7.3	.4	n.s.	No		Poor
158	61	F	UP	46	1000	9.3	6.2	.3	6.4	.2	n.s.	No		Poor
33	58	M	BP, SA	58	800	8.3	10.2	.2	9.8	.2	n.s.	Yes		Poor[c]

[a]BP=bipolar, UP=unipolar, SA=schizoaffective.
[b]Despite a significant improvement in depression ratings, this patient continued to cycle between depression and hypomania during carbamazepine treatment.
[c]There is evidence that carbamazepine had a prophylactic effect on this patient's depression during follow-up maintenance treatment.
[d]After 3 weeks this patient was treated with carbamazepine and chlorpromazine and demonstrated an unequivocal benefit from carbamazepine. She relapsed twice; on both occasions her dose had been reduced.

was a statistically significant group improvement ($p<.001$). Improvement in mania ratings was also significant by the more conservative paired t test ($p<.05$).

Figure 1 illustrates the marked improvement in the nurses' ratings of mania across three trials in a 39-year-old woman (patient 131) who had been transferred from a nearby state hospital for treatment of a manic episode of 3 weeks' duration. The onset of her illness began with a hypomanic episode at age 19. The patient experienced four depressions over the next 10 years; these often required hospitalization and ECT. In the past 10 years she had been hospitalized 15 times, primarily for manic episodes. Despite continued outpatient treatment with lithium, she had become manic five times in the 2 years before admission to NIMH. On admission she was hyperactive, talking rapidly, confused, hypersexual, and markedly labile in her affect. She alternated between joviality, weeping, and irritability and was mildly paranoid without evidence of hallucinations or delusions.

Two days after carbamazepine treatment was instituted her mania began to improve; she was completely recovered after 14 days (see figure 1). When placebo was substituted for carbamazepine, she became hypomanic the next day and by the fourth day was as severely manic as she had been before the carbamazepine treatment. When carbamazepine was again reinstituted, she improved markedly after 3 days and remained well while receiving carbamazepine. Because of the possibility of poor compliance in taking medication at home it was unclear whether she had previously not responded to lithium. Therefore, she was switched back to lithium, continued to do well, and was discharged on lithium alone. Two months af-

FIGURE 1
Unequivocal Antimanic Response to Carbamazepine in a 39-Year-Old Manic Woman

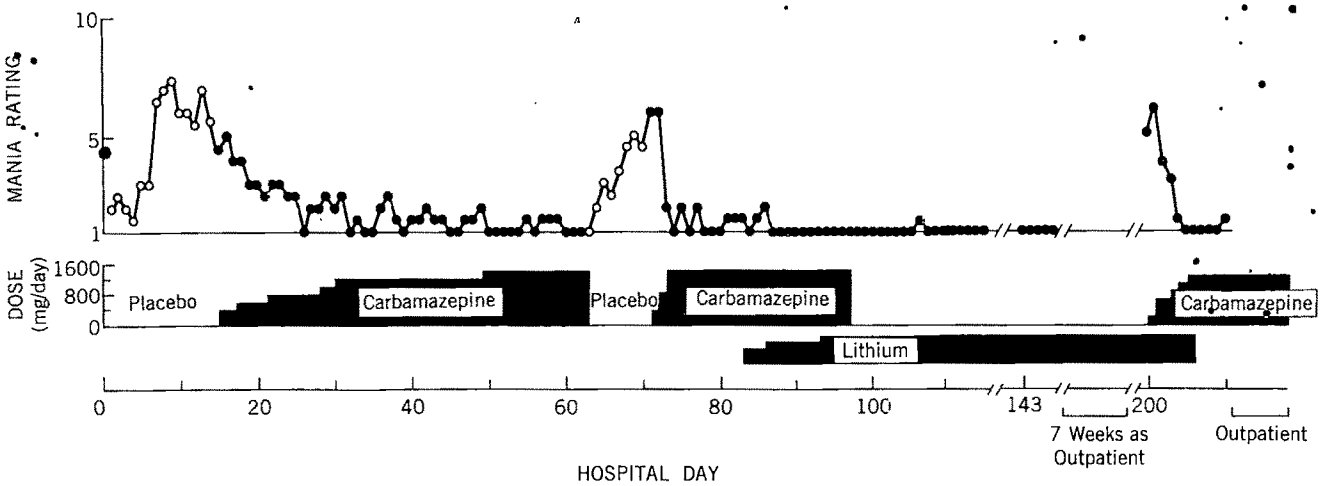
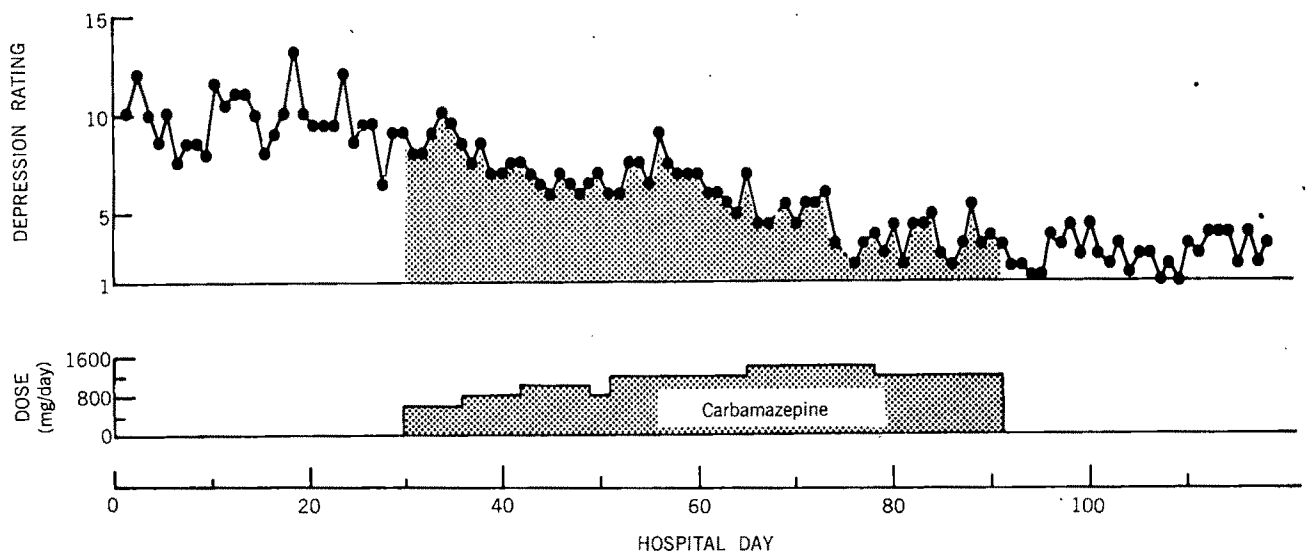


FIGURE 2
Improvement in Depression in a 63-Year-Old Man with Bipolar Illness Treated with Carbamazepine



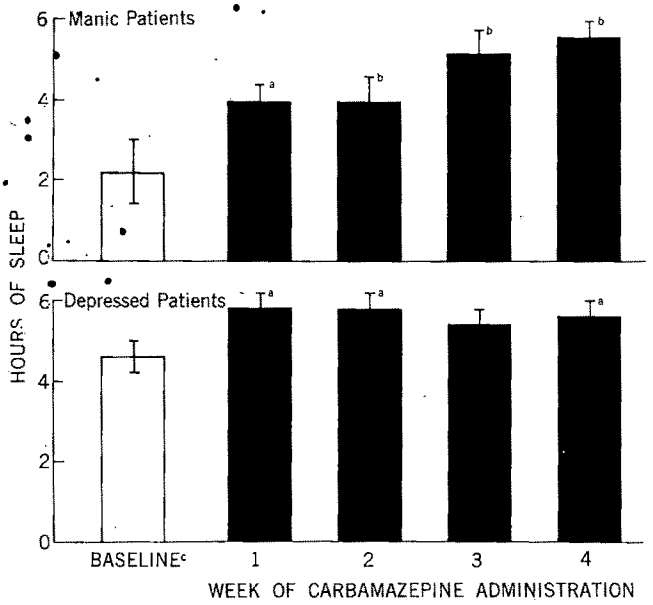
ter discharge she became manic while on lithium at therapeutic blood levels and was rehospitalized. She was again treated with carbamazepine, with rapid resolution of her third manic exacerbation (see figure 1), and discharged on carbamazepine.

Antidepressant Responses

Table 2 summarizes the antidepressant responses in the 13 depressed patients treated with carbamazepine to date. Four patients had good overall clinical responses, and 1 had a moderate response. Eight additional patients had partial or poor antidepressant responses to carbamazepine. Although 3 patients with incomplete clinical improvement had limited relapses when placebo was substituted and 3 more had probable relapses, only 1 patient (patient 103) had a definitive and major clinical relapse from a recovered state. Four patients who responded to carbamazepine and 2 who did not had evidence of previous incomplete or poor responses to lithium. Three patients had evidence of carbamazepine prophylaxis against recurrence of depressive episodes. This included 1 patient (patient 103) who has remained well for 22 months as an outpatient except for a brief relapse following a dose reduction.

When the baseline depression ratings from the week before carbamazepine treatment was instituted were compared with those from the last week of carbamazepine administration by group t test for each individual, improvement was statistically significant in 5 of the 13 trials (see table 2). When t values from these individual trials were combined (53), there was a statistically sig-

GURE 3
fect of Carbamazepine on Sleep in 9 Manic Patients and 13 Depressed Patients



[a]The patients' sleep significantly improved ($p<.01$, paired t test).
[b]The patients' sleep significantly improved ($p<.05$, paired t test).
[c]The medication-free period for the manic patients was 2 days; for the depressed patients it was 1 week.

nificant group improvement ($p<.001$). However, in contrast to the trial in mania, there was only a trend for group antidepressant response by two-tailed paired t test ($p<.10$).

Figure 2 illustrates the complete resolution of a depressive episode in a 63-year-old man (patient 112) with a 23-year history of bipolar mood disorder. Initially the patient was severely depressed, with psychomotor retardation, sleep and appetite disturbances, crying spells, anxiety, periods of confusion and unresponsiveness, and feelings of helplessness. The third night after carbamazepine treatment was started his sleep improved, and after 7 days his depression and periods of confusion lessened. By the third week of treatment with carbamazepine (serum level, 7.5 μg/ml), he was concentrating better and his appetite and energy had returned (see figure 2). Because the patient maintained his improvement when placebo was substituted, his marked response was not considered as unequivocally related to the carbamazepine.

Improvement in Sleep

Duration of total sleep was preliminarily monitored by half-hour sleep checks by nurses blind to the clinical trial (see figure 3). There was a statistically significant increase over baseline in the sleep of both the manic and depressed patients that began in the first week and continued through the fourth week. EEG-monitored sleep recordings in 3 of the depressed patients also documented an increase in total sleep during carbazepine administration based primarily on increases in slow-wave sleep.[1]

TABLE 3
Side Effects of Carbamazepine in 24 Affectively Ill Patients

Symptom	Patients with Symptom	
	Number	Percent
Dizziness	7	29
Ataxia	5	21
Clumsiness	4	17
Drowsiness	3	13
Slurred Speech	3	13
Diplopia	3	13
Rash (pruritic)	2	8
Tremor	2	8
Nausea	2	8
Poor memory	1	4
Confusion	1	4
Aching in legs	1	4
Weakness in legs	1	4
Blurred vision	1	4
Paresthesiae	1	4
Cardiac arrhythmias	1	4

Side Effects

The side effects encountered were those generally expected with an anticonvulsant medication and were consistent with the side effects observed with the use of carbamazepine in other conditions (54). The most common side effect was dizziness; 30% of the patients complained of this symptom sometime during their trial. A smaller percentage of patients complained of such effects as ataxia, clumsiness, drowsiness, slurred speech, diplopia, and aching or weakness in the legs (see table 3). We were able to manage these side effects in each case by either slowing the increase of the drug or transiently lowering the dose. Sedation was not a common side effect, and no patients developed extrapyramidal side effects.

One patient developed a pruritic rash during carbamazepine treatment that rapidly resolved on discontinuation. In another patient a similar rash cleared after placebo substitution, but because this patient had previously not responded to lithium, carbamazepine was carefully reintroduced; the rash did not reappear. However, this patient did develop moderate suppression of her hematologic indices; carbamazepine was therefore discontinued. An additional patient, a 61-year-old woman, developed atrial fibrillation while taking carbamazepine; this resolved 1 week after discontinuation of the drug. Various degrees of heart block have been observed with carbamazepine (55, 56), and changes in pulse rate, particularly slowing, should be carefully evaluated.

Although major dermatological difficulties (57, 58) and isolated cases of pneumonitis (59) and hepatitis

[1]The improvements in sleep were studied in collaboration with Dr. J. Christian Gillin, Unit on Sleep Studies, Biological Psychiatry Branch, National Institute of Mental Health.

(60) have been associated with the use of carbamazepine, the most serious potential side effect is that of the bone marrow suppression. Ten cases of aplastic anemia felt to be "possibly" secondary to carbamazepine treatment have been reported (61, 62). Although 3 fatal cases were felt to be "probably" related to carbamazepine use, all were observed in middle-aged or older patients treated for tic douloureux. To our knowledge, there have been no cases reported since the late 1960s despite increased carbamazepine use in temporal lobe epilepsy.

In a 1975 review of carbamazepine Parsonage (63) stated, "Its safety has still not been seriously questioned, even after more than ten years of usage, and there seems to be no reason to adopt more than ordinary precautions when administering it." Although most investigators concur that carbamazepine is a safe medication, most recommend that prescribing physicians be alert to signs of fever, sore throat, and petechiae or other hemorrhage and obtain complete blood counts weekly for the first month, every other week for several months, and monthly thereafter to monitor for suppression of hematological indices.

White cell counts, hemoglobin, hematocrit, and platelet count were obtained initially on a weekly basis in our patients. There were no significant changes in hemoglobin, hematocrit, or platelets during the trial. However, there was a significant drop in total white cell count by the second week ($p<.01$), reaching a maximum reduction of 25% at week 2. White cell count rose to baseline and above within 1 week of discontinuation of carbamazepine. The decrease in from approximately 8,000 to 6,000 WBC/mm^3 was still well within the normal range. Significant decreases occurred in both polymorphonuclear leukocytes and lymphocytes; both returned to baseline 2 weeks after discontinuation.

As mentioned above, one patient developed a pruritic rash with carbamazepine that resolved on discontinuation. When carbamazepine was reinstituted, the rash did not recur, but the patient developed decreases not only in white count (from 8200 to 5500 WBC/mm^3), but also in hematocrit (from 42% to 37%) and platelet count (from 322,000/mm^2 to 119,000/mm^2). All hematologic indices returned to normal within 6 days of discontinuation of carbamazepine. The decreases in these indices were not clinically significant, and it is unclear whether or not serious bone marrow suppression would have occurred had carbamazepine been continued.

DISCUSSION

We are reporting the first double-blind, placebo-controlled clinical trial of the anticonvulsant carbamazepine in manic and depressive patients. Although our study is ongoing, the early evidence indicates that carbamazepine has antimanic action in at least a subgroup of manic-depressive patients and that it has a therapeutic spectrum different from lithium carbonate in that some patients who do not respond to lithium respond well to carbamazepine. We also have preliminary evidence that carbamazepine may function as an antidepressant in manic-depressive patients as well as have prophylactic value against recurrences of either type of affective episode.

Four of 9 manic patients had marked clinical responses to carbamazepine; a total of 7 out of 9 had a positive antimanic response to carbamazepine used alone. The drug's antimanic effects were observed within the first week of treatment and continued throughout the 4-week trial. This occurred in the absence of notable daytime sedation and with relatively few side effects. Antidepressant effects had a different time course of onset. Although sleep improved early within the first week of active drug administration, antidepressant effects were not evident until after 2 weeks, as has been reported with many other antidepressant medications.

The on-off design of the trial produced particularly convincing evidence of individual response in several patients. When relapse occurred following placebo substitution, reinstitution of carbamazepine represented an additional clinical trial in that particular patient, although these ratings were not included in table 1. In the 2 patients in which this occurred, carbamazepine was also effective when active drug was administered a second time, and in another patient carbamazepine was markedly effective on three occasions (see figure 1). The character of these responses is also illustrated in our paper in which 10 patients were presented in individual case reports (45). The relapses on placebo substitution did not appear to represent a toxic withdrawal syndrome. Mental status was not clouded, and the manic episode that re-emerged was qualitatively and quantitatively similar to that originally observed before carbamazepine therapy. Moreover, there was no evidence of a withdrawal reaction in the patients who did not respond to carbamazepine, which again suggests that the clinical exacerbation was a reappearance of the original symptoms following carbamazepine discontinuation rather than a toxic drug withdrawal reaction.

Although carbamazepine's antidepressant effects are somewhat less definitively demonstrated at this point, its potential as an antidepressant is no less intriguing. Four of 13 patients had good overall clinical responses; a total of 7 had evidence of positive responses to the drug. The ratings from the last week (see table 2) were chosen to maximize our ability to observe antidepressant effects. However, when ratings from week 4 were compared to the baseline week, the overall significance of the results was not altered. Since there were fewer instances of definitive depressive relapses after placebo substitution, we feel that

the antidepressant effects of carbamazepine remain to be unequivocally demonstrated at this point.

Two depressed and 2 manic patients had evidence of prophylactic benefit from carbamazepine treatment. One patient who had had treatment-resistant rapid cycling for 10 years before carbamazepine treatment and another patient who had had one to four depressions per year for the previous 18 years experienced notable prophylaxis in that they have had almost complete resolution of their recurrent affective episodes for periods of 18 and 22 months, respectively. Another 3 patients showed partial evidence of prophylactic benefit: their affective episodes lessened in severity or incidence. One outpatient, not included in this trial of acute inpatients, was given open carbamazepine for treatment of rapid cycling between severe depression and hypomania that was nonresponsive to lithium; she experienced a complete remission with no affective disturbances for 10 months.

In 2 manic patients who had positive antimanic effects from carbamazepine and in 2 others who failed to respond completely to carbamazepine alone, additional neuroleptic medications subsequently proved to be useful. There is suggestive evidence from Okuma and associates (48) that treatment of manic-depressive illness with carbamazepine may be enhanced by concomitant treatment with neuroleptics or lithium for maximum clinical response.

Patients appeared to respond to carbamazepine doses that achieved blood levels between 7 and 12 μg/ml, similar to the high therapeutic range for epilepsy (64, 65). Despite constant dosage, after 2–3 weeks of treatment serum levels usually decreased, presumably related to the well-described decrease in half-life of carbamazepine secondary to induction of hepatic enzymes (66).

We feel that further clinical trials of carbamazepine are indicated and that carbamazepine may emerge as an additional agent in treating manic-depressive illness, perhaps especially in those patients who are nonresponsive to lithium. If our results are confirmed in further investigations, carbamazepine may find a place in the psychopharmacologic treatment of affective illness either as an adjunctive or alternative treatment. It should be determined whether carbamazepine used in conjunction with other medications, such as neuroleptics or lithium, affords additional benefit beyond the use of the traditional treatments alone. This strategy would then be similar to the combination of several different types of anticonvulsants in the treatment of the epileptic patient. Carbamazepine may also prove to be useful as an alternative and primary treatment in patients who are unresponsive to lithium. It is our preliminary impression that some chronically ill, treatment-resistant, and rapidly cycling patients respond well to carbamazepine. Carbamazepine also has neither the tremor nor renal and thyroid side effects of lithium and, despite widespread use has not been reported to have the acute parkinsonian side effects of the neuroleptics or the more important side effect of tardive dyskinesia.

Carbamazepine, with its antimanic properties, may prove to be free of the problem of induction of mania in bipolar depressed patients that is associated with many other antidepressant medications (67). If this proves to be the case, carbamazepine may assume a unique place in the treatment of depression in bipolar patients in that it would be the only medication in current use, other than lithium, which does not have the potential side effect of inducing mania. If carbamazepine continues to demonstrate antimanic, antidepressant, and prophylactic effects in both mania and depression in larger clinical trials, it could prove to be a treatment alternative to currently available therapies in addition to having a different spectrum of both therapeutic effects and side effects.

It is of considerable theoretical interest that the neuroanatomical substrates and biochemical mechanisms involved in carbamazepine's therapeutic effect (44, 45) may differ markedly from those of the other agents routinely used in the treatment of affective illness. Carbamazepine has been demonstrated to suppress after-discharges (68) and spontaneous and induced seizure discharges preferentially in limbic areas over other cortical areas (69–73). It also suppresses seizures kindled in limbic areas by repetitive subthreshold electrical stimulation more readily than those kindled in cortical areas (74–76). Transmission across polysynaptic pathways is depressed by carbamazepine (72, 73, 77–79), especially transmission into limbic areas and from limbic nuclei to diencephalic nuclei (32, 70, 80).

These neurophysiologic actions of carbamazepine could be expected to be efficacious in treating the type of abnormal limbic neuronal sensitization we have postulated to be present in affective illness and psychosis (41–46). Not only would it suppress a potential subcortical limbic focus kindled in amygdala or hippocampus by repetitive stimuli, but it would also be effective in retarding the spread of functional abnormalities to other limbic nuclei or brain regions. It is not known at this point whether these neurophysiologic properties are responsible for carbamazepine's psychotropic effects in affective illness; however, its efficacy does permit and encourage new hypotheses and research into neurobiologic substrates of the affective disorders.

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Newborn Minor Physical Anomalies and Problem Behavior at Age Three

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The authors followed up 136 three-year-olds who had high or low scores for minor physical anomalies of face, head, hands, and feet at birth. Interviews with parents revealed that high-anomaly infants were somewhat more likely to have problem behaviors at age three; this was particularly true for hyperactive-impulsive behavior for boys. Preschool teachers' ratings of hyperactivity, however, did not show a significant relationship to anomaly score. The results suggest some congenital contributors to behavior disorders of childhood, but the relationship between anomalies and problem behavior is weak and limits clinical usefulness of this measure when used alone for identifying a high-risk population.

There has been considerable interest in etiological factors that could explain hyperactive behavior in early childhood. Such factors might help to identify high risk individuals, indicate preventive measures, and help untangle the heterogeneous features of the childhood hyperactivity syndrome.

Recent studies have found an association between the number of minor physical anomalies of hands, feet, head, ears, face, and mouth and behavior and/or learning problems originating in early childhood (1–4). These anomalies are found in children with Down's syndrome but also are associated with other genetic defects and occur within presumably normal populations as well (5).

Quinn and Rapoport (3) reported that hyperactive grade-school-age boys with high anomaly scores (weighted score of 5 or more) were more likely to have had an early onset of hyperactivity (before age three) than were the hyperactive boys in the same study who had a total score of 3 or less. In addition, hyperactive children with high anomaly scores were more likely to have had either a mother who had a history of obstetrical difficulties in this or other pregnancy or a paternal history of hyperactivity. A genotype-phenocopy model was proposed to account for the varied factors that are known to influence the formation of these features in the first trimester of fetal development (5, 6). The finding of more minor physical anomalies in populations of hyperactive children has been replicated by Firestone and coworkers (7, 8), while Campbell and associates (9), Walker (10), and Links and associates (11) have found a higher incidence of anomalies for other behaviorally deviant groups of children. Waldrop and Halverson (2) documented the stability of minor physical anomalies during early and middle childhood and suggested that a measurement of anomalies in newborns may be a useful part of a newborn evaluation to detect infants at risk for later behavioral or developmental deviation. Recently Waldrop and associates (12) reported a significant association between high newborn anomaly scores and nursery school measures of inattentive, hyperactive behavior for 23 boys. Because the findings were based solely on the nursery school observations of a relatively small sample, the question of the clinical usefulness of newborn anomaly score as a predictor of later behavior disorders was not addressed.

In a continuing study (13–16) we examined the newborn minor physical anomaly score and behavior at ages one and two in a subsample of infants selected from a large newborn screening population. An association between high anomaly scores and some "difficult" behaviors was found for both boys and girls. However, there was little consistency in behavioral measures across situations or over time and few significant predictors of behavior problems at age two based on measures from the newborn period or at one year of age. Although the newborn anomaly score did not predict a total score for "problem behavior" at age two, follow-up of this sample to age three was considered important. Behavioral observations at age three were thought more likely to show greater stability across

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time and setting, as reported by other investigators (17), and measures at age three were thought to have more clinical relevance than earlier observations (18).

This is a report of the follow-up evaluation of 136 children at three years of age who had been selected in the newborn period on the basis of their minor physical anomaly score. We used a validated behavioral screening interview (18, 19) appropriate for this population and hypothesized that the newborn anomaly score would predict later behavior disorder, particularly hyperactive-impulse disorders in boys.

METHOD

Subjects

From June 1, 1974, to Sept. 1, 1975, all white infants receiving private care in the Georgetown University Hospital newborn nursery were screened for minor physical anomalies using the Waldrop scoring system (1). A total of 933 infants were seen, and interrater reliability for anomaly score was .92. The frequency distribution of these weighted anomaly scores has been reported elsewhere (13).

Mothers of newborns with weighted anomaly scores of 5 or more had been asked to participate in a prospective study of temperament in early childhood. Children with low anomaly scores (a weighted score of 0–3) were enrolled in the study throughout the screening year as a control group. A total of 147 mothers agreed to participate in the study, 73 mothers of high-anomaly and 74 of low-anomaly newborns. The mothers agreed to yearly clinic evaluations of their children through age three. Follow-up sampling at ages one and two are described elsewhere (13, 15).

When the infants were three years old we made a great effort to contact the families of all 147. We were able to contact 136; 108 were available for a clinic visit, and 28 parents were reached by telephone only. Of the 28 parents contacted by telephone, 20 had moved from the immediate vicinity and 8 refused to come in for a clinic visit. The follow-up group of 136 children is 93% of the original sample and the 108 children available for clinic visits is 74% of the initial sample. Of the 136 children followed up, 64 were boys and 72 were girls; 67 had high anomaly scores and 69 had low scores. Of the 108 seen at the clinic, 56 had high and 52 had low anomaly scores.

Procedure

The developmental assessment battery included the Stanford-Binet Intelligence Scale (Form L-M) (20) and the articulation test from the Zimmerman Preschool Language Test (21). An IQ and articulation age were computed for each child. The psychologist (C.B.) used a five-point bipolar scale to make behavioral ratings of attention, activity level, emotional independence, mood, and persistence based on observations during the evaluative session. For these behavior ratings, interrater reliabilities between two of us (C.B. and L.S.B.) were obtained from 24 consecutive evaluations; they were r=.84 for attention, r=.85 for emotional independence, r=.90 for reactivity, r=.76 for mood, and r=.88 for persistence.

We conducted an interview with the accompanying parent using the Richman-Graham interview (22), which was devised for use with the parents of three-year-olds and later used in an epidemiologic survey. This questionnaire obtains information concerning various aspects of development, health, and behavior and has been used to identify children with behavioral difficulties, marked developmental delay, or physical handicaps. An interrater reliability of .95 from 14 consecutive subjects was obtained for this interview during the two-year follow-up assessment (15). Ratings of problem behaviors were obtained from the Behavioral Screening Questionnaire (BSQ) (19), which is comprised of 12 items selected from the Richman-Graham interview (22). These items are eating, sleeping, soiling, activity, concentration, relationships with siblings and peers, dependency and attention seeking, control, tempers, mood, worries, and fears. A score of 10 or above has been found to have considerable validity in identifying mild–moderate problem behaviors (18, 19). Items are scored 0, 1, or 2 and are summed to provide a behavior score.

In addition to the total score, two subscores were obtained from the BSQ. A hyperactivity score was computed from the sum of the six items dealing with activity, concentration, difficulties getting along with peers, being difficult to manage, temper tantrums, and negative mood. A second subscore was obtained for negative-withdrawn behavior from six items dealing with worries, fears, dependency, negative mood, and difficulty concentrating (two items, negative mood and difficulty concentrating were also used for the hyperactivity subscores). We obtained these two scores because the majority of studies on minor anomalies have found the greatest prediction for hyperactive behavior in boys, and one study has suggested that withdrawn, negativistic behavior was associated with high anomaly scores in girls (23).

A subgroup of 29 children was seen during the clinic visit by a child psychiatrist for a half-hour structured play interview. Details of the interview, subsample, and scoring procedures are presented in full elsewhere (24). During the interview ratings were made of the child's ability to separate from parents, numbers of toys touched, and aggressivity in peer doll play.

Of the 136 children in the three-year follow-up group, 90 were enrolled in various preschool programs. For this group, the preschool teachers were requested to rate the child's behavior on the Preschool Behavior Rating Scale (25). We selected the Preschool Behavior Rating Scale because it had been used by Waldrop and associates (12) in their study of newborn

TABLE 1
IQ and Behavioral Scores for 136 Three-Year-Olds with Newborn Minor Physical Anomalies

				High-Anomaly[a] Group (N=67)						Low-Anomaly[b] Group (N=69)					
	Total			Boys		Girls		Total		Boys		Girls		Total	
Measure	Mean	SD	Range	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Stanford-Binet IQ (N=104)	125	15	71–159	121	12	131	17	125	15	121	16	129	13	125	16
Richman-Graham Behavioral Screening Questionnaire (N=136)	5.53	2.61	0–13	6.76	2.14	5.45	3.19	6.10	2.78	5.11	2.57	5.09	2.13	5.10	2.35
Hyperactivity subscore	2.43	1.59	0–8	3.12	1.48	2.25	1.93	2.67	1.77	2.29	1.64	2.25	1.21	2.27	1.44
Negative-withdrawn subscore	1.75	1.21	0–6	2.25	1.45	1.77	1.12	2.00	1.30	1.58	1.23	1.59	1.01	1.59	1.12
Preschool Behavior Rating Scale (N=90)															
Hyperactivity factor	21.7	10.5	8–15	25.4	11.8	18.81	7.34	22.32	10.38	25.08	11.33	18.42	9.85	21.69	11.02
Withdrawal factor	10.1	6.3	3–31	9.16	6.2	8.12	3.93	8.68	5.21	10.92	6.22	10.15	5.46	10.53	5.8

[a]Weighted score of 5 or more.
[b]Weighted score of 3 or less.

anomalies and hyperactivity at age three. The scale evaluates children for several dimensions of hyperactive or withdrawn behaviors. The hyperactivity factor is comprised of six scales of hyperactivity: frenetic play, induction of intervention, inability to delay, emotional aggression, nomadic play, and spilling and throwing. The withdrawal factor has three scales: vacant staring, closeness to adult base, and chronic fearfulness. The extreme and intermediate points of each scale are defined and the teacher was instructed to use these points as a basis of judgment to rate the child's behavior from 1 to 11 for each scale. Clinic raters were kept blind to anomaly scores until completion of the study.

RESULTS

The measures of principal interest, Stanford-Binet IQ, the behavior score from the Richman-Graham interview, the hyperactivity and negative-withdrawn subscores from the Behavioral Screening Questionnaire, and the Waldrop preschool hyperactivity and withdrawal scores are given in table 1 for the total follow-up sample divided into high- and low-anomaly groups and broken down by sex. A total of 9 children, or 6.7% of the group, had BSQ scores of 10 or above. The sex and anomaly scores of this group are shown in table 2; 7 children had high anomaly scores, 3 had low scores.

For the total sample, children with high newborn anomaly scores had a slightly but significantly higher total score on the 12-item BSQ. High-anomaly children had a mean score (±SD) of 6.10±2.78, while low-anomaly children had a mean score of 5.10 ±2.37 (χ^2=11.35, df=3, p=.01). Only 1 item discriminated between groups; parents of high-anomaly children reported that their children had difficulties getting along with peers more often than did the parents of low-anomaly children (χ^2=11.37, df=2, p=.003).

TABLE 2
Sex and Newborn Anomaly Scores of Three-Year-Old Children with a Behavioral Screening Questionnaire (BSQ) Score of 10 or Above

Subject Number	Sex	Anomaly Score	BSQ Score
1	F	1	10
2	F	7	10
3	M	2	13
4	F	7	10
5	F	6	12
6	F	5	11
7	M	5	12
8	F	5	10
9	M	1	10

In view of Waldrop's findings (23) of a higher incidence of hyperactive behavior in three-year-old boys who had high newborn anomaly scores and withdrawn behavior in high-anomaly grade-school girls, the relationship between newborn anomaly score and three-year measures was analyzed separately for boys and girls (see table 1). The results for boys were consistent with the findings for the total sample; high-anomaly boys had significantly higher scores on the BSQ (χ^2=7.05, df=1, p<.01) and were also reported to have more difficulties relating to peers (χ^2=8.29, df=2, p=.02). The BSQ score did not differ significantly between high- and low-anomaly girls (χ^2=.21, df=1, n.s.).

The hyperactivity subscore of the BSQ was compared to newborn anomaly score for the total sample and for boys and girls separately. Of the 59 boys, 43 had a weighted newborn anomaly score of 3 or below; 23 of these scored below the median on the hyperactivity scale of the BSQ, and 13 scored above. Of the 26 boys who had high anomaly scores (5 or above), 8 scored below the median on the hyperactivity scale,

and 18 scored above (χ^2=6.62, df=1, p=.01). The association between high newborn anomaly score and hyperactive behavior was highly significant (χ^2=7.76, df=1, p=.0053). This association was not significant, however, for the total sample or for the girls. The negative-withdrawn score showed no significant relationship to anomaly score for girls, boys, or for the sample as a whole.

Newborn anomaly score did not relate significantly to the Binet behavioral ratings, to any of the Nursery School Teacher scale (23) items, or to the hyperactivity or withdrawal factors of the BSQ either for the sample as a whole or for male or female subjects. Because these children did not constitute a clinical sample, we did not have a formal diagnostic category for these children against which to validate behavioral measures. However, the hyperactivity subscore of the BSQ did show significant relationship to independent ratings of hyperactive behavior by the child psychiatrist, psychologist, and teacher. Specifically, this subscore correlated significantly with ratings of "aggression in doll play" (r=.40 p<.05), nursery teacher reports of "induction of intervention" (r=.25, p=.03), and psychologists' ratings of activity and of inattentiveness (r=.23 and .22, respectively, p=.03).

Low but significant correlations were found between similar items across settings. For example, the preschool teacher's rating of hyperactivity correlated significantly with the psychologist's rating of inattentiveness and with parent ratings of "difficulty with peers" and "difficulty in managing" the child. Similarly, children considered withdrawn by the teacher were more likely to be seen as dependent by the psychologist and as less active by the parent, indicating some degree of validity for these ratings.

DISCUSSION

In our follow-up of this sample at ages one and two, newborn minor physical anomaly scores were significantly associated with "difficult" temperamental characteristics reported by mothers and by direct behavioral observations. High-anomaly infants were found to be more difficult at both ages, with greater consistency for males. At age three there were fewer significant associations between the newborn measure of minor physical anomalies and problem behaviors. However, for males significant association between hyperactive impulsive behavior reported by the parent and high newborn anomaly score was consistent. Behavioral ratings by the psychologist during the cognitive assessment and nursery school teachers' ratings, however, were not significantly associated with newborn anomaly scores for the total sample or for boys separately. These findings, therefore, are partially consistent with Waldrop's earlier report (12) of a significant correlation between a high anomaly score at birth and inattentive, hyperactive behavior for boys at three years of age in her research nursery school setting. They do, however, provide some modest support for an association between anomalies and hyperactive-impulsive behaviors seen at home, even for a normal newborn screening sample.

Correlations between ratings of similar behaviors measured in different settings were significant but low. This lack of strong agreement across settings may be accounted for by the variety of ratings, variability in behavior, situational specificity, or differences in parent and teacher attitudes. Such low correlations are also found in studies with older children, however, and are not unique to preschool settings. In fact, the degree of agreement between parents and teachers found here is quite close to that reported for older children (26). It must be stressed, though, that behavioral ratings by 90 different preschool teachers are likely to be of only marginal validity.

In view of the poor agreement about behaviors across settings and across time, "continuous problem cases" were examined; the only 2 children in the group who were consistently reported as difficult at ages one, two, and three were both girls with high newborn anomaly scores. Unfortunately, this small number does not permit clinical generalization.

As a significant proportion of the children in this study were selected on the basis of a high newborn anomaly score, one would have anticipated a higher percentage of children classified as having disordered behavior on the basis of the Richman-Graham interview if newborn anomaly score was a good single predictor of behavior problems at three years of age. However, less than 7% of our group was classified as having disordered behavior based on the Richman-Graham BSQ score, which is consistent with findings reported using this screening instrument for a randomly selected population of three-year-olds (22). The notion of a single biological predictor of behavior and/or learning problems is simplistic; the screening population from which this study sample was selected was not considered to be at high risk because infants from the intensive care nursery were excluded at time of sample selection. If such a high-risk population were examined for minor physical anomalies in the newborn period, it is possible that a high anomaly score combined with other risk factors would be a more meaningful predictor of later developmental difficulties. The intial report of an association between obstetrical problems and newborn physical anomalies for a hyperactive child population suggests that infants from intensive care nurseries or with background risk factors should be screened. One of us (P.O.Q.) has screened patients in a high-risk clinic for minor physical anomalies and is currently investigating whether anomalies predict later developmental problems for such a population (27).

The present study also did not explore the dif-

ferential vulnerability of high- and low-anomaly children to environmental stress. It is possible that infants with a high newborn anomaly score who are subjected to stress from an unfavorable environment may be more likely to develop behavioral problems of clinical significance. Such an interaction between biological and environmental influences has been shown by Werner and coworkers (28).

Our study differed from Waldrop's in that ratings of the nursery school behavior were obtained from the teacher of the program in which the child was currently enrolled. Therefore, both the lack of uniformity in settings and the likelihood of differences in the teachers' ability to assess behavior were likely to decrease the reliability and validity of the nursery school ratings. It would have been beneficial to have confirmation of the teachers' ratings by an independent observer. Waldrop and associates (12) found a significant association between newborn anomaly scores and inattentive, hyperactive behavior in a research nursery school for 23 boys. However, all the children in Waldrop's study attended a research nursery school so that there were controls for both setting and observers. In addition, multiple observations were made for each child, and data from a mechanical activity recorder were used. Finally, because only 90 children attended preschool and it is possible that some, more deviant children were not considered ready for school, the preschool study sample may be biased; for example, Richman (18) found language-delayed children were less likely to have been enrolled in preschool.

Behavioral ratings by the clinic psychologist were carried out in a one-to-one novel setting. Although these ratings were reliable and made by the same observer, it is possible that in this situation the children were more likely to be calmer and more attentive than in a naturalistic setting.

Our methods of behavioral evaluation lack the technique of some more intensive studies, which are more feasible with small samples. Walzer and associates (29), for example, after studying young children with sex chromosome abnormalities, reported a method for the longitudinal study of behavioral development in infants and children that used blind observers to confirm data obtained from structured interview or from parent report, in which interviewer bias or the distortions of an untrained observer may have been introduced. On the other hand, our approach did approximate the naturalistic clinical setting.

Firestone and associates (8) studied minor physical anomalies in hyperactive, retarded, and normal control children and their families. Their findings suggest that if an anomaly score alone was used as a marker in the early detection of problem children, the number of false positives in a single family would be very high. Our findings in children at three years of age support this point. In spite of some associations between newborn anomaly score and behavioral problems, newborn anomalies are not acceptable as clinical predictors of behavior problems, because many false positives (as well as some false negatives) would be identified.

Although the correlations between high anomaly score and parents' reports of difficult behaviors are of considerable interest, interpretation of this association remains difficult because these effects may be mediated by environmental factors. For example, high-anomaly infants might have high-anomaly mothers who in turn could be irritable; poor maternal rapport could produce these differences in infant behavior. Alternately, a high-anomaly father could create a disturbed home environment that disturbs the mother and/or child. Further studies on the epidemiology and behavioral correlates of minor physical anomalies should include parental anomaly and temperament ratings as well as a more traditional clinical assessment of family setting.

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The Case of Anna O.: A Reformulation

BY MARC H. HOLLENDER, M.D.

The best known of all psychotherapy patients, Anna O., entered treatment with Josef Breuer in December 1880. Modern psychotherapy begins with that encounter. Information gathered in the intervening years places us in a favorable position to review and reformulate the case. To do so may have practical and theoretical, as well as historical, importance.

After the passing of a century, one in which much has been learned about psychotherapy, we are in a favorable position to review and reformulate the germinal case of Fraulein Anna O. For our purposes, three rich strands of data are available: 1) Breuer's account of the illness and treatment of the patient, an account more detailed than was customary at the time but sketchy by present-day standards, 2) biographical information about the later life and work of Anna O. (Bertha Pappenheim), and 3) biographical information about the therapist, Josef Breuer.

ANNA O.'S ILLNESS

Anna O. had nursed her father, who was dying of a "peripleuritic abscess" (presumably tuberculosis), for six months before she became incapacitated. Breuer (1) stated,

> During the first months of the [father's] illness Anna devoted her whole energy to nursing her father, and no one was much surprised when by degrees her own health greatly deteriorated. No one, perhaps not even the patient herself, knew what was happening to her; but eventually the state of weakness, anemia and distaste for food became so bad that to her great sorrow she was no longer allowed to continue nursing the patient. The immediate cause of this was a very severe cough . . . a typical *tussis nervosa*. (p. 23)

Breuer was called because of the cough, but he quickly perceived that much more than the cough required his attention.

To treat each of a myriad of symptoms, the patient herself devised a procedure she referred to, when speaking seriously, as a "talking cure" and jokingly as "chimney-sweeping." It soon became evident to her physician that

> every one of the spontaneous products of her imagination and every event which had been assimilated by the pathological part of her mind persisted as a psychical stimulus until it had been narrated in her hypnosis, after which it completely ceased to operate. (1, p. 32)

Breuer (1) also stated that "complexes were disposed of by being given verbal expression during hypnosis."

TREATMENT OF THE SYMPTOMS

How did Anna O. happen to invent the talking treatment that has come to be known as "Breuer's Cathartic Method"? We do not know for certain, but there are some clues. It seems likely that she was influenced by Jacob Bernay's book on the Aristotelian concept of catharsis, which was published in 1880. According to Ellenberger (2), the book, which was discussed by scholars and became a topic of conversation in Viennese salons, stimulated a widespread interest in catharsis. Thus, it is likely that Anna O. was introduced to catharsis as a method and then put it to use as a means of capturing and holding the attention of her scientifically minded physician. If such was her conscious or unconscious purpose, she succeeded admirably.

We might now ask: Is the talking cure effective and, if so, how does it work? Breuer (1) stated that "each symptom disappeared after she [Anna O.] had described its first occurrence." To explain this phenomenon, Freud (3) postulated that "the illness occurred because the affects generated in the pathogenic situations had their normal outlet blocked." As a result, the "'strangulated' affects were then put to an abnormal use." Freud added,

> In part they remained as a permanent burden upon the patient's mental life and a source of constant excitation

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for it; and in part they underwent a transformation into unusual somatic innervations and inhibitions, which manifested themselves as the physical symptoms of the case. For this latter process we coined the term "hysterical conversion." (p. 18)

The schema used to explain the response to the cathartic method involves a hydraulic model. When a closed valve, producing a reaction (symptom), is released (by catharsis and abreaction), pressure is dissipated and a normal state reestablished. However, clinical experience tells us that catharsis and abreaction by themselves are effective only in grief reactions and perhaps traumatic neuroses, and then merely to a limited extent. They are not usually effective in treating hysterical symptoms. Ellenberger (2) came to the conclusion that "the famed 'prototype of a cathartic cure' was neither a cure nor a catharsis." Breuer (1) himself acknowledged that the disappearance of symptoms after being talked away might very well "be explained by suggestion."

It may be, however, that the talking cure did work but that it actually consisted of more than catharsis and abreaction. In the process of talking, Anna O. converted a nonverbal message, expressed in pantomime or body language and called a hysterical symptom, into verbal language. In other words, in her free associations Anna O. converted or translated her message from one language into another. Once she expressed herself directly and openly, she no longer had need for indirect and disguised communication (the so-called symptom).

It is noteworthy that Breuer's treatment of Anna O. at a time when she was not using the talking cure involved decoding the content of a hysterical symptom, interpreting its meaning, and overcoming a blockage by obliging the patient to talk about the inhibiting factor. Breuer (1) presented the following account:

> For two weeks she [Anna O.] became completely dumb and in spite of making great and continuous efforts to speak she was unable to say a syllable. And now for the first time the psychical mechanism of the disorder became clear. As I knew, she had felt very offended over something and had determined not to speak about it. When I guessed this and obliged her to talk about it, the inhibition, which had made any other kind of utterance impossible as well, disappeared. (p. 25)

NATURE OF THE "ILLNESS"

Breuer and Freud's formulation of Anna O.'s disorder was essentially symptom-oriented, intrapsychic, and psychobiological. The formulation that I will now propose is essentially person-oriented, interpersonal, and psychosocial.

The nature of a major problem faced by Anna O. was described in the first paragraph of the case history. Breuer (1) stated,

> She [Anna O.] was markedly intelligent, with an astonishingly quick grasp of things and penetrating intuition. She possessed a powerful intellect which would have been capable of digesting solid mental pabulum and which stood in need of it—though without receiving it after she had left school. (p. 21)

Anna O., a bright and lively person, lived at the wrong time and in the wrong place. A woman's opportunities for a professional career in nineteenth century central Europe were extremely limited. As Ellenberger (2) pointed out, the universities were still closed to women. Consequently, Anna O.'s situation was a "contrast between the ambitions and the obstacles set to ambitions." Her plight was compounded by the fact that she was a daughter in an Orthodox Jewish family, in which sons traditionally were accorded preferential treatment. Schooling for Anna O. ended when she was 16. Her brother, who was approximately one and one-half years younger and possessed neither her intellect nor her talent, was afforded educational opportunities closed to her and became a lawyer.

Not only was Anna O., as a female, relegated to an inferior position in her family with future prospects limited to that of becoming a wife and mother, but at the age of 21 she was suddenly called on to assume the onerous chore of nursing her father.

After describing the nature of Anna O.'s problem as a woman in Vienna in 1880, Breuer (1) mentioned the manner in which she tried to cope with it:

> This girl, who was bubbling over with intellectual vitality, led an extremely monotonous existence in her puritanically-minded family. She embellished her life . . . by indulging in systematic day-dreaming, which she described as her "private theatre." (p. 22)

Breuer quickly recognized the nature of Anna O.'s plight, but evidently he did not as quickly become aware of how fascinated he was by his bright, attractive, and personable patient. Jones (4) pointed out, "In those days, to devote hours every day for more than a year to a single patient, and an hysteric at that, signified very special qualities of patience, interest, and insight." Perhaps the words "personal involvement" should be substituted for "insight." While Breuer and Anna O. focused on her symptoms, a relationship developed between them, based on intellectual and sexual attraction, that might aptly be characterized as mutual infatuation.

As Pollock (5) has suggested, a special circumstance may have played a substantial part in drawing Breuer to Anna O. She may have evoked a profound reaction in him by rekindling repressed longings for his mother, who had died "in the blossom of her youth and beauty" when he was between 3 and 4 years of age. The mother, as remembered by her son, and Anna O. may have been alike in many respects. Perhaps the early "imprinting" of an image, unconscious and indelible,

was at work. Whatever the roots—and the reactivation of feelings about the therapist's mother would certainly be important—the relationship between physician and patient became intense and engrossing.

Breuer's fascination must have been evident to Anna O. not only because of the time he devoted to her care but also because of the nature of the attentions he showed her. Clearly, his interest went well beyond defining the cause of her symptoms and curing them, although the treatment of these symptoms superficially remained the currency used in their interpersonal exchange.

Anna O., in turn, was in all likelihood captivated by Breuer. Instead of taking refuge almost exclusively in her "private theatre," she had his company to help relieve the tedium and boredom of her confined life. The myriad of symptoms she developed justified constant medical attention and absolved her of guilt for withdrawing from nursing her dying father. The form of treatment she invented was well designed to keep her physician engaged and to make him hopeful of achieving a complete cure.

It is noteworthy that Anna O. would engage in the symptom-relieving treatment only with Breuer. He stated (1) that while he was away on vacation "no 'talking cure' had been carried out, for it was impossible to persuade her to confide what she had to say to anyone but me." On returning from vacation, he found his patient "in a wretched moral state, inert, unamenable, ill-tempered, even malicious." Failing to recognize that she was angry at him for leaving (deserting) her, he attributed her reaction to a "physical stimulus" that had not been discharged. He arranged for her to be brought back to Vienna from the country for a week so that she could tell him three to five stories each evening. This special attention was effective, but Breuer attributed the change in the patient to her narrations under hypnosis. Breuer continued to focus on the effect produced by catharsis, while the influence of the relationship between patient and physician went unnoticed.

Anna O.'s relationship to Breuer also became clear in his description of events that occurred shortly after her father's death (1).

> I was the only person whom she always recognized when I came in; so long as I was talking to her she was always in contact with things and lively. . . . She had eaten extremely little previously, but now she refused nourishment altogether. However, she allowed me to feed her, so that she very soon began to take more food. (pp. 26, 27)

At no point did the nature of the relationship between Anna O. and Breuer become clearer than at the time of the termination of treatment. According to Strachey (6), "The patient suddenly made manifest to Breuer the presence of a strong unanalyzed positive transference of an unmistakably sexual nature." This young woman in whom it was said "the element of sexuality was astonishingly undeveloped" (1) was seen to be, as Jones (4) described it, "in the throes of an hysterical childbirth [pseudocyesis], the logical termination of a phantom pregnancy that had been invisibly developing in response to Breuer's ministrations."

Freud (7) in writing about the event in a letter to Stefan Zweig, stated,

> On the evening of the day when all her [Anna O.'s] symptoms had been disposed of, he [Breuer] was summoned to the patient again, found her confused and writhing in abdominal cramps. Asked what was wrong with her, she replied: "Now Dr. B's child is coming!". . . . Seized by conventional horror he took flight and abandoned the patient to a colleague. For months afterwards she struggled to regain her health in a sanatorium. (pp. 412–413)

The evidence seems conclusive that Anna O., unbeknown to her physician, had continued to daydream—to hold performances in her "private theatre." There was, however, an important change in the cast; her physician now played the male lead.

ANNA O.'S SOLUTION

Breuer (1) noted that Anna O., even during her illness, "was greatly assisted by being able to look after a number of poor, sick people, for she was thus able to satisfy a powerful instinct." In 1895 Bertha Pappenheim accepted a position well suited to her talents and her emotional needs; she became the housemother of a Jewish orphanage in Frankfurt-am-Main (8). No longer did she have to live her life largely in fantasy. The woman who had been placed in an unacceptable position as a female could now turn her energies to the care of children and campaign for the rights of women. Had she not found these outlets for her strength, ideas, and gifts, she might have fallen ill again (9).

Not only did Bertha Pappenheim's work, at first with orphans and later with wayward girls, bring her to the fore as a social worker, but her writings marked her as a feminist leader. In 1899 she translated Mary Wollstonecraft's book, *A Vindication of the Rights of Women* (1792), from English into German. The book was a plea for the education of women. In writing about the Jewish problem in Galicia the following year, her chief point was that "the intellectual education of girls was being neglected" (8).

In 1899 Bertha Pappenheim wrote a soap opera-style but revealing play entitled "Women's Rights." As Karpe (10) noted, the play focused on the political, economic, and sexual exploitation of women. In the third and final act, a lawyer's wife persuades her husband to accompany her to the apartment of a poor, sick woman. As they enter the apartment, the dying woman rec-

ognizes the lawyer as her former lover, who deserted her when she became pregnant. His shocked wife tells him that because of their children she will not leave him, but that from now on she will refuse marital relations as "her woman's right." Is a theme repeated? Was this poor woman, deserted by her lover when she became pregnant, really Anna O., who, when pregnant (pseudocyesis), was deserted by Breuer?[1]

COMMENT

Limited opportunities existed in central Europe before the turn of the century for women of talent like Anna O. Her aspirations, if expressed, were not likely to be accorded a sympathetic reception. It is little wonder that she turned to fantasy. Such was her chief outlet at the time when her father became ill and, in keeping with the prevailing custom, she was called on to nurse him. For six months she cared for him before the tedium of her life as a woman and the ambivalence of her feelings toward her father as a man led to incapacitating hysterical symptoms. (Her lot must have been especially difficult to bear as she watched her brother continue his schooling.)

The intimate relationship Anna O. developed with her physician filled a void in her life. His attentions and her elaboration on them in fantasy were, in all likelihood, the basis of her response to treatment. Although it is likely that some symptoms were dispelled by catharsis and abreaction, it is unlikely that the release of dammed up or strangulated affects can explain the favorable response. It is safe to state that the nature of her relationship to Breuer was fundamental and that the dissolution of the relationship left her once again without an outlet for her pent-up feelings. Within five weeks a recurrence of symptoms required treatment, and this time she was admitted to a sanitarium, where she remained for several months (2).

Little is known about Anna O.'s life during the period between 1883 and 1895. Beginning in 1895 when she took on the role of housemother to an orphanage, she succeeded in sublimating her feelings in her work and her writings. She at last found an outlet for her talents and a cause to which she could dedicate herself.

Ellenberger (2) described the transition from the incapacitated patient, Anna O., to the effective and successful social worker and feminist, Bertha Pappenheim, as follows:

> Anna O.'s illness was the desperate struggle of an unsatisfied young woman who found no outlets for her physical and mental energies, nor for her idealistic strivings. It needed much time and effort before she succeeded in sublimating her personality into the respectable figure of a pioneer of social work, fighter for the rights of women and the welfare of her people—Bertha Pappenheim.

What role did Anna O. play in the development of modern psychotherapy? Before the invention of the talking cure, hypnosis was used mainly for suggestion and the physician was a doer. With the shift to catharsis, the physician became a listener. This step may seem trivial today, but in its time it was monumental. As a result of it, Breuer could state that his patient's life became known to him to an extent to which one person's life is seldom known to another. It also shifted the focus from biology to psychology. Instead of asking what caused the *disease* labeled hysteria, the physician now asked about the emotional antecedents of the disorder (11). Thus, Breuer's treatment of Anna O. started us on the road that would lead to definitive forms of psychotherapy. It led the way, too, to the giant steps taken by Breuer's young friend, who was so profoundly impressed when he heard about this remarkable case.

[1]This connection was suggested to me by Jane R. Weinberg, M.D.

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Relapse in Schizophrenia

BY MARVIN I. HERZ, M.D., AND CHARLES MELVILLE, PH.D.

Although schizophrenia is a chronic illness with exacerbations and remissions, there has been surprisingly little systematic study of early signs of relapse. The authors gave 145 chronic schizophrenic patients and 80 family members a structured interview regarding early signs of relapse and other information related to the relapse period. Most patients and family informants were aware of a prodromal period during which patients experienced such symptoms as having trouble sleeping, having trouble concentrating, loss of appetite, and feeling depressed. The authors discuss the implications of these findings for the treatment of chronic schizophrenic patients.

Although schizophrenia is a chronic illness often characterized by exacerbations and remissions, there has been surprisingly little systematic study of early signs of relapse and the decompensation process. In contrast, there has been a great deal of investigation of the early warning signs of dyscontrol of chronic physical illnesses, such as diabetes; it would be unheard of for physicians not to alert patients and family members to these early danger signals so that the development of serious complications can be prevented. Psychiatry may not have addressed the question of relapse in schizophrenia because in the past most patients were housed in custodial institutions and little attention was paid to fluctuations in the course of the illness. Most patients now live in the community and are visible when they relapse, especially if they become aggressive or disruptive and require rehospitalization. This paper will describe the results of a survey of 145 patients and 80 family informants who were interviewed about early signs of relapse and other information related to the relapse period.

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J. Simon, N. Gutterman, R. Kennedy, J. Kadick, and Marianne Bradley conducted the clinical interviews.

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The impetus for this study came as a result of our interest in finding ways to diminish the incidence of untoward side effects related to prolonged use of maintenance antipsychotic medication. Hogarty and associates (1) conducted a large scale study of the effects of antipsychotic agents and concluded that drugs administered during the aftercare phase of schizophrenia do not reduce symptoms but are prophylactic, i.e., raise the patient's threshold for further episodes. Hogarty and associates found that patients on medication who were later given placebo did not have a steady downhill course. Instead, most remained in remission, and their symptom patterns and severity, measured cross-sectionally, were similar to those of the drug patients; however, many of the placebo patients suddenly relapsed at some point during the 2 years of the study and had to be hospitalized or removed from the study in order to receive active drug treatment. If the major effect of antipsychotic drugs is prophylactic for most patients, perhaps drugs should be given only at the point of early relapse rather than on a maintenance schedule.

Studying early signs of relapse not only provides guidelines for initiation of medication for patients but helps psychiatrists to determine when medication should be increased in patients already on maintenance therapy. Many patients who take active drugs also relapse. Davis and associates (2) found in 23 controlled studies 20% of patients taking active drugs relapsed compared with 52% taking placebo. In order to deal with the issue of drug noncompliance, Hogarty and associates (3) compared schizophrenic patients taking oral fluphenazine with those taking intramuscular fluphenazine and found a relapse rate approaching 40% in both groups by the end of 1 year. The fact that patients relapse regardless of maintenance medication indicates a definite need to study early signs of relapse.

Our search of the literature revealed little data regarding early signs of relapse in schizophrenia or the length of the prodromal period. The length of the prodromal period is important if efforts at early intervention to prevent full relapse are to be successful. Docherty and associates (4), who recently reviewed the literature on stages of onset of schizophrenic psychosis, stated that despite its obvious importance, there are very few hard data available on the decompensation process. Most studies have been carried out by gifted clinicians whose usual approach is a detailed

case study, in which information is gathered retrospectively from patients and family members. Donlon and Blacker (5) took a different approach; they interviewed schizophrenic patients who were being withdrawn from medication and were beginning to relapse. Their observations, however, did not involve systematic ratings and their data were reported anecdotally. In their review Docherty and associates (4) stated that despite the different methods used to study the question, there was a concordance in the descriptions of identifiable premonitory signs as well as of regular and sequential unfolding of psychological states before psychotic breakdown. They differentiated five stages in schizophrenic decompensation, the first two stages of which are nonpsychotic:

1. *Overextension*. During this phase the person begins to experience a sense of being overwhelmed.
2. *Restricted consciousness*. During this phase a variety of mental phenomena appear that seem to bring about a limitation of the person's range of thought.
3. *Disinhibition*. During this phase relatively unmodulated impulse expression appears. This period may bear a close resemblance to hypomania.
4. *Psychotic disorganization*. The three subphases are destructuring of the external world (with increasing perceptual and cognitive disorganization), destructuring of self (with loss of self-identity), and total fragmentation (with complete loss of self and control).
5. *Psychotic resolution*. Less anxiety and psychotic reorganization, either of a delusional system or massive denial of unpleasant affect and responsibility.

METHOD

Since the literature is largely anecdotal, we decided to study systematically a large sample of patients from two locations over a 2-year period. In Atlanta (group A) outpatients (N=99) and family informants (N=80) at two community mental health centers were interviewed. Later, in an attempt to obtain data closer to the time of relapse we added group B, which included 46 hospitalized patients in Buffalo who had recently experienced a psychotic episode.

All patients were diagnosed as schizophrenic according to *DSM-II* and had no secondary diagnosis. The outpatients were being maintained on antipsychotic medication at the time of interview. Most patients had been hospitalized at least two or three times. At the time of the study, outpatients were considered to be functioning at a stable level and inpatients had recompensated sufficiently to cooperate with the research interviewer.

We collected demographic information from both groups. Group A was primarily suburban or rural and white; 67% were women and 33% were men. Their average age was 38 years and their average level of education was 9.8 years. The level of employment was low—only 15% were employed and 85% were unemployed. Only 33% were married, 21% were divorced or separated, 6% were widowed, and 40% had never been married. Approximately 73% had been hospitalized three or more times.

Group B was largely urban. Of the 46 patients, 70% were women and 30% were men; 68% were white, 28% black, and 4% Puerto Rican. Their average age was 38 years and average level of education was 9.2 years. The level of employment was somewhat higher than that of group A—26% were employed, 67.5% were unemployed, and for 6.5% data were unavailable. Most patients were unmarried: 23.9% were married, 30.4% were divorced or separated, 6.5% were widowed, 37% had never been married, and for 2.2% data were unavailable. Information on patients' living arrangements was collected only from group B. Only 17% lived alone, 70% lived with either a spouse, child, or parents, and 9% lived in a supervised facility; for 4% information was unavailable. More than half of the patients in group B were involuntary hospital admissions.

We constructed a structured interview, with a few open-ended questions, based on items contained in the Psychiatric Status Schedule (6), Research Diagnostic Criteria (7), and on clinical experience. The questionnaire, modified in Buffalo on the basis of experience gained during the Atlanta study, contained 52 items describing symptoms and feelings and other questions relevant to the period of relapse. An interview normally lasted from 30 to 45 minutes.

Each patient was interviewed individually by a research member of the staff, after being asked if he or she was willing to answer questions about his or her illness. The patient was informed that the information obtained would be included in the patient's chart and used in treatment planning. The same interview was conducted with available family members. In Buffalo the relative sample was very small because of difficulties arranging appointments; therefore, these data are not included and family data are reported for the Atlanta group only.

RESULTS

In response to the question, "Could you tell that there were any changes in your thoughts, feelings, or behaviors that might have led you to believe you were becoming sick and might have to go to the hospital?" approximately 70% of patients in both groups noticed changes and 30% did not. Families were much more likely to notice changes in the patients (92.6%) than were the patients themselves. In about 66% of the cases, the patient and the family informant agreed that there were recognizable early signs of decompensation, indicating a high level of concordance.

The time interval between the beginning of symp-

TABLE 1
Time Between Perception of Symptoms and Hospitalization in Schizophrenic Patients

Time	Buffalo Group (N=46)		Atlanta Group			
			Patients (N=99)		Families (N=80)	
	N	%	N	%	N	%
<1 day	3	6.5	8	8.1	9	11.2
1-3 days	7	15.2	8	8.1	6	7.5
4 days-1 week	4	8.7	14	14.1	7	8.8
1-2 weeks	1	2.2	8	8.1	16	20.0
2-4 weeks	9	19.6	16	16.2	3	3.7
>1 month	14	30.4	23	23.2	35	43.8
Unknown	8	17.4	22	22.2	4	5.0

toms and the need for hospitalization is extremely important if medication and crisis intervention techniques are to be used to help prevent full relapse. According to the results of our interviews (table 1), only 7%–8% of patients and 11% of family members stated that the time period was less than 1 day. Approximately 15% of group B patients and 8% of group A patients and their families noticed a prodromal period of only 1–3 days. Most of the patients and family members, however, said that it takes more than a week—52% of group B patients, 48% of group A patients, and 68% of group A families. Thus, for most patients, relapse is not abrupt and sudden, and it is possible to institute effective crisis treatment to prevent full relapse.

When asked, "What did you do about these changes when you realized that they might become serious?" almost 50% of patients responded that they sought professional help and 60% of the relatives said that they arranged for professional help for the patient. Approximately 33% of group B patients and 26% of group A patients reported that they did nothing. Since antipsychotic medication is especially effective in treating acute symptoms, it is disconcerting that less than 4% of all patients and family members said that patients took more medication when they realized that symptoms were becoming serious.

Approximately 24% (N=24) of group A patients and 33% (N=15) of group B patients claimed that no medication had been prescribed for them, but it is possible that some of them chose simply to ignore their prescriptions. Only 26% of group B patients (N=12) and 50% of group A (N=49) patients stated that they were taking medication as prescribed. The primary reason given by patients for not taking medication as prescribed was that it did not help (group A, N=12; group B, N=6). The next most commonly mentioned reason was that it had unpleasant side effects (group A, N=2; group B, N=6). If withdrawal of medication does not affect symptoms adversely for most stabilized patients, they might conclude that the medication was not helping and therefore was not necessary. In fact, there are reports of an increased energy level and sense of well being when medication is terminated (8). For some patients this may be due to an amelioration of an unrecognized extrapyramidal side effect, akinesia (9, 10).

We assessed the importance of precipitating events by asking patients if anything occurred in their lives that might have led them to become upset. Approximately 40% of the patients and family members did not know of any such event or believed that biological factors were responsible. Approximately 33% of the patients and relatives were able to relate the most recent episode to a stressful life event such as loss of a significant other, loss of a job, marital problems, etc. Only about 2% of patients attributed relapse to stopping or incorrectly using their medication. Other reasons given by patients and relatives were taking alcohol or drugs and feeling socially isolated. Davis and associates (11) reported that a large number of patients are not aware of a precipitating life event; however, a prospective study conducted during time of early relapse might reveal events important to the patient that are later denied, repressed, overlooked, or forgotten, such as changes in the emotional climate of the family along dimensions described by Vaughn and Leff (12). Patients are liable to relapse when there is a good deal of criticism or overprotectiveness by family members.

What were the symptoms that either appeared for the first time or worsened in the prodromal period before full relapse and hospitalization? The Atlanta patients were outpatients in a relatively stable condition and the Buffalo patients had recently been hospitalized and were recovering from an acute episode. In spite of these and other differences between the two groups, the rank-order correlation of symptoms that appeared or worsened before hospitalization between the Buffalo and Atlanta patients was .85 ($p<.001$) (table 2). Becoming tense and nervous was the symptom reported by most patients in both groups. Generally, the symptoms most frequently mentioned by patients and family members were symptoms of dysphoria that nonpsychotic individuals experience under stress, such as eating less, having trouble concentrating, having trouble sleeping, depression, and seeing friends less. Among the first 10 most commonly noted psychotic symptoms were feelings of being laughed at or talked about and possibly increased religious thinking.

The rank-order correlation of frequency of symptoms reported by Atlanta patients and their relatives was also very high ($r=.78$, $p<.001$). Some relatives who were sensitive to the patients might notice dysphoric signs before the patient did, although insensitive relatives who were not in touch with the patient's feelings or behavior might tend not to notice early symptoms and rate more extreme psychopathology high. The only psychotic symptom for which there seemed to be a large degree of disagreement was talking in a nonsensical way, which was ranked 3rd by families and 13th by patients. Relatives ranked hearing voices and seeing things 10th and

TABLE 2
Symptoms That Appeared or Worsened Before Hospitalization in Schizophrenic Patients[a]

	Buffalo Group (N=46)		Atlanta Group			
			Patients (N=99)		Families (N=80)	
Symptom	Rank	%	Rank	%	Rank	%
Tense and nervous	1	80.4	1	70.7	1	83.3
Eating less	2	71.7	10	49.5	17	52.5
Trouble concentrating	3	69.6	5	56.6	3	76.3
Trouble sleeping	4	67.4	3	61.6	7	68.8
Enjoy things less	5	65.2	8	52.5	8	67.5
Restlessness	6	63.0	4	58.6	2	78.8
Can't remember things	6	63.0	14	46.5	10	60.0
Depression	8	60.9	2	63.6	3	76.3
Preoccupied with one or two things	9	59.6	12	48.5	9	65.0
Seeing friends less	9	59.6	7	54.5	18	50.0
Am being laughed at, talked about	9	59.6	9	51.5	14	53.8
Loss of interest in things	12	56.5	5	56.5	6	73.8
More religious thinking	13	54.3	10	49.5	19	47.5
Feeling bad for no reason	13	54.3	19	40.4	22	37.5
Feeling too excited	15	52.2	25	30.3	14	53.8
Hear voices, see things	16	50.0	17	42.4	10	60.0
Feeling worthless	17	48.8	15	44.5	12	56.3
Talking in nonsensical way	18	45.6	13	47.5	3	76.3
Believe someone else is controlling	18	45.6	18	41.4	21	38.8
Bad dreams	18	45.6	21	38.4	23	33.8
Too aggressive (pushy)	18	45.6	24	32.3	20	40.0
Feeling angry at little things	22	41.3	22	33.3	14	53.8
Not caring about way I look	22	41.3	19	40.4	12	56.3
Having trouble with spouse, mate	24	30.4	26	29.3	29	20.0
Thoughts of hurting, killing self	24	30.4	22	33.3	26	31.3
Frequent aches, pains	26	28.3	27	28.3	23	33.8
Fear of "going crazy"	26	28.3	15	44.5	23	33.8
Thoughts of hurting, killing others	28	23.9	29	10.1	27	27.5
Drinking more, using drugs	29	21.7	28	20.2	28	25.0

[a]Spearman rank-order correlation for Buffalo patients and Atlanta patients = .85 and for Atlanta patients and families = .78 ($p<.001$).

patients ranked it 17th. These differences could also be related to patients' denial of psychotic symptoms.

When patients were asked, "What were the first changes in thoughts, feelings, or behavior that might have led you to believe that you were becoming sick and might have to go to the hospital?" there was little agreement. Among the symptoms mentioned were tension, anxiety, nervousness, insomnia, and depression. Hearing voices was mentioned by 5 group B patients and 8 group A patients as the first change noticed.

When asked whether the pattern of development of symptoms was similar for each episode, 50% of the patients answered "yes" and 50% answered "no." Whether or not individual patients have the same sequential pattern of symptoms each time they relapse is of great importance to the psychiatrist who plans the treatment strategy.

Only Buffalo patients were asked, "Can you tell me which of the following symptoms are present when you are feeling well?" Sixty percent stated that they had feelings of loneliness. This seems high considering that many of these patients lived with a relative. A large number of neurotic symptoms, such as tension and nervousness, feeling sad or blue, and worrying were reported by 48% of patients. A fewer number of psychotic symptoms, such as thought broadcast (reported by 30%) and hearing voices (reported by 26%), were reported during a state of presumed stability. Astrachan and associates (13) reported similar findings after interviewing schizophrenic patients 2–3 years after hospital discharge.

CONCLUSIONS

Creer and Wing in England (14) took a survey of 80 relatives of schizophrenic patients which revealed that virtually none of the relatives had received any sensible advice about the nature of the condition, about how to supervise medication, about the likely outcome of treatment, or about how to best respond to disturbed or disturbing behavior. Usually when a family member has a chronic physical disability, such as paraplegia or renal failure, management is worked out in detail and patients and relatives are taught to predict and prevent many problems and to know what to do in time of crisis. We recommend that the treatment of every schizophrenic patient include a major focus on teaching the patient and family not only to recognize early signs of decompensation but also what steps to take to ensure the initiation of prompt and effective treatment. Because the prodromal period usually lasts

more than a few days, it should be possible to abort an incipient acute psychotic episode. It may even be possible in some well-stabilized patients who are cooperative in treatment to use antipsychotic medication only when there are early signs of relapse rather than the usual practice of maintaining such patients on drugs indefinitely. In either case, patients and families should be educated about the special value of antipsychotic medication during the relapse period.

Our finding that there is usually a nonpsychotic prodromal period confirms Docherty and associates' results (4). Our patients, however, did not report two distinct stages of symptoms during the prepsychotic phase of relapse described by Docherty and associates. Whether these prepsychotic stages have general validity could be determined from prospective studies while the symptomatology is unfolding. It may be that for the usual chronic schizophrenic patient who has little insight these stages are not recognizable. Further research is needed in order to differentiate when the appearance of nonpsychotic symptoms signals an impending relapse and when it does not.

Our data on relapse in schizophrenic patients are based only on retrospective reports by patients and families; however, the data reported by Atlanta patients and their families were later substantiated by reports from a second sample of patients in Buffalo. Future research in this area should be prospective rather than retrospective and should use patient and family interviews as well as psychophysiological and biochemical measures.

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Male Rape: Offenders and Victims

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In an analysis of 22 cases of male rape in a community setting, the gender of the victim did not appear to be of primary importance to some of the rapists, but for others, males appeared to be specific intended targets, and the rapists' assaults were an effort to deal with unresolved and conflictual aspects of their lives. For all offenders the sexual assault was an act of retaliation, an expression of power, and an assertion of their strength and manhood. The impact of rape on the male victims was similar to that on female victims, disrupting their biopsychosocial functioning; however, male rape appears to be underreported due to the stigma associated with it.

Although the professional literature has increasingly focused on the clinical dynamics of rapists (1, 2) and the trauma of victims (3, 4), investigators have almost exclusively studied offenses committed against women and children. Except for a few articles on the sexual assault of male inmates in a prison setting (5, 6), little attention has been given to the forcible sexual assault of adult males (7).

Police statistics would appear to support this impression. For example, in Boston in 1976, of 355 reported rapes, only 4 male victims (1%) were 17 years or older.[1] In addition, a survey of the Philadelphia Women Organized Against Rape crisis center revealed that of the 382 client contacts over an 8-month period in 1977, 22 (6%) of the victims were male, but of these only 1 (0.3%) was over the age of 16.[2]

In our clinical work we have had access to a number of male offenders who raped adult males or male agemates and to a number of male victims of sexual assault. Although we are reporting on a small sample and thus cannot generalize any findings, we believe the data provide observations of interest to professionals. This paper focuses on 1) descriptive characteristics of the offense, the dynamics of the offender, and the impact on the victim, and 2) similarities and differences between male and female rape.

METHOD

Data were obtained from a total of 22 subjects—16 males who admitted having sexually assaulted other males and 6 additonal males who identified themselves as victims of rape. The rapists were convicted offenders referred for evaluation to the Center for the Diagnosis and Treatment of Sexually Dangerous Persons in Bridgewater, Mass.; the Whiting Forensic Institute in Middletown, Conn.; the Harrington Hospital Forensic Mental Health Program in Southbridge, Mass.; or the Connecticut Correctional Institution in Somers, Conn. The victims were either self-referred or referred to us by police or hospital personnel. None of the offenders was convicted of the sexual assault. This study's offender and victim samples, then, were independent and unrelated.

Information about the nature of the offenses, the dynamics of the assailants, and the reactions of the victims was retrieved and recorded through interviews with the subjects. In addition, their available clinical records were examined. Informed consent was obtained from all of the subjects, and none refused to participate in this investigative study.

Characteristics of the Offenders

The offenders ranged in age from 12 to 41 years. Three were juveniles between the ages of 12 and 16, and the majority (N=13; 81%) were young adult males; the average age was 24. These 16 offenders sexually assaulted a total of 24 victims, who ranged in age from 10 to 30 (average age=17½).

Most of the offenders (N=13; 81%) and their victims (N=23; 96%) were white. Three offenders and 1 victim were black. Only 2 assaults, involving a black offender and a white victim, were interracial.

The majority of the offenders (N=12; 75%) were total strangers to their victims. Three of the offenders knew the victims casually, and 1 offender was the vic-

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[1] P. Sadler, personal communication, Dec. 24, 1976.

[2] B. Lakey, Women Organized Against Rape data report. Personal communication, Dec. 21, 1977.

tim's brother. Four offenders had codefendants; the other 12 had acted alone.

At the time of their offense all of the men in the offender sample were actively engaged in consenting sexual encounters or relationships. Eight men (50%), 4 of whom were married, confined their consenting sexual activity to members of the opposite sex. Six offenders (38%), 2 of whom were married, engaged in consenting sexual encounters with both men and women; 3 of these men were extremely conflicted over their sexual encounters with other males. Two of the men in this sample confined their consenting sexual encounters almost exclusively to other men, but neither was committed to a permanent relationship.

Characteristics of the Victims

The 6 victims interviewed in this study ranged in age from 16 to 28 years (average age=17½ years). They were raped by men ranging in age from 16 to 30 years (average age=22 years). All of the victims were white, as were all but 1 of their assailants. Half of these subjects were sexually assaulted by strangers. One was raped by a casual acquaintance, and 2 by close friends (in one case the offender was a classmate; in the other he was the victim's employer). One subject was gang raped by 3 men; the rest were each sexually assaulted by 1 man.

Three of the victims had a heterosexual lifestyle; 1 was bisexually oriented; 1 was homosexually oriented; and 1 had not been sexually active before his victimization but subsequently adopted a homosexual lifestyle.

CLINICAL OBSERVATIONS

Combined data on 22 cases of male rape retrieved from both the offender and victim samples were analyzed in regard to six major factors: access to the victim, style of attack, type of sex acts committed, motivational components of the assault, history of sexual assault, and impact on the victim.

Access to the Victim

Men are assaulted where they live, work, travel, and relax. They appear to be at greater risk when engaged in solitary activities, such as hitchhiking. In 8 of the cases (36%) the victim was hitchhiking and the offender gained access to him by offering him a ride. In 1 additional case the victim was a passenger in the offender's car. In 8 cases (36%) the victim was engaged in an out-of-doors activity, such as swimming at a beach or hiking in the woods, when the offender approached him, and in 2 instances the victims were walking along the street, headed for a specific destination. Two other victims were in the offender's home, and 1 victim was a customer in the bar where his offender worked.

Style of Attack

The offender gained sexual control of his victim through three major methods: entrapment, intimidation, and/or physical force. In 3 cases the offenders entrapped their victims by first getting them drunk and then taking advantage of the victim's intoxicated state to sexually assault him.

> One victim, Mr. A, was at an office party and had too much to drink. His boss offered to drive him home and passed out in the car. A short time later he awoke to find himself naked on the backseat being sodomized by his boss.

A more common style of attack, however, was through some type of intimidation. In 12 cases the offenders intimidated their victims into submission by threats of physical harm and/or by brandishing a weapon.

> Mr. B was hitchhiking and was offered a ride by four men. He got into the back seat of the car, and as they drove off one of the men put a knife to Todd's throat and forced him to perform fellatio.

An equally frequent approach was an unanticipated attack in which the offender approached the victim by suddenly striking or physically overpowering him.

> Mr. C was sleeping in his barracks when he was awakened by a man who punched him in the chest and stomach and choked him. He then held Mr. C's face down on the bed and sodomized him.

Type of Sex Acts

In little more than half of the cases (N=12; 55%) only a single type of sexual activity occurred during the offense, and in a little less than half of the cases (N=10; 45%) several types of sexual acts (oral, anal, and masturbatory) took place. In the majority of the assaults (N=14; 64%) the offender sexually penetrated his victim in some fashion, whereas the offender accepted penetration (that is, he performed fellatio on his victim) in only 7 of the cases (32%). The most common type of sexual act was sodomy; in 10 of the assaults the offender performed anal intercourse on his victim. Oral penetration was the next most frequent act; in 8 of the cases (36%) the victim was forced to perform fellatio on his assailant. In 2 of the cases the offender made one victim perform sexual acts on another victim. One case consisted of the victim masturbating the offender, and in another case the offender used a foreign object to anally penetrate the victim.

What seems especially significant is that in half of the cases the offender made an effort to get his victim to ejaculate. Two offenders masturbated their victims, 7 performed fellatio on their victims, and 2 forced co-victims to fellate each other to the point of ejaculation.

Motivational Components

The motivational intent of the offender was analyzed through the accounts and details of the offense. Five major psychological components were prominent.

1. *Conquest and control.* All assaults served as an expression of power and mastery on the part of the offender.

> Afterwards he had tears in his eyes and said something like, "I hope you're satisfied," or, "I hope you got what you wanted," and I felt kind of funny because he was so upset. What was really exciting, though, was that all during the assault I felt in total control of him.

2. *Revenge and retaliation.* In some cases of male rape, the offense is activated by the assailant's anger toward his victim and is regarded by him as a form of retaliation.

> I cornered him in the showers and said, "You think I'm a punk? I'm going to prove you are what you called me." He was real scared and said, "Forget about what I said," and then he asked me if I wanted a hand-job. I said, "What do you think I am, a homo?" Then I told him, "You're going to give me some ass," and I fucked him. It wasn't for sex. I was mad and I wanted to prove who I was and what he was.

3. *Sadism and degradation.* For some assailants, aggression itself becomes eroticized, and they find excitement and gratification in the sexual abuse and degradation of their victim.

> I had the guy so frightened I could have made him do anything I wanted. I didn't have an erection. I wasn't really interested in sex. I felt powerful, and hurting him excited me. Making him suck me was more to degrade him than for my physical satisfaction.

Sexual sadism may be evidenced by bondage, ritualistic torture, and coprophilic acts. In extreme cases it may lead to lust murder, but for a variety of reasons such male homicides may not be identified as sex-related.

4. *Conflict and counteraction.* Another component in some male rapes is the assailant's attempt to punish the victim as a way of dealing with his unresolved and conflictual sexual interests.

> Mr. D, aged 20, drove around a city block where male hustlers cruised and picked out a man about his own age. They drove to a roadside rest area and parked. "After I came, I dragged him out of the car and punched him out and called him a punk. I told him I was going to kill him. Then I threw his clothes out of the car and took off. I was angry at him. I don't know why. At what I was doing, I guess, is what I was really angry at."

Such offenders seem caught in a bind: unable, on one hand, to admit their interests in sexual encounters with other males and unable, on the other hand, to abandon pursuing such encounters. They do not feel comfortable or secure with themselves in regard to their sexual identity or orientation. In some cases they project their feelings onto the victim and see him as a temptation. Then they become angry and attempt to punish him for arousing such interests or feelings in them.

5. *Status and affiliation.* Some offenders feel pressured to participate in gang rape to maintain status and membership with their peers. In addition to other motives, acceptance and recognition by one's peers becomes a dynamic in group rape, and mutual participation in the assault serves to strengthen and confirm the social bond among the assailants.

History of Sexual Assault

The current offense constituted the first conviction for a sexual assault for only 4 of the 16 offenders. (No dependable data were available in regard to the assailants of the victims interviewed). Of the 12 offenders who had a prior criminal record of sexual offenses, 6 (38%) had previously committed sexual assaults against other males; 4 had committed sexual assaults against females and 2 had sexually assaulted both males and females. Seven of the subjects with prior offenses had sexually assaulted age-mates, 4 adult subjects had assaulted adolescents, and 1 adult subject had assaulted a child. Apart from their sexual offenses, 9 of the offenders (56%) had no criminal record; 7 (44%) had a serious criminal history in addition to their sexual offenses.

Impact on the Victim

Male victims make it clear that 1) rape is stressful, 2) reporting the assault is difficult, and 3) one's lifestyle becomes disrupted.

That rape is often life-threatening to male victims is validated through the terminology used to describe the rape ("I was terrified . . . thought he was going to do me in"). Victims experience a wide range of mixed emotions and disbelief ("I was flabbergasted and didn't know what to do"; "I wanted to kill the guy but in the same token was afraid").

The male victims felt pressured into not reporting the rape for several reasons: 1) societal beliefs that a man is expected to be able to defend himself against sexual assault ("I don't think a lot of people believe it could happen . . . I'm 6'2" and weigh 220 pounds"), 2) the victim's sexuality may become suspect ("The police asked me a lot of wild questions . . . reporting puts your masculinity in jeopardy"), and 3) telling is distressing ("It's embarrassing to tell someone you've been raped").

The disruption in male victims' lifestyle could be seen in terms of physical reactions ("I couldn't eat or sleep for a while"), psychological reactions ("I think a lot about it by myself"; "I had a fear of men for quite a

while"), social reactions ("My friends kept asking what happened and I just felt they didn't believe me and I just got away from having friends"; "Didn't tell my family. They are conservative and who knows what they would think"), and sexual changes ("My sexual activity . . . ended").

Although male victims made concerted efforts to resume their usual lifestyle as quickly as possible ("I tried to get all organized . . . get to class . . . get everything done so I don't get totally behind"), on long-term follow-up they reported unresolved issues when the rape was not adequately addressed as a trauma at the time it occurred.

> Even a couple of years after, whenever I'd feel depressed or tired or something, this whole incident would come back to me. I'd think . . . what could I have done to prevent it?

Male victims also seemed to evidence considerable anger at having been raped and fantasized or planned retaliation against their assailants.

> I kept trying to figure out how I could get back at him. I thought of getting a gun and going out to revenge myself.

In those cases in which the offender attempted or succeeded in getting his victim to ejaculate, the victim was especially confused and concerned about this aspect of the assault.

> I always thought a guy couldn't get hard if he was scared, and when this guy took me off it really messed up my mind. I thought maybe something was wrong with me. I didn't know what it meant and this really bothered me.

DISCUSSION

Since rape is commonly misconstrued to be a sexually motivated crime, it is generally assumed that males are unlikely targets of such victimization, and that when it does occur, it reflects a homosexual orientation on the part of the offender. However, the cases of male rape that we have had an opportunity to study do not lend much support to either assumption. The gender of the victim did not appear to be of specific significance to half of the subjects. They appeared, instead, to be relatively indiscriminate with regard to choice of victim, that is, their victims included males and females, adults and children. This would tend to suggest an undifferentiated, or polysexual orientation. For the other half of these subjects, males appeared to be the specific targets of their sexual offenses. All of these offenders appeared to be sexually active with other males, but only 2 of them led predominantly homosexual lifestyles, in which the selection of a male victim would constitute a counterpart to the selection of a female victim by a heterosexual rapist. The other 4 offenders led bisexual lives but were conflicted about their sexual involvement with other males. The selection of a male as the target of their sexual assault can be seen in part as an expression of this unresolved aspect of their lives. The victim may symbolize what they want to control, punish, and/or destroy; something they want to conquer and defeat. The assault is an act of retaliation, an expression of power, and an assertion of their strength or manhood.

The selection of a male as a victim seemed even less significant than the choice of sex as the means of domination and retribution. Rape is a complex, multidetermined act prompted more by nonsexual than by sexual motives (2). There are several close parallels between cases of male rape and female rape. The age of the offender is quite similar in both types of rape, for example. Adolescence and young adulthood appear to be times of stress that activate the potential in some males who are prone to rape.

Although it is commonly believed that a male is powerful enough to defend himself from a sexual assault, he is in fact susceptible to the same techniques by which assailants gain control over their female victims. In many cases a combination of entrapment, intimidation, and brute strength were employed in the commission of the assault.

A major strategy used by some offenders in the assault of males is to get the victim to ejaculate. This effort may serve several purposes. In misidentifying ejaculation with orgasm, the victim may be bewildered by his physiological response to the offense and thus discouraged from reporting the assault for fear his sexuality may become suspect. Such a reaction may serve to impeach his credibility in trial testimony and discredit his allegation of nonconsent. To the offender, such a reaction may symbolize his ultimate and complete sexual control over his victim's body and confirm his fantasy that the victim really wanted and enjoyed the rape. This fantasy is also prominent in the rape of females (8).

As in the case of sexual assault of females, male rape (whether it occurs in the community or in an institutional setting) does not appear to be motivated primarily by the need for sexual gratification. Satisfaction and pleasure in male rape appear to be experienced in the sense of power, the discharge of anger, and the erotization of aggression more than in sexual release. Offenders equate manhood with being in control, being aggressive, and being the penetrator in sexual acts. Offenders differ in their need for control. Some offenders wish to control, others to control and to degrade, still others to control, degrade, and destroy. Sex becomes an instrument of control, retaliation, degradation, and punishment.

In conclusion, although we would not expect the incidence of male rape to approach that of female rape in our culture, we believe it is not so rare an event as would appear from reported incidents. We believe

existing stereotypes are responsible for males being reluctant to report rape, much as the attitude and atmosphere existed for female rape victims a decade ago. Thus, we believe it is important for clinicians to be especially alert to suspected sexual trauma in males of all ages and to be sensitive to such victimization when it is reported.

The average age for the victims referred and interviewed in this study was identical to that of the victims of the offenders interviewed. Both were 17½, an age that represents a transition from adolescence into adulthood. We suspect that as males become older they become increasingly reluctant to acknowledge sexual victimization. Although some states have revised their criminal codes in regard to rape to include male victims, other states have not, and such assaults do not constitute rape but are dealt with as sodomy offenses. According to most such statutes, both participants in an act of sodomy are guilty of "an unnatural act" or "a crime against nature," and although the victim may persuade others that the sexual encounter was forced rather than voluntary, such an admission may lead others and the victim to feel that his manhood has been tampered with. If the victim is gay, his sexual orientation may impeach his credibility in regard to his victimization. Although federal guidelines have precluded the introduction of the female victim's sexual reputation as a defense in rape cases, the sexual lifestyle of the male victim can still be used as a defense (9). As a result, then, of a combination of cultural, social, legal, and psychological issues, male rape remains one of the most unaddressed issues in our society today.

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The Concept of Culture in Psychiatric Residency Education

BY EDWARD F. FOULKS, M.D., PH.D.

Diagnosis and treatment in psychiatry are heavily influenced by cultural factors. The author argues that the subject of culture warrants a place in the curriculum of psychiatric residency education. He outlines approaches to the teaching of cultural psychiatry that have been used successfully in several departments of psychiatry. He feels that although the medical model of understanding mental illness has demonstrated its effectiveness in chemotherapeutic, psychotherapeutic, and other treatments, the cultural model can often provide unique insights and approaches in patient care.

More than in other branches of medicine, diagnosis and treatment in psychiatry are never free from cultural factors. Clinical judgments, especially in psychotherapy, are never free from the influence of the therapist's cultural values. When the patient is from the same social class and ethnic group as the treating psychiatrist, therapy is often successful. Problems are encountered, however, when the psychiatrist attempts to use the standards of his or her background to evaluate and treat patients from a different background. Psychiatry, and especially psychotherapy, can never be completely free of such biases. At best, psychiatrists can attempt to develop sensitivity to and awareness of cultural differences between themselves and certain patients and in this respect to develop an appreciation of the patient's point of view. Insight into another's cultural background often provides therapeutic approaches that reduce the patient's misunderstandings and enhance a working doctor-patient relationship. Psychiatrists often obtain knowledge of and sensitivity to other cultures in their undergraduate education; sometimes these develop through personal experiences. In this paper I will argue that, in addition, the subject of culture warrants a place in the curriculum of psychiatric residency education.

Culture is an important factor in many aspects of psychiatric practice and theory; yet faculty who are involved in psychiatric education have struggled with designing courses in cultural psychiatry that would prove relevant to the clinical experiences of residents and students. In this paper I will outline approaches to the teaching of cultural psychiatry that have been used successfully in several departments of psychiatry. The purpose of presenting this outline is twofold: 1) to disseminate ideas regarding unique training approaches in cultural psychiatry and 2) to suggest an approach to the development of a sequential core curriculum in cultural psychiatry that would apply to all four years of psychiatric residency education.

Until recently, anthropology has been only peripherally related to the practice of psychiatry and has accordingly played a small but interesting, if not exotic, role in education programs for residents. Residents' seminars centering on, for example, speculations regarding the function of circumcision in primitive societies and its relation to the Oedipus complex have provided intellectual delight to many programs. With the advent of the community mental health movement, many psychiatry trainees faced problems of dealing clinically with patients whose cultural background was different from their own. In this situation the relevance of understanding cultural values, beliefs about the causes of and cures for illness, differences in status, working in an intercultural setting, and the role of the psychiatrist-physician in relation to other kinds of healers became major problems of clinical importance. Cultural psychiatry, however, is not limited or relevant only to residency education in community mental health centers serving ethnically diverse groups; it is fundamental in the training of general psychiatrists working in any setting.

Increasing efforts have been made recently in psychiatric residency education to develop an explicit curriculum. In this regard, the work of the American Association of Directors of Psychiatry Residency Training, the American Medical Association's Liaison Committee on Graduate Medical Education, and other agencies has resulted in increasing uniformity in the

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curriculum and clinical experience currently offered by residency education programs. I hope that the suggestions offered in this article will provide an impetus for developing a curriculum in cultural psychiatry as an integral part of basic residency training in psychiatry.

In 1978 the American Psychiatric Association created a Task Force on Cultural and Ethnic Issues in Psychiatry under the direction of Dr. John Spiegel. This group is charged with heightening the awareness of the importance of understanding the specific cultural, national, or ethnic backgrounds represented in our culturally diverse society. The task force is to emphasize the specifics of culturally differentiated diagnostic and treatment techniques for various ethnic groups. They are attempting to generalize and correlate a multi-ethnic and culturally appropriate mental health approach with other medical specialties. This group is also charged with conducting an inventory of the curricula of residency training programs with respect to the inclusion of the subject of cultural psychiatry and to develop model courses and bibliographies for use in psychiatric residency programs in the future.[1]

The need for training in this subject has long been recognized by residency accrediting agencies. The Liaison Committee on Graduate Medical Education has clearly recommended the inclusion of cultural psychiatry in residency education:

> The curriculum must include . . . sufficient material from the social and behavioral sciences, such as Psychology, Anthropology, Sociology, to help the resident understand the importance of economic, ethnic, social, and cultural factors in mental health and mental illness. . . . [Residents] must have experience in the care of patients . . . from a wide variety of ethnic, social, and economic backgrounds. . . . The residency program should provide its residents with instruction about American culture and sub-cultures. Many physicians may not be sufficiently familiar with the attitudes, values, and social norms prevalent among the various segments of contemporary American life; therefore, the curriculum should contain instructions about these issues adequate to enable the resident to render competent care to those patients from various cultural backgrounds for whom he has clinical responsibility in the course of his residency. (1, pp. 361–362)

In spite of these recommendations, many training centers have had difficulty developing a formal approach to the teaching of cultural psychiatry. This may represent difficulties inherent in the medical model of mental illness, which is sometimes interpreted as meaning that, for example, schizophrenia is schizophrenia and depression is depression, no matter what the patient's cultural background. There is, at times, an ethnocentric or at least a self-assured idea that an accurate diagnosis determines and prescribes an inevitable treatment process. Clinical experience often demonstrates, however, that the way individuals experience, identify, interpret, and communicate psychic function is determined to a very large extent by their culture. Furthermore, when, how, and from whom they seek relief, what treatments they understand and accept, and how long and to what extent they will comply are further functions of their background and culture. The value of an accurate scientific diagnosis is diluted when a patient does not show up for a follow-up appointment or cannot take blue pills for a "cold" illness or feels that the malady is caused by being out of grace with God.

Psychiatry training programs need to devote more attention to formalizing cultural factors in patient care and management. An increasing awareness of the pluralistic nature of our society has brought many of these issues to the fore. In addition, the advent of the community psychiatry movement, with its increased awareness of minority group issues, has brought a special focus on the practical relevance of the concept of culture in providing health and mental health services to these populations.

Culture plays a major role in shaping how people think, behave, and feel. Culture determines how and by whom children are raised, how they are cuddled and fed, how they acquire rules of behavior, how they are punished, and how they learn about sex, gender roles, and marriage. Culture may affect personal psychology and shape character. Culture provides standards and values according to which one evaluates the self, one's group, and outsiders. Culture provides guidelines and rules for recognizing or diagnosing emotional illness and for its management and, sometimes, treatment. When psychiatrists raised in one culture encounter people raised in another, some conflict and misunderstanding may result regarding many issues, such as how children should be raised, rules of behavior, gender roles, marriage patterns, standards and values for evaluating self and others, and how mental illness is perceived and dealt with (2).

CURRICULUM APPROACHES

Cultural Sensitivity Groups

Sensitivity and insight into one's own attitudes, reactions, and biases toward others have always been important aspects of residency training. Many residents seek or are directed to their own therapy or personal psychoanalysis to gain a better understanding of themselves and to increase their degree of objective empathy with their patients. In a paper presented at a conference at Boston University in April 1979, Charles and Elaine Pinderhughes proposed the usefulness of cultural sensitization and introspection in psychiatric residency training through the use of special short-term sensitivity groups in which each member is given

[1]John P. Spiegel, M.D.: Personal communication, Feb. 20, 1979.

the opportunity to talk about and explore his or her own ethnic background and assumptions as well as the stereotypes attributed to other ethnic groups. Actively exploring one's own cultural heritage with its unique values often brings to the fore many attitudes toward members of other ethnic groups that had been implicit in thought and action but out of the resident's awareness. The group experience is designed to render residents more sensitive and appreciative of differences between their own culture and that of their patients and others. As is usually the case with sensitivity groups of any kind, the group leader should be from outside the institution. Groups could be formed as a special retreat or for a short time and could use a visiting consultant whose purpose would be to create awareness of class and cultural stereotyping.

Didactic Courses and Their Content

Favazza and Oman (3) outlined major works in cultural psychiatry that relate the concept of culture to the practice of psychiatry and psychiatric theory. The scope of these studies includes 1) cross-cultural comparisons, 2) emics versus etics (culturally defined within-group definitions versus categories imposed by Western science), 3) subjective culture, 4) culture and personality, 5) culture, mental health, and mental illness, 6) the culture of mental hospitals, and 7) folk beliefs and rituals, which include folk classifications and etiology of mental illnesses, folk diagnosis, folk treatments, and folk healers, and 8) psychiatric epidemiology.

In an effort to structure a course in cultural psychiatry, residency programs in Canada have developed a set of educational goals and objectives that includes the contributions of the sociocultural disciplines to psychiatry (4). The final objectives determined most relevant to residency education included the following:

1. To attain an understanding of some of the concepts of culture and the idea that cultures have arrived at a variety of greatly diverse solutions to the problems of survival and the achievement of satisfaction.
2. To learn the range and diversity of marital and family patterns across cultures and the ecological forces that may determine them.
3. To have an awareness of some of the research concepts related to the mental health aspects of culture change and migration.
4. To become familiar with some of the main concepts and terms used by sociology when considering psychiatric phenomena.
5. To attain an understanding of what social class means and how it may be related to psychiatric disorders, psychiatric treatment, and the attitudes of psychiatrists.
6. To become familiar with research findings concerning relationships between various ecological factors and differential rates and patterns of psychiatric disorders.
7. To attain an awareness of some of the findings of studies concerning the social systems of psychiatric wards and the implications of these findings for reform.
8. To have an awareness that all cultures generate psychiatric disorders but that their forms may vary from culture to culture in a way that is linked to cultural symbols, belief systems, or modal personalities.
9. To learn that the prevalence, age of onset, duration, and outcome of psychiatric disorders may vary across cultures and that variations are often best explained in cultural terms.

This outline, with accompanying enabling objectives, provides the initial framework for a basic science course in cultural psychiatry for residents. Such a course would obviously be more theoretical than clinical but would have application and relevance to the field of psychiatry. Further topics that might be developed in a basic science didactic course include medical anthropology (5), basic theoretical approaches to understanding disease and illness (6), social integration and disintegration in relation to psychiatric symptoms (7), sociocultural change, modernization, and psychiatric disorder (8), poverty and psychiatric disorder (9), stress, coping, and adaptation in relation to class and ethnicity (10), alternative healing systems (11), and cross-cultural issues in psychiatric assessment and treatment (12, 13). A focus of the course should be on comparative studies of traditional healing and its relationship to psychotherapy, indigenous drugs and the use of special states of consciousness, and cross-cultural epidemiological studies of mental illness. Cultural influences on cognition, affects, and coping processes should be studied as they influence culture-bound syndromes and manifestations of psychosis, depression, and dissociative states. Videotapes and movies of healing rituals, trance states, and shamans' performances such as those developed by Simons[2] might prove useful in this regard.

The Society for Medical Anthropology has recently published a curriculum guide of undergraduate, graduate, and medical school courses in medical anthropology (14). Training programs, in liaison with colleges or universities, could provide opportunities for residents to take courses in sociology and anthropology. Interested residents might pursue formal graduate work toward a master's degree or doctorate when this could be conveniently integrated with clinical rotations.

Field Experiences

Residents learn material best when it is closely related to clinical experience. Therefore, it would be ideal if a didactic course in cultural psychiatry were accompanied or followed by an opportunity to apply the knowledge to a clinical setting. In a paper present-

[2]Ronald P. Simons: "Floating in the Air, Followed by the Wind" (16 mm. sound-color film). Gunter Pfaff, filmmaker. Indiana University Audiovisual Services, 1974.

ed at Boston University School of Medicine in April 1978, Alexander Leighton proposed guidelines for developing clinical applications of cultural psychiatry. Such experiences are intended to provide the resident with an understanding of culture and cultural processes. Leighton's recommendations were to develop a curriculum in cultural psychiatry that would foster, among other things, learning to interview with a reluctance to make assumptions based on one's own values without collaboration from the patient and learning to be sensitive in the interview to one's own cultural assumptions that might inhibit, embarrass, or intimidate the patient.

Curriculum development should proceed first by identifying an ethnic group often encountered in a clinic situation and then reading scientific literature relevant to that group. Residents should spend time acquiring the language or idiom of that group with some fluency, or at least learning about the language. They should enter relationships and friendships with people representing the culture (for example, they could arrange to live with or visit families from the culture). Residents might also be assigned to obtain from selected members of the cultural group the story of their lives; ideally, both men and women and young and old individuals should be interviewed. Some programs have provided opportunities for interested residents to spend time working within another culture in a foreign or rural area.

Study Groups

In Philadelphia we have extended seminars throughout the entire four years of residency training by offering an elective weekly study group in cultural psychiatry. The group is composed of Anglo and Hispanic residents, staff psychiatrists, psychologists, social workers, and other therapists who work in clinical settings that serve a predominantly Puerto Rican population. Special topics related to clinical care, such as family structure, the role of women, eating behavior, management of acute excitement, and problems of intercultural psychotherapy, are subjects for ongoing discussion, study, and research.

Clinical Experiences

If cultural psychiatry is to attain a high level of educational importance to residents, it should be directly linked to the problems they encounter in the actual diagnosis, treatment, and management of psychiatric patients. Psychiatric residents should treat some patients from cultural backgrounds other than their own. Special problems in the treatment of these patients are often encountered. Initially, the psychiatric resident might profit from supervised experiences in interviewing mentally ill patients from cultures other than his or her own. Supervision of interviewing should focus on developing a style that is as free as possible from implications or subtle slips reflecting cultural biases or ethnic or racial stereotyping. Cultural differences between clinician and client often generate many misunderstandings in both parties. True understanding of one another is made difficult not only by possible language barriers but also by the use of different body postures and expressions, the holding of different value systems, and the different modes used to communicate distress and affect.

The Anglo clinician might consider obesity in a Puerto Rican woman as a sign of emotional problems, but if the woman is married she might feel a certain pride and strength in her weight because it means she is a complete woman. What is considered an *ataque de nervios* in Puerto Rican culture, an expression of social (usually marital) turmoil, might be misinterpreted by an Anglo clinician and misdiagnosed as an acute schizophrenic episode. The fact that a native American patient does not reply to questions about how his mother or father might think or feel, which might be a manifestation of the patient's high regard and respect for his elders, might be interpreted by a non-native-American clinician as an inability to empathize with the feelings of his parents. The Western clinician treating the Asian-American patient might interpret complaints of belching in the absence of organic pathology as hypochondria. The patient, on the other hand, might be expressing the loss of *ch'i* ("life energy") and might more accurately be considered depressed. The American clinician treating a patient from a Middle Eastern background might also be impressed with the high degree of hypochondriasis, particularly complaints regarding the area of the heart, and not fully appreciate that the patient is directly communicating depression. The locus of affect in many people from Iran, for example, is in the heart and not in the mind.

The congruence of the patient's explanatory model of illness in relation to the clinician's explanatory model of illness often creates misunderstandings that interfere with adequate treatment. Patients who believe that the cause of their problems and illness lies outside of themselves and their control are often considered as lacking insight and not easily reached in psychotherapy. Implicit in this situation is a real difference between the patient's model of illness and the physician's model of illness. One maintains that the locus of causation is in the social situation or in the environment, the other that the locus of causation is within the psyche of the patient. The converse situation also occurs in psychiatric practice. Patients feel that the causes of their problems are in their childhood experiences or in their current manner of relating to others and do not want to be "drugged," but the physician recognizes symptoms of manic-depressive illness and prescribes lithium.

Noncongruence of explanatory models often leads to impasse in therapy and noncompliance and reinforces patients' stereotypes of clinicians and clinicians' stereotypes of patients. Many difficulties en-

countered in psychiatric treatment relate precisely to the differences between the scientific medical model of diagnosis and treatment of disease based on the realities of our modern empirical world versus other models of the world and human emotion and functioning that are often found in our patients. Many of these models are not fully translatable into the medical model. In fact, many are difficult for the physician to fully understand. At best, by explicating these models to their fullest, we should be able to appreciate the power of culture in determining what the patient feels is an illness, whom he calls on for help, and what modes the patient uses for treatment and in explaining why the patient will or will not comply with prescribed psychiatric treatments.

When folk models of medicine are at gross conceptual variance with standard psychiatric models, true understanding in the doctor-patient relationship will be precluded and treatment will be made more difficult. Cultural psychiatry provides possibilities for informed negotiation and for more efficient implementation of medical treatments in such populations. If the cognitive realities of the folk system are well understood, aspects of the folk system might actually be fruitfully used and incorporated into the delivery of adequate medical treatment. When discrepancies exist between the folk model and the medical model, programs might be instituted to better acquaint patients with the medical model—to attempt to transform their systems to correspond more with those with which we are more familiar.

Other Suggestions

A list of psychiatrists with expertise in treating patients from certain ethnic groups might be developed for purposes of special consultation or supervision. A more comprehensive consultant list might include clinicians from institutions other than the principal training program.

The works of Kleinman and associates (6, 15), Fabrega (16), Snow (17), and others in medical anthropology provide a theoretical framework for designing effective therapeutic approaches in cultural psychiatry. These approaches focus on the effect on diagnostic and therapeutic processes of patients' conceptions of their illness, the psychosocial problems associated with their illness experience, patient requests and expectations from specific clinical interactions, and negotiations aimed at establishing therapeutic goals. These approaches have been found particularly useful to residents doing hospital consultation-liaison with patients with nonpsychiatric illnesses.

Grand rounds might include detailed presentation of both psychiatric and sociocultural aspects of the patient's life situation. Formal discussants could include a sociologist and/or an anthropologist.

A journal club could discuss journals that focus on cultural psychiatry. Important journals in this field currently include *Transcultural Psychiatry Research Review, Culture, Medicine and Psychiatry, Social Science and Medicine,* the *Journal of Operational Psychiatry,* the *International Journal of Social Psychiatry, Ethos,* the *Journal of Cross Cultural Psychology,* the *Journal of Nervous and Mental Diseases,* the *Journal of Psychological Anthropology,* and the *Psychoanalytic Study of Society.*

MODELS OF MENTAL ILLNESS

Three models of understanding mental illness—the medical, the cultural, and the ethnopsychiatric—are fundamental to a curriculum in cultural psychiatry. I recently discussed these models in another paper (18). The medical model assumes basic biological and cognitive universals and uses the diagnostic categories familiar to psychiatry, the cultural approach centers on the various manifestations of psychiatric disorders in different societies, and the ethnopsychiatric approach attempts to discover the folk classifications that are based on the particular realities and world view of a certain cultural group. The clinical value and information derived from each of these approaches is often quite different. However, from a clinical perspective each may provide unique approaches to the alleviation of a patient's symptoms.

The medical model has demonstrated effectiveness in chemotherapeutic, psychotherapeutic, and other treatments of mental disease. The cultural model provides recognition of basic psychiatric disorders occurring in the context of culture and manifested in unique and culturally stereotyped ways. The ethnopsychiatric model provides an approach that is concerned with defining what is wrong from the patient's point of view and giving these concerns an important position in developing a treatment plan. This model also provides for consultation and liaison with healers and others from the patient's community in delivering treatment and follow-up care.

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EDITORIAL

Beyond the Superego

What small child has not said to his parents, "It's not fair"? Parents themselves can be heard to remonstrate with their young children, "Now, be fair!" or, on the other hand, "Life isn't fair!" Both children and parents seem to know what each other means by "fair"—each tacitly agreeing to the concept of equitable treatment. Boszormenyi-Nagy and Spark have described in *Invisible Loyalties* (1) how parents act on the basis of their notions of fairness, which derive from their own experiences of just and unjust treatment. These legacies of transgenerational justice are passed on wittingly or unwittingly—perhaps leading to unfair or unrealizable claims on their offspring.

In "Trust-Based Therapy: A Contextual Approach," which appears in this issue of the *Journal,* Boszormenyi-Nagy and Krasner assert that intrafamilial trust is the basis of mental health and the foundation of psychotherapy, a radical shift in our current thinking. Their implied assertion that an important aspect of human relating is an intuitive sense of justice is akin to John Rawl's definition of "justice as fairness" as a basis of the social contract. This article—a summation of work with families over many years—integrates the insights of psychoanalysis and the concepts of systems theory as applied to families but proposes a more inclusive scheme based on intrafamilial ethics.

The ethical dimension that Boszormenyi-Nagy and Krasner emphasize is neither the superego of the Freudian model nor the traditional morality of religion. The authors refer to this dimension as "merited trust"; it refers to the long-term balance of fairness between the generations and within couples and derives from the existential fact that human beings are born into some form of the family and, inevitably, must come to terms with what they deserve, can expect to receive, and what is expected of them within that institution.

People act in ways they expect will gain them reciprocal behavior in return, and when they are treated in ways that they feel their past behavior has not merited, they feel treated unjustly. The bond of trust has been broken and the consequences are often mental "ill health." Parents expect to be and feel accountable to both their children and society for their treatment of them and in turn expect a certain loyalty from their children. Children do feel and act "loyal" to their parents—whatever forms this loyalty assumes. Uniquely among psychiatrists, Boszormenyi-Nagy and his collaborators have observed, investigated, and "treated" the twisted and fractured bonds of trust within families. We agree that a sense of justice is a dimension of human affairs which transcends individual psychology and is the relational matrix within which people seek and find value in their lives.

Researchers such as Bowlby have given us ample documention that "attachment" is a fact of human existence. Students of object relations have provided clinical data and explanatory theories to this effect, perhaps best expressed by an abused child patient of Fairbairn's who said, when asked if she wanted a "new, kind mummy," "No, I want my own mummy" (2). It is a human characteristic that we do have and need relationships and that each of us judges the ethical quality of these relationships.

The work of therapy is a "pragmatic strategy." It consists of identifying the remaining resources of trustworthiness in troubled families and disentangling the "invisible loyalties" that have perhaps come to be expressed in distorted ways.

However, that parents are "accountable" to their children and that children are "loyal" to their parents is accepted as a basic premise, whatever way these loyalties are expressed.

Just as parents are accountable to their children, therapists are accountable to their patients and to the significant others who will be affected by changes in one individual. Therapists cannot be neutral but must rather be partial in a "multidirectional way"—hearing each family member's claims on his or her own terms. Primary relationships and loyalties continue even if actual relationships with parents and grandparents have been severed. They are based on "roots in common." These are not substitutable by any other relationship and "contribute to an individual's uniqueness." Second, common bonds constitute an "ethical claim" requiring that relationships remain in fair balance and that trust be merited and maintained. Therapists have the opportunity to help a family to restore its uniqueness and integrity.

What is important about this article is that it makes us sit up straight and reexamine certain assumptions as we go about our work. It asserts that human beings *are* "entitled" to and do deserve a modicum of fairness, that "mutual trust" based on consideration and caring accumulates "merits," and that "merits" accumulated deserve to be recognized and repaid over the long term. This does not imply that life will always be "fair" in the short run or that people are "entitled" to all that they might wish. It does, however, maintain that long-term bonds of familial trustworthiness help people through periods of unfairness. It denies "the trap of the contemporary observer of human relationships: in this view repayment of obligations emanating from procreation is optional and depends on the participants' feelings and a variety of power manipulations."

Some questions come to mind: How does love enter the picture? What of giving that is motivated by the wish to give pleasure to someone one loves? Perhaps the authors are implicitly suggesting that the unconditional love between parent and child or in marriage exists as an aspect of roots in common while "merited" trust is a broadened conception of the semiconditional love of adult life. The complexities of how these dimensions of trust and love are worked out in human families need further elaboration.

In conclusion, however, we feel that this approach to helping people should enrich the work of all therapists, whether they choose to see individuals or families, and, we hope, further the already visible movement toward integration and an end to sectarian squabbles. It behooves all therapists to consider seriously their patients' "loyalties": it suggests that even in work with individuals the enduring value of the individual's family and social context must be appreciated or therapy may be merely temporarily palliative or even destructive. The overriding implication is that civilization and decency in human conduct need not be a repressive force but can be, and often is, a liberating source of mental health and creativity.

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BRIEF COMMUNICATIONS

Effect of Anticonvulsants on Plasma Haloperidol and Thioridazine Levels

BY MARKKU LINNOILA, M.D., PH.D., MATTI VIUKARI, M.D., KYOSTI VAISANEN, M.D., AND JUHANI AUVINEN, M.SC.

Patients who had therapeutic plasma levels of phenobarbital and/or diphenylhydantoin had significantly lower plasma levels of haloperidol and mesoridazine, the active metabolite of thioridazine, than patients who did not receive anticonvulsants. Plasma thioridazine levels per se were not affected by concomitant anticonvulsant treatment. Biperiden, an antimuscarinic, antiparkinsonian agent, did not affect the plasma levels of these three neuroleptics.

Phenobarbital and diphenylhydantoin are well-known inducers of the cytochrome P_{450} enzyme system of the liver (1), which is responsible for the biotransformation of many drugs and hormones (2). The significance of the induction of this enzyme system for the metabolism of most psychotropic drugs has not been studied in man.

Neuroleptic drugs are initially transformed mainly by the cytochrome P_{450} system in the liver (e.g., haloperidol) or both P_{450} in the liver and the intestinal sulfoxidase, a different species of cytochrome P_{450} (e.g., chlorpromazine and thioridazine) (3, 4). Therefore, the rate of metabolism and the plasma levels of different neuroleptics may be differentially affected by the induction of the cytochrome P_{450} system of the liver. Since the steady-state plasma levels of neuroleptics are probably related to the antipsychotic potency of these drugs (5), knowing the effects of enzyme induction on the plasma levels of neuroleptics in man is clinically important.

In the present study we investigated the effects of concomitant treatment with phenobarbital and/or diphenylhydantoin on the steady-state plasma levels of haloperidol, thioridazine, and thioridazine's active metabolite, mesoridazine (6).

METHOD

The subjects were 30 mentally retarded patients (21 men and 9 women) from Vaalijala Hospital (range of WAIS IQs, 50–70) who suffered from agitation or aggressive outbursts that were severe enough to be treated with high doses of neuroleptics. Nine patients (7 men and 2 women; mean age, 23.3±4.4 years) who had a grand mal seizure disorder required anticonvulsant medication; 2 received phenobarbital, 3 received diphenylhydantoin, and 4 received a combination of these drugs. All the other patients (7 women and 14 men; mean age, 26.8±7.4 years) required no anticonvulsant medication. All patients had been in Vaalijala Hospital for a minimum of 3 months and received a regular hospital diet. No medication other than the anticonvulsants, the neuroleptics, and an antimuscarinic, antiparkinsonian drug were used during the study. Patients consented to participate in the study, which was accepted by the committee of clinical investigations at Vaalijala Hospital.

The experiment was designed as a double-blind crossover, with 2-week washout periods between the treatments with psychotropics, which were administered in a random order. The duration of the treat-

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ments was 6 weeks each. The dose was increased to the maintenance level during the first week.

We investigated haloperidol, 10 mg t.i.d., thioridazine, 200 mg t.i.d., and placebo, all of which were administered in tablets with similar dissolution rates and identical appearance and taste. Biperiden, 2 mg t.i.d., was added to the regimen of 11 patients (mean age, 25.7±8.3 years, mean weight, 61.6±14.1 kg) at the discretion of the clinical investigator when patients exhibited extrapyramidal symptoms; 19 patients (mean age, 25.5±7.6 years; mean weight, 54.5±10.8 kg) were not given biperiden.

Blood samples (5 ml) for the measurement of the anticonvulsants and neuroleptics were drawn from an antecubital vein at 8 a.m. on the 14th, 28th, and 42nd mornings, 11 hours after the previous drug dose.

Plasma levels of anticonvulsants were quantitated by use of a routine gas chromatographic procedure (7). Haloperidol was measured with gas chromatography (8). Thioridazine was quantitated fluorometrically (9), and the level of mesoridazine was calculated by subtracting the thioridazine level from a fluorometrically measured combined level of thio- and mesoridazine (10). Significance levels were determined by analysis of variance.

RESULTS

The mean (±SD) anticonvulsant plasma levels of the 9 patients requiring such medication were within the established therapeutic ranges for diphenylhydantoin (12.0±5.1 g/ml) and phenobarbital (16.4±4.1 g/ml). They were not affected by the concomitant administration of haloperidol or thioridazine.

On the contrary, mean (±SD) haloperidol levels on the 14th, 28th, and 42nd mornings were 5.9±6.4, 6.6±3.2, and 19.4±5.7, respectively, in the patients receiving anticonvulsants and 9.9±6.8, 23.2±9.8, and 36.6±16.5 ng/ml in the patients not on anticonvulsants. The patients receiving anticonvulsants had significantly lower plasma haloperidol levels on day 14 ($p<.05$) and on days 28 and 42 ($p<.001$).

Plasma thioridazine levels were not significantly different between the two groups. The patients taking anticonvulsants had mean plasma thioridazine levels of 210±130, 430±220, and 580±280 ng/ml on days 14, 28, and 42, respectively, and in the patients not taking anticonvulsants they were 360±230, 520±210, and 650±260 ng/ml.

Mean plasma mesoridazine levels in the patients taking anticonvulsants were 1250±580, 2170±1070, and 2310±1070 ng/ml on days 14, 28, and 42, respectively, and in the patients not taking anticonvulsants they were 2230±1200, 2870±1110, and 3080±1100 ng/ml. The patients taking anticonvulsants had significantly lower plasma mesoridazine levels on days 28 and 42 ($p<.05$).

Biperiden did not affect plasma levels of the neuroleptics. Mean plasma haloperidol levels in the patients taking biperiden were 9.22±5.67, 16.50±12.33, and 27.82±20.23 ng/ml on days 14, 28, and 42, respectively, and in the patients not taking biperiden they were 8.38±7.50, 19.18±10.95, and 31.21±16.97 ng/ml. In patients taking biperiden mean thioridazine plasma levels were 350±100, 540±230, and 680±270 ng/ml on days 14, 28, and 42, respectively, compared with 290±230, 460±210, and 600±260 ng/ml in the patients who were not. Mean plasma mesoridazine levels on the 14th, 28th, and 42nd days were 910±760, 2510±1100, and 2600±1160 ng/ml, respectively, in the patients receiving biperiden and 1880±1380, 2630±1130, and 3060±1330 ng/ml in those not receiving biperiden.

DISCUSSION

We found that concomitant anticonvulsant medication significantly reduced the plasma levels of haloperidol ($p<.001$) and mesoridazine, the active antipsychotic, sulfoxide metabolite of thioridazine ($p<.05$) compared with an age- and sex-matched patient population with similar mental disorders and diets. Thioridazine levels did not differ significantly between the groups. Haloperidol doses need to be increased 200%–300% and mesoridazine doses 25%–40% (assuming linear kinetics) in patients on anticonvulsants to produce plasma levels comparable with patients not on anticonvulsants.

This pharmacokinetic report is part of an investigation to elucidate the necessity of chronic high dose neuroleptic medication to control agitation and aggression in an institutionalized mentally retarded patient population. The rationale for choosing haloperidol and thioridazine was to provide a comparison between a drug that was relatively free of autonomic nervous system side effects but had a significant potential to induce extrapyramidal symptoms and a drug that had the opposite pharmacologic profile.

The lack of an effect of anticonvulsants on plasma thioridazine levels may indicate that the sulfoxidation of this drug, which leads to the formation of mesoridazine, is the rate-limiting step of its biotransformation in man. The same step is probably rate-limiting also in the biotransformation of chlorpromazine (11). A given plasma level of mesoridazine is likely to have more neuroleptic activity than thioridazine because mesoridazine has a low degree of binding to plasma proteins (12). High levels of this metabolite are produced during oral therapy with thioridazine; therefore, it may be worthwhile to increase the dose of thioridazine in patients taking anticonvulsants who do not respond to a usual dose of the neuroleptic.

Earlier findings that trihexyphenidyl, lithium, and aluminum ion (13–17) lowered plasma chlorpromazine

levels could not be extended to the combinations of biperiden and haloperidol and biperiden and thioridazine in this study, perhaps because haloperidol is not sulfoxidated in the intestinal wall and thioridazine per se is a potent antimuscarinic agent (18). The antimuscarinic potency of thioridazine may be strong enough to abolish any effects of biperiden on gastric motility, the slowing of which by trihexyphenidyl, lithium, and aluminum has been suggested as the cause of the reduced bioavailability of chlorpromazine (13–17).

We conclude that anticonvulsants profoundly reduce plasma levels of neuroleptics whose biotransformation is primarily dependent on the cytochrome P_{450} enzyme system in the liver. To sustain therapeutic plasma neuroleptic levels, patients taking anticonvulsants may require doses of haloperidol that are 200%–300% higher than those required by patients not taking anticonvulsants; for patients taking mesoridazine and anticonvulsants, a 20%–40% dose increment of the neuroleptic may be required. The effect of anticonvulsants on haloperidol is similar to their effect on tricyclic antidepressants, which have been reported to induce low plasma levels in patients taking phenobarbital (19).

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Psychiatric Problems in Third-Year Medical Students

BY MICHAEL H. SACKS, M.D., WILLIAM A. FROSCH, M.D., MARTIN KESSELMAN, M.D., AND LYNDA PARKER, M.D.

Each year as many as one out of four medical students experience sufficient emotional pain to seek psychiatric consultation. In many cases the precipitating stress relates to specific stresses associated with the phase of training. In the third year increased clinical responsibility may evoke feelings centered on caretaking, sexuality, and aggression that cannot be contained by an as yet fragile emerging professional identification. The authors present four case histories of third-year medical students to illustrate the complex interaction between family background, motivations to enter medicine, and the specific patient experience that resulted in sufficient distress to require psychiatric consultation.

As many as one-quarter of medical students seek a psychiatric consultation before graduation from medical school (1). Although students' reasons for seeing a psychiatrist differ, each year of medical school has distinctive patterns of stress (2). Characteristically, first-year students experience anxiety or depression in response to confronting a mass of information that cannot be learned in its entirety. This situation contrasts with the student's earlier undergraduate experience, in which specific guidelines were provided regarding the material to be mastered. Alternatively, the student finally in medical school may feel free for the first time to question his or her motivation for entering medicine. In the second year, the student's initial experiences with death and pathology often result in hypochondriacal fears and preoccupation. In the third year, patient contact often provokes a crisis related to issues of intimacy and closeness. Increased responsibility and anticipation of yet greater responsibility may produce sufficient emotional discomfort in fourth-year students to result in a psychiatric consultation.

Although this phase-related scheme is of some value, its application requires caution. The interaction between premorbid personality and the stresses of a particular year may not be simple. This scheme does, however, draw attention to the possible importance of school experiences to a student's request for psychiatric help (3).

In this paper we will describe four medical students who received treatment during their third year. Our purpose is to focus on the complex interactions between premorbid personality, life experience, and the particular stresses of the clinical year of medical school.

CASE REPORTS

Case 1. Mr. A, a 27-year-old man, sought treatment after receiving a low but passing mark in medicine that severely depressed him because it seemed unfair. He emphasized that the grade did not reflect his devotion to patients. In particular, he described intense involvement with a preadolescent female patient who had leukemia; he said that unlike other students he was spending an extraordinary amount of time with his patient, and at times could not study or complete written work because of a need to see how she was doing.

Initially treatment focused on Mr. A's belief that he was entitled to special treatment despite his not having accomplished the expected work. This expectation was related to his becoming the "man of the house" at age 10 when his father died. His wish to become a physician also appeared then, partially as a result of an identification with a well-known uncle. This feeling of entitlement was accompanied by intense guilt, which Mr. A began to appreciate when he realized that he frequently provoked people in authority to reprimand him.

After mentioning that he had once again unknowingly provoked an attending physician, Mr. A reported the following dream: "I have a bar in my head. It extends from temple to temple. It is removed by a plastic surgeon in the OB-GYN operating suite." Mr. A associated the bar in his head with the devil's horns and related the dream to his belief that he was an evil person with evil wishes. The psychiatrist suggested that perhaps the horns represented sexual wishes and that his need to punish himself for them might have been responsible for his having provoked the attending physician to rebuke him. Mr. A became silent and then said, "I guess I

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might as well tell you." He reported an encounter with an 11-year-old girl whom he had befriended that had sexual overtones which disturbed him.

Mr. A justified his interest in the girl on the basis of his wish to become a specialist in adolescent medicine. In subsequent sessions it emerged that during his adolescence he had become overwhelmed by intense sexual feelings toward his younger sister. Reaction formation toward these feelings emerged as a central determinant in his decision to become a physician. Being closer to this goal during his medical clerkship intensified these latent sexual urges and the guilt attending them. This intensification resulted in his symptomatic preoccupation with caring for the young female patient, which resulted in his neglecting his academic work and seeking therapy.

Case 2. Ms. B, a 27-year-old engaged woman, had an intense anxiety reaction during her psychiatry clerkship. The precipitant was not clear. As treatment proceeded Ms. B spoke of her wish to be a "superwoman." By this she meant that she should be very successful as a wife and mother as well as a physician. The anxiety attack occurred while she was caring for a patient whose mother was a professional. She feared that her own professional strivings might result in her proving to be an inadequate mother. Her own mother had abandoned a promising career to devote herself to raising a family and Ms. B felt that a woman's primary responsibility was her family and that only men were entitled to a profession. This conflict over role also appeared in the severe crisis she had had in choosing her specialty. She wanted to be a cardiologist but felt that a woman should be a psychiatrist.

While on a medicine rotation, Ms. B was complimented on her work and encouraged to become a cardiologist. That afternoon while at rounds she felt isolated and had the following fantasy: "It was like they were players on a football team in a huddle drawing diagrams of the next play and I could never join the huddle." The therapist related this to previous feelings that she could not be successful at what she wanted because she was not a man. Ms. B recalled that she had always felt this way. As a preadolescent she would go horseback riding with friends and they would always play Indian boys because "Indian girls did not get to ride horses and did not have fun like Indian boys." She felt she could not become a cardiologist unless she became a man, which caused her anxiety in anticipating her future role as a wife and mother. She had long felt that she would have to become a psychiatrist or a pediatrician because those were the parts of medicine that were for the "squaws." It was now easier for Ms. B to examine her envy of men and the privileges and excitement that they seemed to have, as well as her feelings of inadequacy as a woman. The reasons behind her previous rejection of a career as a nurse became clear; it was a woman's profession and one primarily involved with caring. The only good professions were those that belonged to men. The anxiety attack during her psychiatry clerkship was determined by multiple conflicts over professional and sexual roles that were triggered by the specifics of a particular clinical relationship.

Case 3. Ms. C, a 24-year-old woman, entered treatment early in her third year because of the acute onset of anxiety and depression. Her mother had given up her career on the birth of her first child. Her father was marginally successful and had been forced to give up greater aspirations when Ms. C was an infant. Her mother felt she had "come down in the world" as a result of her husband's failure and therefore the sacrifice of her own career became particularly difficult to accept.

Ms. C's parents often argued. The arguments were generally initiated by the mother, and the father would withdraw from her anger with a tolerant and long-suffering air. Between these arguments the mother would appear depressed and resentful, sometimes withdrawing into her room for days. The "crazy" behavior of her mother frightened Ms. C profoundly, while the father's distant attitude aroused her contempt, a feeling she now frequently felt toward men.

During her childhood Ms. C played the role of the troublesome scapegoat, drawing her parent's blame and rage on herself. Inside she felt empty, bad, and ineffective. Away from her home for the first time, during college she reacted to these feelings by use of drugs and sexual behavior out of keeping with the conventional values held by her family. Her decision to go to medical school represented a dramatic change in this rebellious pattern. However, in medical school she joined organizations that sought to protest injustice in medical care for the poor. Although she found close women friends, partly through these political activities, her relations with men were sporadic, distrustful, and vaguely contemptuous. A victim-victimizer pattern characterized her intimate relationships and was accompanied by vacillations between intense anger at intimates and guilty depression.

In contrast to her competence in clinical situations, Ms. C appeared frightened, disorganized, and fragile in treatment. At other times she complained that her male therapist was critical, harsh, and cold. One session she arrived acutely and severely depressed. She had barely slept for three nights, which she had spent at the bedside of a young mother dying of a brain tumor. She was preoccupied by the thought of the dying woman's children, who would never really know their mother. Her feelings toward the patient were less painful, although complex; at times horror and pity that one could die so young, sometimes rage, at times survivor guilt (the patient and she were the same age). Her long bedside vigils, which were inappropriate, appeared to represent an attempt to rescue the child-patient with love where medication failed. For the next three days Ms. C's depression mounted, culminating in her taking a nonprescription sleep medication in what she recognized to be a suicidal gesture.

For this borderline woman, intellectual achievement and identification with various social ideologies had permitted a superficially adequate level of vocational and social function in spite of the disruptive rage she experienced in close interpersonal relationships. The choice of medicine as a career was a final ideological commitment, an altruistic defense against her own demandingness and rage. As long as she could cavil at the coldness and technological alienation of her fellow students and house officers, she could maintain her posture of being aloof and uniquely virtuous. However, the alternation of her identification with the abandoned children of the dying woman with guilt over her failure to assuage the mother's suffering appeared to evoke feelings previously dissociated from her professional life.

Case 4. Mr. D, a 24-year-old medical student, had a history of panic attacks and depression, which began during his OB-GYN clerkship. He had suicidal ideation, which seemed to be precipitated by a letter from his girlfriend ending their

four-year relationship. In treatment he later revealed that he had never had a sexual relationship with her and suspected that she was homosexual.

As a child, Mr. D's older sister was always considered sick and required psychiatric treatment. He remembered that she would throw temper tantrums which gained her parents' attention; he unsuccessfully attempted to gain their favor by being the "good child." When Mr. D was ready to attend college both his parents sought additional professional training. This left no money for Mr. D's education at the private college he had wished to attend. He accepted the situation without complaint and attended a local college. He could not recall why he decided to enter medicine, only that his family was not impressed when he was admitted to a medical school.

Mr. D described his mother as incredibly bitter and he resented her confiding to him about his father's sexual inadequacies and her suspicion that he was having an affair. Mr. D was also angry that his mother saw herself in her profession as a caring and nurturing woman, which he saw as incongruous with her harshness, bitterness, and castrating attitudes toward his father. He also had disturbing memories of how his mother punished his sister when they were young, and he thought these experiences were somehow responsible for their problems.

When Mr. D began his pediatric rotation, he found that he often became enraged at children's families. This would then trigger suicidal thoughts. He related this anger to his own family's neglect during his childhood and during his recent psychiatric difficulties, when they refused to be involved in his treatment.

Mr. D realized that the suicidal thoughts related to guilt regarding his "annihilating anger" and that his decision to be "good" as a child was not related to a passive acquiescence to his parents' wishes but rather to competition for the attention of his mother, first with the sister and later, during college, with his father. This anger at his mother may have been intensified during his OB-GYN rotation when he had to care for older women, but this hypothesis was never directly confirmed. The loss of his girlfriend at this time did, however, evoke similar feelings of rage, which Mr. D was unable to contain by being the "good little boy" or the good and caring doctor.

DISCUSSION

These four patients experienced anxiety during their third-year clerkship. The anxiety was occasioned by their work with patients whose situations resonated with the unconscious conflicts associated with becoming a physician. These conflicts were related to childhood conflicts over sexuality (patients 1 and 2), anger at maternal deprivation (patients 3 and 4), and sibling rivalry (patients 2 and 4). During the clerkship year they found it particularly difficult to contain their anxiety and had to seek psychiatric care. It is apparent that their decisions to enter medicine were partially determined by insufficiently sublimated conflicts.

The clerkship year may specifically stress those students whose motivation for entering medicine is to provide for others what they felt they did not themselves receive as children. Vaillant (4) found that physicians appear to be more vulnerable to psychiatric difficulty if they experienced their childhood as unhappy and deprived. If in caring for their patients these physicians were unable to obtain care for themselves they were particularly vulnerable. Closely related to this problem is channeling the intense anger at the depriving parents into behavior toward patients, families, teachers, or toward the self, as happened in the suicidal impulses of patients 3 and 4. Although issues of being cared for and mothering were important for all the patients, for patients 1 and 2 other issues were also involved.

In the case of patient 1 unresolved sexual problems were predominant. In the case of patient 2 guilt over her aggressive wishes to be able to compete and her envy of men were central.

In each case conflicts contributed to the career choice of medicine. Although one assumes that such conflicts often contribute to career choice, these four patients were unable to maintain the adaptive aspects of their attempted conflict resolution. During the preclinical years they had minimal difficulty; their ideas of the doctor-patient relation were still idealized, not yet subject to the intimate confrontation that actual care of patients requires.

The doctor-patient relationship for these students evoked feelings that centered predominantly on caretaking, sexuality, or aggression rather than on professional concerns. Many medical students struggle with such neurotic determinants of career choice and use a wide variety of adaptive maneuvers, both group and individual, to successfully master the conflicts that are stirred up by patient contact. Chronic detachment, the depersonalization of the patient to a diseased organ, overinvolvement with nonmedical aspects of the patient, and identification with a house officer are examples of common adaptive maneuvers. All of these four students were acutely aware of this usual process; they experienced part of their problem as a sense of isolation from their peers, of being unable to treat patients in the way their peers seemed to. By paying attention to the student's struggle with a fragile sublimation, the therapist who treats the third-year student can help him or her understand the fantasies and anxieties that working with patients produces.

When medicine is chosen to resolve insufficiently sublimated personal conflicts, specific clinical experiences may so resonate with intrapsychic problems that the adaptive aspects of the choice are lost and symptoms appear. While this may in part be phase-related, it is also particular to the individual student and life experience. While phase-related aspects of the clinical clerkship are perhaps most important for the treatment of the healthy student who experiences difficulty, it is critical not to overlook the importance of individual conflicts in students with more serious pathology. If these are not adequately dealt with in treatment, they

are likely to recur periodically during practice, too often to the detriment of patients, physician, and the profession.

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Adult Psychiatric Disorders in Parents of Hyperactive Children

BY JAMES MORRISON, M.D.

The family histories of 140 children and adolescents with hyperactive child syndrome were compared with the family histories of 91 psychiatrically ill, age- and sex-matched patients who had a primary diagnosis other than hyperactive child syndrome. Parents of hyperactive children were more likely to have antisocial personality and Briquet's syndrome (hysteria) than were parents of nonhyperactive children; 11% of the hyperactive children had a parent with such a diagnosis. Parents of nonhyperactive children were more likely to have endogenous psychosis. The diagnosis of alcoholism did not significantly differentiate parents of the two groups of probands. These data support earlier studies and suggest a specific association between hyperactive child syndrome and the personality disorders of antisocial personality and hysteria.

In the early 1970s two studies (1, 2) were published on the family histories of children with hyperactive child syndrome. These studies found a high prevalence of antisocial personality and Briquet's syndrome (hysteria) among parents of hyperactive children, and this finding has provided the cornerstone for the argument that the hyperactive child syndrome is in some way related to personality disorders in adults. One objection to this conclusion is that no study has compared the family histories of hyperactive children with the family histories of nonhyperactive psychiatric patients. This study will report such data.

I compared the family histories of a group of hyperactive children with the family histories of a group of psychiatrically ill children seen in a private-practice setting. In essence, this study replicates the two earlier family investigations of hyperactive child syndrome and at the same time tests the findings of these two studies for specificity.

METHOD

Over a 6-year period, 391 children and adolescents were seen as part (30.1%) of a general private psychiatric practice. Patients and their parents or guardians were administered a structured interview, which covered symptoms, family histories, and demographic and social data (3). From these interviews I selected 140 case histories of children who manifested hyperactive child syndrome. These patients met the criteria of Stewart and associates (4), were not adopted, and had at least normal intelligence. Family history did not serve as a criterion for this diagnosis. For the nonhyperactive comparison group I selected 91 case histories of patients, who were generally diagnosed according to *DSM-II* criteria, were psychiatrically ill, nonadopted children, and were matched for sex and age (within 2 years). In both groups only 1 child was selected from each family.

Diagnosis of adult psychiatric disorders in the families of these patients was based on the criteria of Feighner and associates (5). In a few instances, particularly those involving divorced parents who were not

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TABLE 1
Diagnoses of Parents of Hyperactive Children and Parents of Nonhyperactive Children

	Parents of Hyperactive Children (N=140)				Parents of Nonhyperactive Children (N=91)			
	Mothers		Fathers[a]		Mothers[a]		Fathers[a]	
Diagnosis	N	%	N	%	N	%	N	%
Affective disorder	20	14	4	3	16	18	8	9
Schizophrenia, schizophreniform psychosis	0		1	1	4	4	4	5
Alcoholism	19	14	30	22	8	9	14	16
Briquet's syndrome	6	4	0		1	1	0	
Antisocial personality	2	1	9	7	1	1	0	
Drug abuse	2	1	1	1	0		0	
Undetermined[b]	26	19	19	14	9	10	9	11
No adult disorder	65	46	71	53	51	57	50	59

[a]No information available for 5 fathers of hyperactive children, 1 mother of a nonhyperactive child, and 6 fathers of nonhyperactive children.
[b]Psychiatrically ill, but no specific diagnostic information available.

available for interview, the criteria had to be modified as suggested by Andreasen and associates (6). Whenever there was doubt about the diagnosis of a parent, the tendency was to err on the conservative side. This may have resulted in a number of undiagnosed psychotic and nonpsychotic illnesses, even among interviewed parents. Because of the great subjectivity and many meanings attached to hysteria, I made no diagnosis of Briquet's syndrome without full research criteria.

RESULTS

Of the 140 hyperactive children, 111 (79%) were boys; of the 91 nonhyperactive children 73 (80%) were boys. Mean ages were 11.8 years and 13.1 years, respectively. Ten percent of the children in each group were not white.

Definite and probable diagnoses of the nonhyperactive children were as follows: 31 (34%) had affective disorder, 11 (12%) had schizophrenia/schizophreniform psychosis, and 11 (12%) had situational adjustment reaction, 8 (9%) had personality disorder, 6 (7%) had organic brain syndrome, 6 (7%) had habit pattern disturbance, 5 (5%) had alcohol or drug abuse, 1 (1%) had school phobia, and 1 (1%) had anorexia nervosa. Eleven (12%) of these children were psychiatrically ill, but the type of illness could not be diagnosed.

Psychiatric diagnoses of the parents of both groups of children are given in table 1. Parents of hyperactive children were slightly more likely to have a psychiatric diagnosis and to have affective disorder. Schizophrenia and schizophreniform psychosis were more common ($\chi^2=7.63$, $p<.01$) in the nonhyperactive group. Somewhat more parents of hyperactive children were psychiatrically ill, but the specific illness was undiagnosed.

Antisocial personality and Briquet's syndrome were encountered far more often in the parents of the hyperactive group (χ^2 for the combined personality disorders=5.52, $p<.02$). Sixteen (11%) of the hyperactive children had at least one parent with Briquet's syndrome or antisocial personality; only two (2%) of the parents in the comparison group were so affected ($\chi^2=5.32$, $p<.05$).

To determine if the high prevalence of personality disorder in the parents of the hyperactive children could be correlated with previously undetected conduct disorder in the children, these children's records were reviewed again. Even applying *DSM-III* criteria retrospectively, no conduct disorder could be identified in these 16 children. In fact, on closer inspection these children looked like typical hyperactive children: their mean age, 9.9 years, was somewhat below the group mean, and 14 (88%) of them responded in typical fashion to psychostimulants or to tricyclic antidepressants, compared with 69% for the hyperactive group as a whole.

Alcoholism was found to exist slightly more often in the parents of the hyperactive group than in the parents of the nonhyperactive group ($\chi^2=1.84$, $p>.10$).

DISCUSSION

Dubey (7) has discussed several suggestions for investigators studying possible organic etiologies of the hyperactive child syndrome, including a comparison with deviant control groups to demonstrate specificity. In this study the family histories of a group of hyperactive children were compared with the family histories of psychiatrically ill children seen in a private practice setting. The problems of nonblind diagnosis remain, of course, although they are mitigated to some extent by insistence on adhering to strict diagnostic criteria whenever possible. This study replicates the two earlier investigations (1, 2) and tests their findings for specificity.

The most important finding of the previous studies was the high prevalence of antisocial personality and Briquet's syndrome among parents of children with

hyperactive child syndrome. This is the third study that concludes that personality disorders of antisocial personality and Briquet's syndrome are found in excess in parents of hyperactive children. The argument is thus strengthened for a specific relationship between hyperactive child syndrome and these two personality disorders.

The opposite conclusion must be drawn regarding alcoholism. The two earlier studies found a significant excess of alcoholism among the parents and extended families of hyperactive children, but the data reported here cast doubt on the importance of the previous findings. Although in this study alcoholism was nearly as prevalent among parents of hyperactive children as it was in the previous two studies, these figures only slightly exceed the incidence of alcoholism in parents of nonhyperactive children.

The near absence of schizophrenia and schizophreniform psychosis in parents of hyperactive children as compared with parents of nonhyperactive children supports my earlier speculation (8) that hyperactive child syndrome and endogenous psychoses may be negatively associated. However, as schizophrenia does occur in formerly hyperactive adults (9), the two conditions cannot be considered mutually exclusive.

A genetic mechanism has been proposed for the transmission of childhood hyperactivity from one generation to another; support for this hypothesis was reviewed by Cantwell (10) and earlier by Omenn (11). Although the data presented here cannot support a genetic etiology for this condition, they strengthen the view that the syndrome is a familial condition that is specifically related to the personality disorders of antisocial personality and Briquet's syndrome. Hyperactive child syndrome is probably unrelated to affective disorder and to schizophrenia. The specificity of a relationship between hyperactive child syndrome and alcoholism reported in earlier studies now appears questionable. Heightened awareness of a specific association between hyperactive child syndrome and the personality disorders described may provide the basis for further scrutiny of the symptomatology, pathophysiology, and treatment of this common childhood disorder.

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A Comparison of Cognitive Impairment Due to Benzodiazepines and to Narcotics

BY NELSON HENDLER, M.D., M.S., CINDI CIMINI, M.S., TERENCE MA, AND DONLIN LONG, M.D., PH.D.

In an attempt to determine the source of cognitive impairment in 106 consecutively admitted patients at the Johns Hopkins Chronic Pain Treatment Center, EEG, the Wechsler Adult Intelligence Scale, Memory Quotient, and Bender Gestalt tests were administered. Patients receiving benzodiazepines alone demonstrated alterations in cognitive functioning and EEG evidence of a sedative effect. Patients receiving narcotics alone and a group of patients not receiving medication did not show signs of cognitive impairment. The effects of benzodiazepines on sleep and perception of chronic pain, in combination with the cortical changes that they produce, imply that these drugs should not be used in most patients with chronic pain.

The use of benzodiazepines has become widespread in the clinical practice of medicine, to the point that 97% of physicians in general practice prescribe drugs in this category (1). While benzodiazepines have been used to treat anxiety, insomnia, headaches, hypertension, and other psychophysiological problems, their efficacy in the treatment of chronic pain has, to our knowledge, never been demonstrated. Nonetheless, in a recent study conducted at the Johns Hopkins Hospital Chronic Pain Treatment Center,[1] 43% of the patients were found to be taking benzodiazepines by prescription from the referring physician. In fact, the use of benzodiazepines in patients with chronic pain would theoretically be ill-advised because they reduce the turnover of serotonin (2), thus interfering with natural sleep and lowering the tolerance to chronic pain (3, 4). However, the most significant problem that benzodiazepines create seems to be cognitive impairment with associated EEG changes, which mimic the patterns seen with other sedative and hypnotic drugs (5). Acute, single-dose administration of diazepam does seem to produce impairment in learning, memory, and psychomotor functioning (6).

While the long-term use of narcotic agents in patients with chronic pain is also contraindicated, approximately 80% of the patients admitted to the Chronic Pain Treatment Center of Johns Hopkins Hospital were also receiving these drugs (7). However, the evaluating psychiatrist noted that a great deal of cognitive impairment seemed to occur more often in patients using benzodiazepines than in patients using only narcotics. The following study was conducted in an attempt to verify this impression.

METHOD

Data were collected from 106 consecutively admitted patients at the Chronic Pain Treatment Center at Johns Hopkins Hospital. In an attempt to assess each patient's drug intake for the one month prior to admission, a history was obtained from the patient, the patient's family, the referring physician, and/or a qualitative drug screen for benzodiazepines, sedatives, hypnotics, and narcotics. Cognitive impairment was studied by administering both the verbal and performance scales of the Wechsler Adult Intelligence Scale (WAIS). If the difference between the two scale scores exceeded 12 points, it was considered abnormal. In addition, a Memory Quotient was obtained, and if the score was 15 points below the total WAIS score, it was considered abnormal. Finally, the Bender-Gestalt test was administered, and scores greater than 70 were considered indicative of psychomotor impairment.

EEG recordings were obtained from the laboratory at the hospital and were considered compatible with a sedative or benzodiazepine effect only if a great deal of 12–20 cps or 15–30 cps (beta) activity was reported by an objective observer, who was unaware of the patients' medication intake. EEGs with moderate or fair amounts of beta activity were considered normal. It is important to note that at no time were any of the EEGs

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[1]N. Hendler, C. Cimini, T. Ma, et al: Cognitive impairment due to benzodiazepines, unpublished paper.

TABLE 1
Number of Chronic Pain Patients with Cognitive Impairment

Drug	Impaired (N=16)	Not Impaired (N=15)
Benzodiazepines alone (N=13)	10[a]	3
Narcotics alone (N=13)	4	9
No drugs (N=5)	2	3

[a]χ^2=6.0, p<.05.

TABLE 2
Number of Chronic Pain Patients with Impaired Cognitive Testing Alone (Regardless of EEG)

Drug	Impaired (N=14)	Normal (N=12)
Benzodiazepines alone (N=13)	10[a]	3
Narcotics alone (N=13)	4	9

[a]χ^2=5.6, p<.02.

interpreted as having a pattern indicative of narcotic toxicity. Any patient having an EEG with an epileptogenic focus, or any indication of slowing compatible with arteriosclerotic cardiovascular disease or the effect of psychotropic medication, was eliminated from the study.

Patients below the age of 21 and above the age of 70 were not included in the study. The average age for the group of patients taking just narcotics was 43.4 years (range of 22–69), while the average age for patients taking just benzodiazepines was 41.8 years (range of 26–65). Patients with a history of alcoholism or barbiturate, hypnotic, or stimulant use were not included in the study.

TABLE 3
Number of Chronic Pain Patients with EEG Changes

Drug	EEG Changes (N=8)	Normal (N=18)
Benzodiazepines alone (N=13)	8[a]	5
Narcotics alone (N=13)	0	13

[a]χ^2=11.2, p<.001.

TABLE 4
Number of Chronic Pain Patients with Both Altered EEG and Cognitive Impairment

Drug	Both EEG Change and Cognitive Impairment (N=7)	Either EEG Change or Cognitive Impairment or Neither (N=19)
Benzodiazepines alone (N=13)	7[a]	6
Narcotics alone (N=13)	0	13

[a]χ^2=9.6, p<.002.

TABLE 5
Number of Chronic Pain Patients with EEG and Cognitive Alterations

Drug	Both EEG Change and Cognitive Impairment (N=21)	Only Cognitive Impairment (N=13)	Only Altered EEG (N=4)	Both EEG and Intellect Normal (N=12)
Benzodiazepine with or without narcotic (no hypnotic) (N=37)	21[a]	9	4	3
Narcotic alone (N=13)	0	4	0	9

[a]χ^2=24.3, df=3, p<.001.

RESULTS

Of the 106 patients, 64 (60%) were taking benzodiazepines. This represented the patients admitted during a 6-month period (February through July 1979). Only 6 patients were not taking any medication for 1 month prior to admission; 21 were taking narcotic agents alone; and 18 were taking benzodiazepines alone. Thirteen of the 18 patients taking benzodiazepines alone had an EEG, as well as the full battery of psychological tests (WAIS, Memory Quotient, and Bender-Gestalt); 13 of the 21 patients taking narcotics alone also completed the same battery of tests. Finally, 5 of the 6 patients not taking any medication were also able to complete the entire battery of tests. When these 31 patients were compared using a chi-square analysis (see table 1), there was a significant impairment in cognitive functioning among the persons taking benzodiazepines compared with the patients not taking drugs or narcotics. When this finding was further assessed, using the measures of cognitive functioning alone, regardless of the EEG outcome, it was found that 10 of the 13 patients taking benzodiazepines had signs of cognitive impairment, compared with 4 of the 13 patients taking narcotics alone (p<.02, chi-square analysis; see table 2). None of the 13 patients taking narcotics alone exhibited any EEG changes, while 8 of the 13 patients on benzodiazepines had 12–20 cps or 15–30 cps diffuse fast-wave activity present in a large amount. The difference between these groups of patients was significant (p<.001; see table 3). Finally, it was found that none of the patients taking narcotics demonstrated both EEG changes and impairment in cognitive functioning, compared with 7 of the 13 patients taking benzodiazepines (p<.002; see table 4).

It is significant that 40 of the 106 patients were taking both a benzodiazepine and narcotic at the time of admission. Of these 40 patients, 37 completed the battery of tests; when they were compared with the 13

patients taking narcotics without benzodiazepines or any other sedative drugs, a highly significant difference ($p<.001$) was found (see table 5).

DISCUSSION

The pronounced sedative effect produced by benzodiazepines on EEG activity is well-documented in the literature (8), but unless toxic proportions are reached, narcotics do not produce any measurable EEG effects (with the possible exception of alpha peak slowing) (5, 9, 10). In addition, when narcotics are given at the dosages commonly seen in clinical practice, they do not impair cognitive functioning, memory, or performance of visual and motor-perceptual tasks (5, 11). By comparing the two populations that were mutually exclusive, that is, the patients receiving benzodiazepines alone versus those receiving narcotics alone, one could conclusively state that benzodiazepines were far more likely to produce cognitive impairment, with concomitant EEG changes, than were narcotics. Finally, it is apparent that any cognitive impairment noted in the patient receiving both narcotics and benzodiazepines is far more likely to be due to benzodiazepines than to narcotics.

While neither narcotics nor benzodiazepines should be used on a long-term basis, cognitive impairment was far more apparent with the latter class of drugs. The question of the reversibility of the benzodiazepine effect is the subject of current research, but at this time one may only underscore a recent suggestion by the Food and Drug Administration (12) that benzodiazepines be limited to short-term use (i.e., 4 months).

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Why Psychiatry Is a Low-Paid Medical Specialty

BY STEVEN S. SHARFSTEIN, M.D., M.P.A., AND HARRY W. CLARK, M.D., M.P.H.

The authors examine the effect of economic forces on the income of psychiatrists and compare the economic position of psychiatry with that of the rest of medicine. Since 1970 the income of psychiatrists has been losing ground compared with that of other medical specialists, although recent data suggest a possible improvement in the economic position of psychiatry. The authors believe that psychiatrists' incomes are important as a sign of the value of psychiatric care and as one measure of the economics of practice, that is, the incentives related to the distribution of scarce technology and labor in the service delivery system.

In May 1975 at the 128th annual meeting of the American Psychiatric Association, Dr. Bertram Brown, then Director of the National Institute of Mental Health, predicted, "Psychiatry will continue to grow in size and diversity" (1). In recent years, however, the number of American medical graduates selecting psychiatry has declined and the percent of all graduates has decreased. Since 1970 psychiatrists in private office practice have consistently earned less than other medical practitioners. This paper will examine the effect of economic forces on the income of psychiatrists and relate the economic position of psychiatry to that of the rest of medicine. There are some recent signs that the precarious situation of psychiatry is being improved through better coverage under health insurance, better peer review, and an enhanced status as a medical subspecialty.

THE ECONOMIC POSITION OF PSYCHIATRISTS

Since 1968 the American Medical Association has systematically collected data on the income of U.S. physicians. Since 1970 psychiatry has been losing ground compared with the rest of medicine. Table 1 shows the change within the years 1970–1976. It was during this time that psychiatry lost the most ground. The average net income of office-based psychiatrists increased less than 4% from 1970 to 1974, while the average net income for all U.S. physicians increased more than 20%. As compared with 1970, those contemplating a career in psychiatry during the years 1971–1976 could expect substantially lower earnings than most other medical specialists. In fact, in 1973 and 1974 psychiatry was the lowest-paid medical specialty, with family practice (i.e., both general practice and family practice) and pediatrics being slightly ahead of psychiatry. In 1975 and 1976 psychiatry managed to gain some ground compared with family practice and pediatrics but remained substantially behind internal medicine and the surgical specialties. To be truly comparable, income should be tracked for physicians in the same cohort over time to compare relative productivity rates. The American Medical Association data (2) do not allow for this analysis. Subsequent collections of data are necessary to develop a more rigorous analysis.

Another factor to be considered when comparing average income is the influence of the entry of a disproportionate number of physicians into a given specialty in any one year. This situation would occur if situational factors encouraged a large number of new graduates to select a given specialty. Factors that encourage a large number of existing specialists to reduce their practice or otherwise decrease their productivity are also important, for these developments would slow the growth of the average income for the affected specialty. Furthermore, if a significant number of physicians in a given specialty tended to switch after a decline in productivity, this would leave that specialty with a higher average income. There is no reason to believe that a larger number of individuals selected psychiatry as a specialty during the years addressed by this paper. In 1970, 10.3% of all U.S. and Canadian first-year residents were in psychiatry programs, but this figure dropped to 7% in 1974 and 5.6% in 1976.

However, the relative positions of pediatrics and family practice may have been affected by an influx of new graduates in the early 1970s, causing a slowing of income growth rates for those two specialties.

The impact of technology, labor, investments, and the state of the economy are all critical variables when

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one attempts to comprehend the changes, both long-term and short-term, observed in any income trend. In 1972 and 1973 wage and price controls for medicine were in effect. According to the Congressional Budget Office, from August 1971 to April 1974 the average annual percentage change for physicians' fees was 3.2%. The impact of the economic stabilization period on psychiatrists' income was basically negative. The most negative year for psychiatry, 1971, was a time when all specialties seemed to be enjoying a positive year. Further; 1971–1973 was a poor period for psychiatry compared with other specialties. These three years are important to consider. It could be assumed that the wage and price controls of 1972 and 1973 had a significant impact on the income of all medical specialties. However, in 1971 psychiatrists experienced a significant decline in income from 1970. What this means is not quite clear, since it predated the imposition of wage and price controls. The cloudiness appears because in 1970 there was a 17.9% increase in the incomes of psychiatrists over 1969; this represents a significant rise in fees in 1969. By 1971 wage and price controls made it more difficult to raise fees significantly, so that psychiatry was basically stuck at the 1969 level. Insurance companies, which ratify "usual and customary" fees, helped keep the fees low during 1971–1973. By 1971 psychiatry began its slide in relation to pediatrics and family practice, as well as the rest of medicine.

One must be careful about drawing cause-and-effect conclusions about the relationship between the income of physicians and the economy. However, it would appear to us that psychiatry is more sensitive to a depressed economy than are the other medical specialties. This might suggest that people view assistance from psychiatrists as more discretionary than most medical care and even a luxury during a period of economic recession.

This finding contrasts directly to studies done by Professor N. Harvey Brenner of Johns Hopkins University (3), which show that admissions to mental hospitals rise in times of unemployment and decrease during periods of economic upturns. Clearly, there are more mentally ill individuals during these periods of economic downswing who are unable to afford private psychiatric care, and there is a shift of psychiatric patients from the private sector to the public sector.

ECONOMIC DISADVANTAGES FOR PSYCHIATRISTS

Psychiatry is both labor-intensive and time-intensive. It has not been able to keep up with high-technology medicine and has not been able to divide its services into discrete technological elements for which there can be individual charges. Perhaps the one exception to this occurs in general hospital psychiatry, where insurance coverage is better and provider and facility charges can be broken down into various units such as individual therapy, group therapy, and milieu therapy.[1] Psychiatrists are paid for their direct time spent with the patient, and this makes psychiatry less profitable than surgery, anesthesiology, radiology, and internal medicine.

TABLE 1
Average Net Incomes of Office-Based Psychiatrists Compared with All U.S. Medical Specialists for 1970–1976[a]

Year	Psychiatrists	Percent Change	All U.S. Specialists	Percent Change
1970	$39,986		$41,789	
1972	39,124	−2.1	47,240	+12.7
1974	41,258	+5.5	51,997	+9.9
1976	47,565	+15.3	59,544	+14.0

[a]Data are from *Profile of Medical Practice* (2).

Further, private health insurance usually continues to place special limits on coverage for psychiatric treatment, especially outpatient psychotherapy. These limits consist of higher copayments, lower numbers of visits, and lower dollar limits than for other illnesses. Fees (usual and customary) paid by third parties have remained fairly stable over time. One economic analysis[2] indicated that in 1973 if the copayment rate for psychotherapy had been 20% across the board, the average fee would have been $74.20 instead of the prevailing fee, which was $35.00. In the rest of medicine third-party insurance has increased the income of medical specialists, so physicians' incomes—by and large—have been able to outstrip the rate of inflation, which since 1970 has been considerable. Physicians earning $33,000 in 1970 would have had to earn $58,000 in 1978 to keep pace with the economic changes caused by inflation and taxes. Psychiatrists have clearly not kept pace with their medical colleagues or with the rate of inflation (4).

The most recent data (for the years 1974–1976) suggest that psychiatry is improving its economic position. Since psychiatry has been placing increased emphasis on peer review and accountability, insurance benefits have slowly but surely expanded in the private sector, and serious consideration is being given to inclusion of psychiatric benefits under national health insurance and expansion of psychiatric coverage under Medicare. If these trends continue, the early 1970s might in fact be considered an aberration in the long-term growth and health of psychiatry.

Productivity factors such as the number of hours of practice per week, the number of weeks of practice per year, the total number of patient visits, and the number of hours of direct patient care per week are important elements in a productivity calculation. In each of

[1]Milieu therapy has been successfully challenged by several insurance companies as a discretely billable item.

[2]T. McGuire: The market for psychiatric services, unpublished paper.

these categories, psychiatry is below the national average for medicine and below both pediatrics and family practice. While it is clear that the average number of total patient visits for psychiatrists should be low, given the 50-minute-hour service unit, the other factors that contribute to an apparent lower level of productivity may either be inherent in the practice of psychiatry or in the work characteristics of those who select psychiatry as a specialty.

Competition from nonmedical mental health professionals has intensified in recent years. In many states psychologists have won recognition as independent practitioners and do not need physician referral in order to receive reimbursement for psychotherapy. Efforts to gain similar recognition are being made by social workers and psychiatric nurses. Success of such efforts would lead to some price or market competition for psychiatrists, although the latent demand for psychiatric and other mental health services is so great that there are more than enough patients to go around. The larger issue is the impact on the financing mechanism of a large supply of practitioners. CHAMPUS data (5) show that clinical psychologists usually charge the same fees as psychiatrists, so that there is no price competition with psychiatrists. Social workers, on the other hand, charge less and price becomes a factor in the supply and demand issue.

In Canada the introduction of national health insurance improved the economic position of psychiatrists. Psychiatric income rose 70% over a 7-year period, compared with an average for all medical specialties of 62%. Yet psychiatry is the lowest-paid medical specialty in Canada. Liptzin (6) concluded, "Psychiatrists simply could not generate the additional or more expensive services that other specialists could."

Some policymakers have asserted that if psychiatric treatments had proven their effectiveness, there could be greater third-party coverage and more value ascribed to these treatments. Although the effectiveness of many medical/surgical treatments (such as coronary bypass surgery) is in doubt, there is excellent third-party reimbursement. The problem, then, is that along with psychiatric patients, psychiatric treatments are stigmatized, given less value, and underfinanced.

One area that requires further study is the impact on the psychiatrist of raising his or her hourly fee. This "fee-countertransference" often takes the form of guilt feelings on the part of psychiatrists, who do not feel themselves worthy of $75–$100 per hour remuneration. Hourly fees (in 15-minute segments) for lawyers, on the other hand, are in the $100–$200 range, and surgeons think nothing of charging several thousand dollars for one operation. These professionals feel that they are entitled to such payments for the services they render. Psychiatric care is more open-ended and a continuous drain of money for patients, and the outcome is less certain. The psychiatrist's relationship with the patient is a key issue, and fees play an important role in the definition and structure of the relationship (7).

A final issue is equity. The highest earnings for psychiatrists are located in the private sector, often in communities with large numbers of psychiatric practitioners, such as Washington, D.C. Currently, most psychotherapy is used by college-educated upper-middle-class white persons.

This pattern of usage is reinforced by the current distribution of insurance coverage. It must be convincingly demonstrated that psychotherapy treatments are valuable and relevant to the lower socioeconomic classes so that expanded third-party coverage through national health insurance would not be a subsidy of the rich by the poor. A recent study of federal employees in Washington, D.C. (8), showed that 26% of patients were grade 9 ($17,000–$22,100) or lower and 59% were grade 12 ($24,700–$32,000) or lower. This would indicate that a substantial number of middle-class individuals use psychotherapy when insurance coverage is adequate.

Psychiatrists' incomes, therefore, are important as a sign of the value of psychiatric care and as one measure of the economics of practice, that is, the incentive related to the distribution of scarce technology and labor in the service delivery system.

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Results of Clinical Renal Function Tests in Lithium Patients

BY ROBERT H. GERNER, M.D., JAMES PSARRAS, M.D., AND MICHAEL A. KIRSCHENBAUM, M.D.

Because there have been reports suggesting that patients who receive lithium are at risk for renal damage, the authors carried out extensive noninvasive testing of renal function in 43 patients who had been taking lithium for from 1 to 120 months. Their only abnormal finding was that the urine concentrating ability of these patients was moderately but asymptomatically impaired. They suggest that although patients receiving lithium should be carefully evaluated and tested, there is not enough evidence to justify not initiating or continuing lithium use in patients who might benefit from it.

The use of lithium has revolutionized the treatment of manic-depressive (bipolar) illness and is, without a doubt, the single most effective method of treatment and prevention of this disorder (1), although use of carbamazepine (2, 3) and spironolactone (4) is under investigation. Three decades of experience with lithium have found it to be well tolerated and to have few apparent side effects when given in a well-controlled manner (5). Recent reports of possible serious renotoxicity have cast some doubt on the apparent safety of lithium. Since approximately 1% of the general population has bipolar (manic-depressive) disorder, and a larger number have recurrent unipolar depression that might be responsive to lithium (6), the possibility of serious side effects raises an important public health question. At the heart of all such treatment versus risk questions lies the trade-off of the ratio of another or no treatment to the risk inherent in the treatment.

A wide range of renal side effects associated with lithium have been known for many years. One of the most striking is the incidence of polyuria and polydipsia associated with lithium treatment, which was originally reported by Schou (7). Many other nonpathological effects of lithium on renal physiology have been reported (8–14); these have been reviewed by Jenner and Eastwood (15). More recently, several authors have suggested that lithium therapy may result in chronic, irreversible alterations in renal function and perhaps in structural renal abnormalities shown by biopsy (16–23). However, others have reported no difference in abnormalities obtained by biopsy in patients about to start taking lithium compared with those who had been taking it for an average of 5.5 years (24).

Because these reports suggested a risk to patients receiving lithium, we carried out extensive noninvasive testing of renal function in 43 patients at the Affective Disorders Clinic of the University of California, Los Angeles. Renal biopsy was not done because it carries a morbid risk. We felt it important to determine if there were clinically reliable and safe tests that might differentiate patients at risk for diminished renal function on lithium. In addition, we thought it important to conduct tests that could feasibly be carried out in most patients who are taking lithium rather than concentrate on those tests which require an extraordinary degree of time and technical support.

METHOD

We studied 43 patients (23 women and 20 men aged 17–67). All were bipolar I or II as diagnosed by the Research Diagnostic Criteria (25). Patients had been taking lithium for 1 to 120 months (mean, 26.8 months). All patients were euthymic at the time of urine collection, and none had diabetes, hypertension, autoimmune diseases, or renal disease before starting lithium. Patients were given detailed written instructions and an oral explanation by a physician to facilitate compliance. On day 1 at 7:00 p.m. the patients took their final dose of lithium and then ceased oral intake of food and fluid. A specimen of urine was taken 12 hours later for determination of urine osmolality to assess concentrating ability. The patients then started a 24-hour urine collection. At the end of the collection period, a blood sample was collected to determine lithium level, creatinine, blood urea nitrogen (BUN), uric acid, electrolytes, and osmolality. A spot urine was obtained for amino acid chromatography. Complete history regarding the patient's use of lithium; side ef-

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fects; use of other medications, alcohol, and medications known to affect renal function; and medical history was obtained at this time. Glomerular filtration rates were estimated by endogenous creatinine clearance. Ideal creatinine clearance (C_{cr}) for each individual was calculated according to the method of Cockroft and Gault (26) (we used lean body weight to deal with differences in muscle mass that may affect creatinine levels):

$$C_{cr} = \frac{(140-\text{age})\ (\text{lean body weight in kg})}{72 \times \text{blood creatinine (mg/100 dl)}}$$

The ideal creatinine clearance for women was calculated at 90% of that derived using the formula for men. Chemical determinations were performed using standard laboratory techniques.

RESULTS

Glomerular Filtration Rate

Plasma creatinine (normal range, 0.7–1.3 mg/dl) was obtained for 42 of the patients (mean, .97, SEM, .025). Only 1 patient, a husky 25-year-old man who had been taking lithium for 24 months, had a plasma creatinine level greater than normal (1.4 mg/dl). His creatinine clearance was within normal limits (134 ml/minute). Creatinine clearance could be calculated for only 40 patients because of laboratory errors. Three patients were not included because their urine collections were incomplete and they therefore had extraordinarily low creatinine clearances. No statistical differences were noted when the actual creatinine clearances (mean, 93±5 ml/minute) of these patients were compared with their ideal creatinine clearances (mean, 86±4 ml/minute) by group or paired t test. When the ratio of the ideal to the observed creatinine clearance was calculated, 10 patients were found to have a ratio of less than 1, suggesting a decrement in their creatinine clearance. In 8 of these patients the decrement was greater than 10% and the duration of their lithium treatment, which ranged from 1 to 120 months, did not differ from that of the other patients. There was no significant correlation of decrement in creatinine clearance and duration of lithium treatment in these individuals, nor in the group as a whole. The plasma creatinine levels of these patients was normal (range, 0.7 to 1.0 mg/dl). None of these patients had a documented history of lithium overdose, although 1 patient had overdosed on barbiturates and tricyclic antidepressants. No patients in this group had proteinuria.

When the creatinine clearance was calculated by age, the expected decrease with age was observed. However, this age-related decrease was less in our lithium-treated patients than was predicted, and the slope of the observed lithium/age correlation ($r=-.10$) was less ($p<.10$) than the ideal slope ($t=-.68$), showing that lithium did not exacerbate the well-known decrease in creatinine clearance associated with aging.

24-Hour Urine Proteins

Three patients had minimally elevated levels of protein: 1 woman and 2 men (aged 28, 31, and 37, respectively; all had received lithium for 24 months). They excreted 1.24, 1.03, and 1.07 g protein/24 hours, respectively. Creatinine clearances in these patients were not low (actual/calculated ideal: 95/97, 139/105, and 141/71). No other abnormalities were present on any of the other tests except for dehydration osmolality, which was quite low in 2 of the patients (383 and 395 mosmol/liter), while in the 37-year-old man it was 773 mosmol/kg H_2O.

Blood urea nitrogen (normal, 8–20 mg/dl) was slightly elevated in 4 patients, whose BUNs were 21, 22, 23, and 25. All 4 of these patients had normal creatinine clearances and no proteinuria.

Uric Acid

Serum uric acid was slightly elevated in 2 women (6.4 and 8.6 mg/dl) and 4 men (7.5, 7.5, 7.7, and 7.9 mg/dl) (norms: men, 4.2–7.4, and women, 2.7–6.1 mg/dl).

Urine Lysozymes

Urine lysozymes were not elevated in any of our patients (mean, 1.14±.16, norm, 0–4.6).

Amino Aciduria

Urine amino acid chromatography revealed minor elevations in methylhistadine in 2 patients and glycine in 5. In 4 patients who had repeat examinations, no sustained elevations in amino acids in the urine could be detected, suggesting that dietary factors contributed to the original mild abnormal findings.

Maximal Urine Concentrating Ability

As has been reported consistently in the past, our patients appeared to have a defect in maximal renal concentrating ability; their mean urine osmolality was 602 mosmol/kg H_2O (range, 100–965). None of the patients was hypernatremic. There were no significant ($r=-.22$) correlations of urine osmolality by duration of time on lithium.

Lithium Levels

Lithium levels in all of the patients ranged from 0.6 to 1.2 mEg/liter. Two patients who had a history of overdose with lithium had normal values for all tests. Virtually all of the patients had received other psychoactive medications, including neuroleptics, tricyclic antidepressants, and MAO inhibitors.

DISCUSSION

Our findings confirm those of previous investigators that urine concentrating ability is moderately but usu-

ally asymptomatically impaired in patients receiving lithium. This is the only abnormal finding in our patients as a group. We did not find any elevation in urine lysozymes, which may be elevated early in the presence of parenchymal kidney disease, nor did we find amino acids in the urine, which might reflect proximal tubular dysfunction, as in the Fanconi syndrome. Creatinine clearance for the entire group was slightly better than predicted. A few patients had low creatinine clearances (63%–94% of that predicted). Although the most likely reason for these values is inaccurate collection, this finding suggests that a few patients taking lithium may have a decrease in the glomerular filtration rate; this is not necessarily due to lithium, however. The mild proteinuria experienced by 3 of our patients suggests a defect in glomerular permeability, although it was not demonstrated that this was associated with lithium in these patients. We did not find an associated abnormal creatinine clearance in those patients with slightly elevated plasma creatinine or BUN, suggesting that these elevated values were not related to abnormal renal function. All of the patients who had slight elevations of these tests had creatinine clearance levels that were better than expected. There was no relationship between duration of lithium treatment and creatinine clearance or age-related changes in creatinine clearance.

Our findings suggest that noninvasive clinical testing of patients taking lithium does not reveal consistent abnormalities suggestive of seriously impaired renal function. It appears from our limited data that plasma creatinine and BUN alone are not sensitive enough to use as screening tests for patients receiving lithium because they do not correlate well with the slight abnormalities in creatinine clearance or 24-hour urine protein excretion that were observed in a few patients.

Renal biopsy was not warranted in any of our patients. Rather, we feel that those patients who have proteinuria and/or a decrease in their creatinine clearance should continue receiving lithium and have the test repeated to correct for possible collection or laboratory errors. If the findings are still abnormal, the patient should have a more extensive evaluation and be followed at intervals of 6 months to 1 year. It is not possible at this time to make a judgment on how frequently such tests should be performed on individuals who do not have an initial abnormal value. It is very important to note, however, that the calculation of the ideal creatinine clearance must be done with care because in many individuals with low muscle mass normal uncorrected creatinine clearance values may be lower than those commonly perceived as normal.

Because of the considerable difficulty in obtaining consistent and reproducible urine collections, we have the following recommendations for collection of urine for protein and creatinine clearance that may facilitate outpatient as well as inpatient screening. We suggest that urine collection be done while the patient is at the clinic or office, much as they would be for a glucose tolerance test. Valid calculations for creatinine clearance and urine protein concentration can be gathered by having patients come to the clinic, void, and collect all subsequent urine for the following 4–6 hours. Encouraging patients to drink ample amounts of fluid during this time will result in a greater urine output and more accurate results. All of our patients were quite cooperative with the study; no one stopped taking lithium because of fear of kidney problems. After testing and having the results explained to them, virtually all of the patients expressed relief and gratitude at the interest that was being taken in them and the seriousness with which their condition was being viewed.

In conclusion, we feel that the tests used, which include many that are generally available as well as specialized tests, did not reveal gross clinical impairment in renal function in patients who had been receiving lithium for from 1 to 120 months. We did not find a decrement in function by duration of time on lithium in any of the tests. Although caution and careful psychiatric evaluation of all patients taking lithium need to be maintained, there does not at present appear to be sufficient evidence to justify not initiating or continuing lithium use in patients who might benefit from it. Until the issue is more fully resolved, it may be prudent to obtain, if feasible, creatinine clearance and urinary protein values in patients before or soon after initiating lithium treatment and to retest them 1 year later and periodically thereafter. We agree with Vestergaard (27) that it would not be wise to alter current treatment practices of manic-depressive patients on the basis of preliminary reports of renal toxicity until large-scale investigations are available that will help the physician weigh the benefits and hazards of lithium treatment. Future studies should control for the many other medications most bipolar patients have been exposed to and, additionally, should consider possible renal effects of manic-depression per se.

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Board Certification Anxiety

BY FRANKLIN G. MALESON, M.D., PAUL J. FINK, M.D., AND HOWARD L. FIELD, M.D.

The authors discuss how stress influences the candidate's capacity to effectively prepare for and fully demonstrate his or her abilities in the oral examination in psychiatry given by the American Board of Psychiatry and Neurology. They highlight subtle misconceptions about examiner priorities, attitudes, and evaluation methods and note common errors that reflect both anxiety and these misconceptions. They offer some advice and information to aid the candidate in coping with these examination difficulties.

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Published reports (1-3) in addition to personal accounts suggest that the average candidate takes the American Board of Psychiatry and Neurology certification examination in a state of intense and occasionally disorganizing anxiety. Endless anecdotes attest, such as keys being lost, pipestems crunched, and eyeglasses broken in the simple process of wiping. In the examination, word reversals, amnesias for fundamental information, the assumption of bizarre interviewing postures, and the like are common. One author somewhat hyperbolically describes this state as a "pseudodelirium" (1).

Although some candidates may have neurotic difficulties in taking examinations, the conscious and generally shared sources of oral Board examination anxiety are well known. Preparation requires major personal sacrifice, which candidates and their families are loath to repeat. Pride, reputation, and academic and professional opportunity are often considered to be at stake. These anxieties are brought into a test situation that offers its own unique stresses. Oral examinations have a potential for subjectivity and lack of uniformity in grading. The candidate may fear that his patient will

be highly unusual or uncooperative and that the resulting difficulties will be marked against him. He may fear that the examiners will be capricious, sadistic, or unduly focused on their own narrow clinical or research interests. In short, a helpless anticipation that "anything can happen" prevails. A recent call for a moratorium on the oral examination by more than 200 APA members reflects these concerns (4).

As Board examiners, we have witnessed a variety of performance impairments seemingly derived from stresses specific to the examination situation.[1] Successful candidates are not immune, and it is likely that some who are unsuccessful would be found qualified were it not for examination-specific difficulties. Most of us know colleagues whom we regard as highly competent and yet who have at one time failed or conditioned Part II. Most examiners are familiar with the seemingly adequate candidate whose entire performance disintegrates after he is stumped by one or several questions. About 18% of the candidates who take Part II pass on a second or later attempt (5). Undoubtedly some of these successful repeaters correct genuine clinical deficiencies identified in the first examination—an ideal examination function. It is likely, however, that a large number of successful repeaters do not become more competent in any substantive way but rather develop better test-taking strategies, practice more under simulated test conditions, or have lower levels of anxiety as a result of knowing better what to expect. These speculations have been supported by preliminary analysis of free response data in a recent APA survey designed by the 1977–1978 Ad Hoc Committee to Implement the 1976 Referenda on Psychiatric Certification.[2]

The following information and remarks, offered to aid the prospective candidate by highlighting some typical misconceptions and stress reactions that interfere with maximal demonstration of capability, pertain to the make-up and modus operandi of the examining teams, the preparation process, and the psychological and interactional phenomena in the actual test situation (especially the 30-minute question and answer period when the candidate feels most vulnerable).

THE EXAMINER'S PERSPECTIVE

In the instructions to examiners the following paragraph appears: "Avoid discussion of abstract, obscure matters of questionable importance. If areas are uncovered in which the candidate is not informed, do not dwell exclusively on these topics. Find out what else he may know. . . . Do not restrict the examination to one type of philosophical orientation" (6).

All the examiners we have observed have essentially followed this instruction and failures do not result from the candidate's ignorance of "minutiae." Occasionally a highly technical question is asked but rarely out of context. Sometimes examiners ask such a question to find redeeming areas of special competence in a candidate whose performance is otherwise poor. For example, a candidate who was weak in his discussion of differential diagnosis and hospital management of his manic patient was asked about the significance of platelet monoamine oxidase activity. Although this was a seemingly narrow point, he seemed to know about psychopharmacology and was given an opportunity to demonstrate a deeper knowledge in this area.

Examiners are very concerned with *how* a candidate thinks about a patient, i.e., how he weighs and sorts clinical evidence. Some candidates experience difficulty organizing and articulating case summaries, presenting differential diagnoses, and discussing treatment; rambling and vagueness in these areas have been noted elsewhere (1). To some extent such difficulties may be related to anxiety and may abate after the candidate has had practice in simulated examinations.

It is helpful for a candidate to avoid a guarded or reticient posture and to talk openly about what he knows and how he has reached his conclusions; for example, "The patient's vagueness and history of head trauma made me suspect an organic brain syndrome, but I was surprised when he did so well on mathematical and memory tests." Similarly, there appears to be no reason for a candidate not to mention areas that he may have neglected to explore during the course of his patient interview. This at least demonstrates an awareness of what was missed, which is obviously preferable to the examiner's having to extract such information or concluding that it would never have occurred to him. (Some typical examples include eliciting a family history of mania with a depressed patient and exploring amphetamine use with evidence of a transient paranoid psychosis.) It is also advantageous for the candidate to acknowledge areas of unfamiliarity, to comment on issues felt to be controversial, and to request clarification of an examiner's question when necessary. What examiners are seeking is competence (3, 7, 8) not brilliance or some hypothetically perfect interview.

IMPROPER EMPHASIS IN PREPARATION

The candidate must enter the examination with broad expectations of the range of psychopathology he may encounter and the kinds of issues he will be asked

[1]Dr. Fink has been an assistant and associate examiner for nine years. Dr. Field has been an assistant examiner twice, and Dr. Maleson once. They have observed and conducted numerous examinations.

[2]S.K. Quick, Associate Director, Office of Education, American Psychiatric Association, Washington, D.C., 1979 (personal communication).

to discuss. With the prospect of seemingly endless possibilities, some may have subtly and unconsciously narrowed their mental set, much to their disadvantage. A number of candidates seem surprised to encounter patients who are not overtly psychotic, brain damaged, or addicted; it appears they are not prepared to consider less profound degrees of impairment. One candidate was startled when he belatedly realized that he had not included hysterical conversion in his differential diagnosis, in spite of highly suggestive symptoms and his familiarity with this diagnostic entity. Another candidate, who incorrectly assumed that her patient was hospitalized, wasted more than 10 minutes of precious interview time in a confused misinterpretation of the man's history and never regained her composure after an examiner told her about the patient's outpatient status. (This last anecdote confirms the wisdom of the candidate orienting himself fairly early, in a graceful fashion, to the specifics of the patient's overall situation.)

Many candidates do not seem geared to apply such familiar topics as psychodynamics, principles and problems of psychotherapy, and hospital management to their patients. In contrast, almost all are well versed in the use of psychotropics, particularly lithium, and biological psychiatry in general. Insufficient expertise in the more purely psychological matters could certainly account for some of this trend. Nevertheless, most psychiatrists have had considerable exposure in their training and daily activities to nonbiological issues, so it is possible that these matters are harder to talk about, particularly for those who are not very articulate. Or some candidates may harbor an unwarranted expectation of examiner bias against psychodynamics and psychotherapy, as if these were unfashionable. They may thus be caught by surprise or perhaps even fear being drawn into statements that will prejudice the examiners against them. Others may be overconfident about their ability to verbalize ideas in familiar areas and prepare disproportionately in areas in which they feel vulnerable to factual error.

It is useful to test and practice the presentation of one's skills in multiple clinical reference frames: psychological, biological, diagnostic, therapeutic, and prognostic. Difficulties in discussion of fundamental issues are a much greater source of failure than the feared questions about psychiatric obscurities.

THE EXAMINATION CLIMATE

Another instruction to examiners states, "There is no place for sarcasm or humor at the candidate's expense, or attitudes of condescension or cool hostility. Make every effort to put the candidate at ease and to encourage his best performance" (6).

Although it is likely there are examiners who are unwilling or unable to adhere to these guidelines, our experience and that of others (1, 9) indicate that most examiners struggle for clarity and fairness in their questions and privately empathize with the candidates. Many primary examiners are not far removed from their own stressful experiences as candidates and note a recrudescence of their anxieties during their orientation. They report feeling quite uncomfortable when their fellow physicians stand terrified before them. Candidates with such overwhelming anxiety are a minority but are by no means rare, and it is often difficult to calm them.

The problem is intensified by the requirement that feedback and reassurance, the responses so needed by the especially anxious candidate, be minimized during the examination in order to avoid excessive steering or suggestion of answers. In addition, any adverse examiner comment during the examination, however delicately worded, risks inflaming existing anxiety. Even when a candidate appears to be doing well to one examiner, the need to consult other examiners before arriving at a final grade requires caution in any offering of reassurance. There may not be general agreement among examiners about a candidate's performance and to reassure someone who later fails would only heighten postexamination resentment.

Thus, with many subtle variations, most examiners attempt the difficult task of being friendly and at the same time pointedly neutral. Some could be described as inscrutable; others limit feedback to barely perceptible nods of the head or other cautious agreements. It is easy to understand how the anxious candidate in search of positive signs can interpret such strained postures as cool hostility, condescension, or even impending failure and why it has been a source of resentment and criticism (3, 10). If the candidate does not anticipate a great deal of examiner responsiveness, he may be less flustered by its absence.

This unavoidable limitation of candidate-examiner rapport was exemplified by the reluctance of a number of candidates to revise statements or diagnoses that they themselves had made untenable by their later remarks. It is not always easy for examiners to decide whether this phenomenon reflects deficient clinical ability or an examination artifact, such as fear of being tricked by the examiner, stress-induced rigidity, or a notion that any retraction reveals a fatal indecisiveness. In these situations the candidate was usually not given the benefit of the doubt.

THE EVALUATION PROCESS

Different examiners tend to agree on the scores of the same candidates, although the backgrounds and fields of interest of examining team members are varied. Private practice, administration, teaching, and research are all represented. Expertise ranges from psychoanalysis to psychopharmacology (7). Although dif-

ferent examiners can obviously assess some areas more critically than others, it is clear that the examiner team functions with a satisfactory operational definition of competence.

The candidate is not likely (at least on a statistical basis) to be discriminated against by assignment to one team or another. Over the years there has been good team-to-team scoring agreement, with approximately the same number of candidates passing regardless of the leadership of the team and the clinical setting in which the examination takes place (7).[3]

Candidate performance is treated conscientiously during the evaluation process (1, 9). Examiners usually do not differ in grading by more than 1–2 points, a difference that is crucial only when it occurs around the scores of 70 and 75 (the minimum grades for conditioning and passing, respectively). In such marginal cases the several examiners who have observed the candidate typically spend extra time and energy in vigorous and sometimes agonized debate. This provides a measure of safety through a system of checks and balances (1, 9).

When examiners confer, views appear to be stated with independence and mutual respect. In general, the senior examiners, who usually spend no more than 10 or 12 minutes with any candidate, are willing to defer to the primary examiner when this is warranted. For example, one candidate had been doing quite well until he attempted to pretend knowledge about the contributions of some major researchers whose work pertained to his case. At this awkward moment the chief examiner of the section entered the room. Observing the confused and labored discussion he soon departed, convinced that the candidate would fail with little debate. During the evaluation, the other examiners explained that despite this apparent bluff the candidate's overall performance was good; he passed the examination.

Our remarks about the grading procedure will not satisfy those who question the existence of any uniformly acceptable or meaningful standard of excellence, at least as measured by such an examination (3, 11, 12). We hope that our description of the essential fairness of the grading process within the existing testing conditions will provide some reassurance to the candidate.

[3]R.I. Shader, Boston, Mass., 1978 (personal communication).

ANXIETY OR LACK OF CLINICAL COMPETENCE?

We hope that some of our comments will not be interpreted as useful strategems for the creation of a facade of competence by poorly qualified candidates. It is doubtful that any strategy could carry a truly weak candidate very far. Regardless of what one may think of the validity of the test as a measure of safe clinical competence, in order to pass it, there is no substitute for good training, extensive study, and practice under simulated test conditions. Our advice should aid the prospective candidate in coping with the stresses of the test situation so that his or her abilities are demonstrated to maximum advantage. Such a salutory effect would also aid the examiners. As examiners, we have often been in the dilemma of trying to distinguish between the effects of anxiety and insufficient clinical ability and have had the unwelcome and saddening task of flunking some candidates who we suspected were more knowledgeable than they were able to demonstrate.

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CLINICAL AND RESEARCH REPORTS

This section contains 1) new research findings, including preliminary data from pilot studies, either clinical or laboratory; 2) worthwhile replication studies; 3) case reports that describe a truly new syndrome or cast new light on established ones; and 4) case reports that indicate a new therapeutic procedure of potential value or call attention to adverse effects of drugs or previously unreported complications of therapeutic interventions. Program descriptions and literature reviews cannot be printed in this section. Criteria for format are listed in "Information for Contributors" in each issue; papers that do not adhere to these criteria will be returned to the author.

Anaclitic Depression in a Three-and-One-Half-Month-Old Child

BY THEODORE J. GAENSBAUER, M.D.

Since the classic studies of Spitz (1), it has been generally assumed that anaclitic depression does not occur in infants before the age of six months. Infants younger than this age have been thought to lack the maturity and specific attachment to the mother necessary to produce a depressive picture in reaction to her loss (2, 3). The infant described below, who had been referred to the author for a psychosocial evaluation by her social services careworker, has brought this assumption into question. The information to be presented in this report is based on social service and hospital records, including psychiatric evaluations of the parents; interviews with the mother, both foster mothers, and social service and hospital personnel; and direct observation of the infant.

Case Report

Jenny was the product of a normal pregnancy and delivery. Although her mother treated her at times more like a doll than a person, she was breast-fed, actively cared for, and almost constantly in her mother's presence. Jenny's father was affectionate but had a severe temper. When 2 weeks old, Jenny was found by her pediatrician to have fractured her arm; at 7½ weeks a small bruise was noted during a well baby clinic checkup, and at 3 months she was hospitalized with a second broken arm and a nondepressed skull fracture. On each occasion, the injuries occurred while Jenny was alone with her father. In spite of this severe abuse, neighbors and health care professionals who saw Jenny before her hospitalization and observers in the hospital described Jenny as a happy, highly sociable baby who smiled readily and showed no evidence of withdrawal. Bonding and eye contact with her mother were noted to be good. During Jenny's hospitalization, the only observable effects of the recent abuse were visual hypervigilance, a tendency to squirm when held by unfamiliar people, and increased fussiness, which was no doubt partly related to pain from her injuries.

After a 5-day hospitalization, Jenny was taken to a foster home. This resulted in an abrupt weaning, loss of contact with her mother, and a significant decrease in attention from those caring for her. The foster mother provided Jenny with minimally adequate physical care, but she left Jenny in her crib for long periods without stimulation.

During the next 3 weeks, Jenny and her mother were brought together by the Department of Social Services on three occasions. Her mother commented later, "Each time I saw her, the more out of touch she seemed to be." The first visit, 10 days after Jenny's transfer to the foster home, confirmed the positive relationship between Jenny and her mother. An onlooker noted that Jenny immediately recognized her mother's face and "lit up like a Christmas tree" with broad smiling and excited activity. One week later Jenny seemed more subdued during her mother's visit. Her mother observed that she cried more easily, was less active, had lost interest in her toys, and smiled less readily.

The third visit took place when Jenny was 3 months, 25 days old; she and her mother were evaluated at our play-

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room laboratory.[1] When they were brought together, Jenny's mother and a caseworker noted that Jenny failed to greet her mother positively and that it was difficult to get Jenny's attention. Jenny's appearance during the one-hour evaluation was that of a depressed child. Her hunched-over posture gave the impression that it was an effort for her to hold up her head. She seemed apathetic and disinterested in her environment and could not be engaged in play. Smiling was almost absent. She was easily upset and fussed when she was overstimulated. Jenny's facial expression during most of the session ranged from subdued to definitely sad. An independent videotape analysis of Jenny's facial expressions, utilizing objective scoring systems for the facial expression of discrete emotion (4, 5), confirmed the presence of a sad facies; specific facial components of sadness that were present included a drawing together and raising of the inner corners of the eyebrows and a downturning of the corners of the mouth. The rater observed the most clear-cut facial expressions of sadness during those times when her mother was present. The rater also noted that throughout the visit Jenny avoided eye-to-eye contact with her mother; gaze aversion was particularly unmistakable when her mother returned to the room after a brief separation. Viewers of the videotaped session, including several experienced clinicians, have consistently described Jenny's affective state as "sad," "pitiful," and "painful to watch."

Immediately following the playroom visit, at the request of the first foster mother, who felt overburdened by 3 other foster children, Jenny was placed in a new foster home with a warm and sensitive foster mother. The first day Jenny seemed subdued with a "kind of unhappy look." Her new foster mother also observed that Jenny tended to avoid face-to-face interaction. However, Jenny responded quickly to the optimal mothering during the next couple of days; she began to smile more readily and vocalized in response to her new foster mother's attention.

Five days after Jenny was transferred to her new foster home Jenny visited her mother at her mother's apartment. Jenny was now 4 months old. During the visit, Jenny was apparently quite fussy and her mother had great difficulty in soothing her. When she was returned to the foster home, the foster mother noticed that Jenny acted "coldly, like she was mad." The subdued facial expression, disinterest in people, gaze aversion, and lack of smiling that had characterized Jenny's first day in the foster home persisted for the remainder of the evening. She awoke that night crying, whereas the previous two evenings she had slept through the night.

Two weeks after her initial visit Jenny was evaluated again in our laboratory when she was 4 months, 8 days old. On this occasion she was evaluated with her new foster mother. Jenny's appearance at this time reflected a normally responsive 4-month-old child. She was engaging, alert, and curious about her environment; she smiled frequently and scored well on developmental testing. Her sad facies had disappeared. She still showed intermittent gaze aversion and a propensity to fuss when overstimulated, although these occurred in much more subtle forms.

Discussion

Jenny's history provides evidence that a depressive picture is possible in children at a much earlier age than has previously been assumed. Pathognomonic signs of anaclitic depression that were present in her case included a sad facies, withdrawal, apathy, psychomotor retardation, absence of pleasure, and a tendency toward irritability and distress (6). Jenny's mood disturbance was not a transient one; it emerged over a 3-week period and persisted for several days after adequate caretaking was reestablished.

The precipitating events for Jenny's depression, consistent with those previously described for anaclitic depression, involved a triple loss: separation from her actual mother, abrupt weaning, and affective and stimulus deprivation. The crucial factor in Jenny's depression was the loss of the mothering function rather than the specific loss of her mother. This is indicated by the prompt improvement in Jenny's depressive picture after her caretaking environment improved. Yet the evidence suggests that the loss of Jenny's relationship with her mother contributed to her depression. Jenny showed a clear and positive recognition of her mother during their first visit after their separation. Only as time passed did she develop depressive-withdrawal symptoms and specific avoidance of her mother's face. Further evidence for specificity in Jenny's reaction to the loss of her mother includes the finding that during the first laboratory visit Jenny's sad face was specifically associated with her mother's presence and the fact that after her visit with her mother following her placement in the second foster home there was a transient reoccurrence of the depressive-withdrawal picture.

It is likely that the sight of her mother's face awakened in Jenny, at some level of recognition, the affectively painful experience of her loss, resulting in sadness and fussiness whenever her mother was present. If this hypothesis is correct, Jenny's avoidance of eye-to-eye contact with her mother could be explained as a defensive measure used to avoid experiencing the depressive-affective state (7).

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Haloperidol and an Unrelated Sudden Death

BY ROBERT E. SMITH, M.D., NANCY N. LOW, M.D., AND HENRY A. NASRALLAH, M.D.

When sudden, unexpected deaths occur in psychiatric patients, the psychotropic medications the decedent had been receiving are often suspected of causing or contributing to the fatality. If the autopsy report does not fully explain the cause of death, there is further suspicion for a drug-related death. As a result, neuroleptics have been implicated in some sudden, unexpected deaths. In those cases when an autopsy does not support a definitive cause of death, the postulated mechanisms for neuroleptic-induced deaths include cardiac toxicity, vasodilatation and hypotension, hyperpyrexia, and the production of unspecified neurologic dysfunction.

We report below the case of a sudden, unexpected death in a young man who had received only moderate doses of haloperidol for control of extreme agitation. An autopsy was performed, but the cause of death was not determined until a detailed reexamination of the patient's heart was undertaken. The autopsy finding prevented this case from being another sudden death with no demonstrable cause of a patient who was taking neuroleptics.

Case Report

Mr. A, a 30-year-old married man, was first admitted to the hospital because of obsessions with homosexual thoughts. Multiple stresses preceded and surrounded the onset of his illness. As an outpatient, Mr. A had treatment that included psychotherapy and the administration of antipsychotic medications, which gave him some relief. By the time of his admission, however, his clinical condition had deteriorated to a severely anxious and, at times, psychotic state. While hospitalized, Mr. A was treated with antidepressants and psychotherapy. He was discharged after 8 weeks with his condition improved but tenuous. At the time of his discharge he was instructed to take 200 mg/day of amitriptyline h.s.

Three days after his discharge Mr. A was readmitted because of severe anxiety, poor sleep, suicidal ideation, and increasing impulses to harm his wife. In the hospital he continued to be very agitated. The amitriptyline was discontinued on the day he was admitted, and antipsychotic medication was reinstated. However, his condition continued to worsen, and he developed catatonia with bizarre posturing. These symptoms alternated with periods of extreme agitation. Because he made repeated attempts to injure himself during his periods of agitation, it was necessary to intermittently use four-point restraints. Throughout his hospitalization Mr. A was fully oriented, with an intact sensorium.

Two days after his admission the treating physicians noted clonic-type movements in his upper extremities. A neurological consultation ruled out seizure activity. Four-point restraints continued to be used during his periods of extreme agitation.

At 9:00 the next morning it was noted that Mr. A had respiratory difficulties. His heart rate was very rapid and his blood pressure was unmeasurable. An examination demonstrated no evidence of upper airway obstruction. Mr. A developed cardiopulmonary arrest, and resuscitation was immediately instituted. A cardiac team arrived and implemented full resuscitative measures, including placement of an endotracheal tube and intravenous pacemaker. Despite 45 minutes of ceaseless efforts, however, Mr. A's heart failed to respond and he was pronounced dead. During the 3-day period before Mr. A's sudden, unexpected death he had received a total of 47 mg of haloperidol. On the day preceding his death he was given 25 mg of haloperidol.

A preliminary autopsy examination revealed congestion and edema of the patient's lungs, liver, spleen, and brain. These are nonspecific findings, and although consistent with acute congestive heart failure, they do not explain the cause of death. Further pathology studies of the heart were undertaken to rule out the possibility of a conduction tissue defect or other disease that would not be grossly evident, and this examination determined the pathologic basis for Mr. A's death.

The left anterior descending and circumflex coronary arteries showed focal atherosclerotic narrowing to 50% loss of internal diameter. The coronary circulation was right dominant with focal narrowing to nearly complete obstruction of the right coronary artery from 4 cm to 8 cm from its ostium. Step sections through the right main coronary artery at the level of the most obstructed areas showed severe atherosclerotic narrowing with only a slit-like lumen remaining.

Microscopically, the upper and middle septum showed large areas of stretched myocardial fibers with first-, second-, and third-order waviness. The fibers were variably eosinophilic, and a rare fiber showed flocculated cytoplasm. However, cross striations were present and nuclei were only minimally pyknotic. The waviness extended into the area noted grossly to be thickened beneath the aortic valve ring. The conduction tissue of the AV node and immediately below it showed fragmented small fibers with pyknotic nuclei and congested small blood vessels. Some contraction bands were present. The muscle fibers high in the interventricular septum were enlarged with a tendency to whorl. These are diagnostic findings of early myocardial ischemic change (1, 2).

Mr. A's death, which occurred during acute congestive heart failure, was caused by acute myocardial ischemia with severe segmental coronary atherosclerosis present.

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Discussion

Sudden, unexpected deaths are not unique to the psychiatric patient population. The age-specific rate for sudden, unexpected death in apparently healthy individuals aged 20 years to 39 years is estimated to be 47 deaths per 100,000 population (3). Several studies have examined the causes of sudden, unexpected deaths in apparently healthy, young adults (3, 4). In one study (4) the most frequent causes of death were asymptomatic coronary heart disease and untreated infections. Twenty-eight percent of these patients died from coronary heart disease with or without recent occlusion. Another study (3) found that 61.4% of all sudden deaths were due to coronary heart disease. Because coronary heart disease is the leading cause of sudden death, it must be carefully ruled out before one reports a death as having no demonstrable natural cause and before one questions whether previously administered medications contributed to the death in an unspecified manner.

There are numerous problems inherent in the pathology of sudden death due to coronary heart disease. It has been estimated that routine autopsies underreport the correct proportion of occlusive coronary artery disease by more than half (5). However, even when occlusive arteriosclerotic disease is found, it is reported as an incidental finding unless evidence for myocardial ischemia or infarction is also present. Definitive histologic evidence may be difficult to find in many cases of sudden death because of the rapidity of the terminal event. Recent advances in the histopathological findings in early myocardial ischemia (1, 2) now permit a diagnosis of early myocardial infarction to be made. Such was the case in this patient. We suggest, therefore, that previously reported sudden, unexpected deaths in patients receiving neuroleptics may represent cases of occlusive coronary heart disease with myocardial infarction.

Psychiatric patients with asymptomatic coronary heart disease may be at an increased risk for sudden death because of agitation and heightened arousal secondary to their psychotic state (6). Neuroleptics may be indicated to reduce this risk. Haloperidol for sedation has been reported to be safe in moderate dosages (5 mg i.m. every 4 hours to 6 hours) for patients with acute myocardial infarction (7). Thus, it appears that haloperidol does not further impair a compromised coronary circulation.

To our knowledge, the literature contains only one report of a sudden, unexpected death in a patient receiving haloperidol and no other psychotropic medication (8). An autopsy of that patient reported no demonstrable cause of death. The authors suggested that an idiosyncratic response to haloperidol may have occurred. A similar conclusion may have been reached in this case if a reexamination of the decedent's heart had not been undertaken. Because asymptomatic coronary heart disease is the leading cause of sudden, natural death in young adults, this diagnosis must be carefully ruled out in any sudden, unexpected death involving a patient receiving psychotropic medication.

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Deficient Prolactin Response to Morphine in Depressed Patients

BY IRL EXTEIN, M.D., A.L.C. POTTASH, M.D., MARK S. GOLD, M.D., DONALD R. SWEENEY, M.D., PH.D., DAVID M. MARTIN, AND FREDERICK K. GOODWIN, M.D.

The discovery of opioid receptors and endogenous opioid peptides (endorphins) in the brain (1) has kindled interest in the possible role of endorphin systems in psychiatric disorders (2, 3). The analgesic, euphoric, and calming effects of exogenous opiates suggest that decreased functional activity in endogenous opioid peptide systems could be linked to the pathophysiology of depression. Opiate receptors and endorphins are widely distributed in limbic and hypothalamic areas thought to be involved in depression. Endogenous opioid peptides may modulate noradrenergic and dopaminergic neuronal systems that are implicated in the etiology and pharmacotherapy of depression.

There are anecdotal reports from the prepsychotropic era of the efficacy of opiates in treating depression. Researchers have observed significant depression in opiate addicts after detoxification. Some of these patients have been successfully treated with opiates and tricyclic antidepressants, suggesting that opiate discontinuation resembles the discontinuation of effective psychopharmacologic maintenance treatment. Administration of β-endorphins has ameliorated depressive symptoms in some patients (3). These data support the hypothesis that endogenous opioids influence the maintenance of pathological mood states.

We used a neuroendocrine challenge paradigm to study in vivo endogenous opioid systems in depressed patients. The normally pronounced increase in serum prolactin produced by morphine (4) was markedly blunted in patients with major depressive disorder.

Method

We studied 5 male and 5 female inpatients diagnosed as having major depressive disorder (9 unipolar patients and 1 bipolar patient) according to Research Diagnostic Criteria (5). Their mean age was 44±5 years; the range was 22–62 years. The control group consisted of 2 normal volunteers and 4 inpatients (4 women and 2 men) with a primary diagnosis of personality disorder. Their mean age was 33±8 years; the range was 19–62 years. All subjects gave written informed consent to participate in the study. Seven of the patients with major depression had used psychotropic medication during the 6 weeks before the study. Most of this use consisted of brief trials at low doses: 5 patients received neuroleptics alone; 2 patients received neuroleptics plus antidepressants. (During this time the control subjects had remained drug free.) Patients received no other medication except flurazepam for at least 1 week before the study. The patients fasted overnight; an indwelling venous catheter was inserted at 8:30 a.m. and kept open with normal saline. At 9:00 a.m. we infused 5 mg of morphine sulfate over 60 seconds through the catheters. We obtained small samples of blood through the catheters before and 30, 60, 90, 120, and 180 minutes after the morphine infusion for assay of serum prolactin in duplicate by radioimmunoassay (2). We calculated the maximum prolactin response for each patient by subtracting the baseline prolactin from the maximum prolactin level after morphine infusion. Values are expressed as mean ± SEM. Statistical significance of differences between means was determined by two-tailed t test. Patients filled out an adjective checklist self-rating form before the morphine infusion and at the time of each blood drawing.

Results

The control subjects and the depressed patients did not differ significantly in age or sex distribution. Morphine infusion produced marked, significant increases ($p<.05$) in serum prolactin 30, 60, 90, 120, and 180 minutes after infusion in the control subjects. There was no significant difference in the increase in prolactin levels in the patients with personality disorders compared with the normal volunteers. In the patients with major depressive disorder morphine infusion produced only small, nonsignificant increases in serum prolactin (see table 1). Mean baseline prolactin levels of 9.0±1.4 ng/ml in the control group did not differ from those of 10.5±1.9 ng/ml in the depressed group. Mean prolactin levels in the depressed group were significantly lower than those in the control group at 60 ($p<.01$), 90 ($p<.02$), 120 ($p<.02$), and 180 ($p<.05$) minutes (see table 1). The mean maximal prolactin response of 7.2±2.7 ng/ml in the depressed group was significantly lower than that of 31.9±9.5 ng/ml in the control group ($p<.01$). There was no significant correlation between maximal prolactin responses and baseline prolactin levels. The morphine infusion produced

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TABLE 1
Serum Prolactin Levels of Depressed Patients and Control Subjects After Infusion of 5 mg of Morphine

Minutes After Infusion	Serum Prolactin Levels (ng/ml) Control Subjects (N=6) Mean	SEM	Depressed Patients (N=10) Mean	SEM	Significance
0	9.0	1.4	10.5	1.9	n.s.
30	22.7	5.7	14.6	2.6	n.s.
60	33.0	4.8	15.0	2.3	p<.01
90	34.8	8.9	14.7	2.8	p<.02
120	26.8	4.6	11.6	3.0	p<.02
180	26.1	8.9	10.0	1.4	p<.05
Change[a]	31.9	9.5	7.2	2.7	p<.01

[a]Determined for each patient by subtracting the baseline prolactin level from the peak prolactin level after morphine infusion.

only small, nonsignificant subjective antidepressant and antianxiety effects in both the depressed and control groups.

Discussion

Both exogenous opioids and endogenous opioid peptides are potent stimulators of secretion of the pituitary hormone prolactin in animals (6) and man (2, 4, 7). Morphine in the dosage range we used has been reported to produce large and reliable increases in serum prolactin in normal subjects (4). Prolactin secretion is controlled in part by the dopaminergic tuberoinfundibular tract, which exerts an inhibitory effect over the secretion of prolactin (8). Serotonergic neurons have a stimulatory effect on prolactin secretion and other neurotransmitters and neuromodulators, including norepinephrine and epinephrine, have been reported to modulate prolactin secretion as well (8). Researchers have located opioid receptors on dopaminergic neurons (1). When these opioid receptors are activated, they inhibit the dopaminergic tonic inhibition of prolactin secretion. Such opioid receptor activation would therefore allow increased secretion of prolactin after administration of opioids (6).

Thus the absent or blunted increase in serum prolactin occurring after morphine infusion in our patients with major depressive disorder may reflect abnormalities in central endorphin, dopamine, serotonin, or other neuroregulatory systems. Possible abnormalities in endorphin systems that could account for a blunted prolactin response to morphine in major depression include an opioid receptor deficit, an excess of endogenous opioid antagonist, or elevated endorphin levels with compensatory down-regulation of opioid receptors. Although there was no significant difference in baseline prolactin levels between the control subjects and the patients with major depressive disorder, subtle changes in baseline prolactin secretion or diurnality might partially explain the blunted prolactin response to morphine exhibited by the patients with major depressive disorder. Halbreich and associates reported increased secretion of prolactin in depressed patients during the afternoon and evening (9). An increased prolactin secretion at some time of the day in the patients with major depressive disorder could conceivably have decreased the sensitivity of the prolactin system to stimulation. Possibly the use of psychotropic medication by some of the depressed patients in the 6 weeks before the study may have influenced their prolactin response to morphine. If this were the case, however, one would probably expect a significant difference between mean baseline prolactin levels of patients with major depressive disorder and those of control subjects, or correlation between maximal prolactin responses and baseline prolactin levels. We observed neither of these. Researchers should explore other factors that might influence prolactin response to morphine in depression, such as corticosteroid or thyroid axis abnormalities or pharmacokinetic changes.

While we observed significant miosis, we noted only a small, nonsignificant subjective antidepressant effect in the depressed patients. This lack of antidepressant response to morphine parallels the lack of neuroendocrine response in depressed patients. Perhaps higher doses of morphine or other opioids are needed to stimulate prolactin secretion in depressed patients than in control subjects. Preliminary work shows elevation of serum prolactin in depressed patients after infusion of 5 mg of methadone, which is about twice as potent as morphine (10). Investigators should continue to explore the role of endorphin systems, including the possible antidepressant effects of exogenous opioids and endorphins and their analogues in depressed patients.

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Localized Right Cerebral Hemisphere Dysfunction and Recurrent Mania

BY MARTIN R. COHEN, M.D., AND RICHARD W. NISKA, M.D.

Krauthammer and Klerman (1) recently reviewed the literature for reports of mania secondary to medical and/or pharmacological antecedents. They used operational diagnostic criteria for mania and excluded patients with a previous history of affective disease or accompanying symptoms of acute organic brain syndrome. The authors, who were able to find numerous cases meeting their criteria for secondary mania, concluded that mania should be considered a syndrome with multiple causes. Do these case reports also reveal information about the particular mechanism or neurophysiology involved? Most episodes occurred following a generalized disruption of cerebral functioning. In light of previous reports, we feel the following case report is suggestive of a specific mechanism. To our knowledge, this is the first report in the English literature of recurrent mania associated with localized cerebral psychopathology.

Case Report

Mr. A, a 59-year-old, right-handed man, had been in good health until the fall of 1974 when he suffered a subarachnoid hemorrhage. Evidence of a right temporal lobe hematoma was obtained from angiography, radionuclide scan, and CT scan. He initially manifested a left hemiparesis and left homonymous hemianopsia, but gradually improved so that by the time he was discharged 3 weeks later he had only minimal neurologic residua.

In February 1977 Mr. A was admitted for psychiatric care after an 8-week history of irritable mood and constant waking activity. His sleep time had become shortened and fragmented. His speech was described by relatives as loquacious, loud, grandiose, and tangential. He had begun driving his car at high speeds, buying expensive commodities, and investing his business capital irresponsibly. A mental status examination revealed that Mr. A was elated and irritable and had abortive episodes of tearfulness, with pressured speech and flight of ideas. No delusional or hallucinatory material could be elicited. His cognitive functions, including memory, orientation, serial sevens, and general fund of information, were found to be intact. He was diagnosed as suffering from manic-depressive illness, manic type, and lithium carbonate treatment was begun. Mr. A's condition gradually improved during the next 15 days, at which time he was discharged as euthymic. His lithium level was 0.93 mEq/liter.

During an outpatient follow-up visit that spring, Mr. A manifested some relapse of symptoms, which correlated with poor medication compliance. However, his symptoms abated 2 months later and the lithium was discontinued.

Mr. A was symptom free until July of the following year, when he was hospitalized after 3 to 4 weeks of symptoms similar to those that occurred in his first episode. Over a 3-week period he again responded to lithium and was discharged with a serum lithium level of 1.14 mEq/liter. While he was euthymic, neuropsychometric testing revealed that Mr. A had an average intelligence with intact verbal abilities, verbal memory, and abstract reasoning, but he had impaired visual perception, nonverbal sequencing, and nonverbal memory.

Approximately 1 year and 2 months later Mr. A was admitted to the hospital with his third episode of mania, which had apparently developed over the preceding 2 months. A repeat EEG was consistent with a residual focal right hemisphere lesion. He was again treated with lithium and gradually improved during the next month. Mr. A attained a peak lithium level of 1.62 mEq/liter. As an outpatient, he continued to do well while taking lithium.

Each of Mr. A's three episodes of psychopathology met both the Feighner and associates' (2) and Spitzer and associates' (3) criteria for mania. Despite extensive interviewing of Mr. A as well as many family members, we could find no history of affective disorder in him or his family.

Discussion

We believe Mr. A's mania is not coincidental to his cerebral dysfunction. The onset of mania at age 61 without a personal or family history of affective disorder is suggestive of a causal relationship (1, 4). Mr. A also met Krauthammer and Klerman's operational criteria for secondary mania.

In the English literature there are four reports of mania secondary to localized cerebral lesions (5–8). A striking similarity exists between our report and the case described by Rosenbaum and Barry (5). Their patient was undergoing neurosurgery when his right temporal lobe was traumatized and its anterior two centimeters resected. In the succeeding months, the patient developed a left homonymous hemianopsia and jacksonian seizures in his left arm. An EEG revealed a dysrhythmia involving the right posterior temporoparietal area. Five months later the patient developed mania, which was responsive to lithium. A later attempt to discontinue lithium resulted in recurrence of the mania.

Oppler (6) reported a case of mania that abated with the removal of a parasagittal meningioma involving the right frontoparietal area. Following the operation, there was transitory weakness of the left arm and leg. Stern and Dancey (7) reported a case of unremitting chronic mania in a patient who, at autopsy,

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was found to have a tumor occupying the right diencephalon.

Most recently Jamieson and Wells (8) reported a patient with four mass lesions in the right occipital, temporal, and parietal lobes. Although this patient was manic before surgery, it is notable that following partial right parietal occipital lobectomy the patient developed left-sided weakness and left unilateral neglect. The patient's performance IQ was substantially more affected than the verbal IQ. This patient's mania also responded to lithium. There was no recurrence of the mania after lithium withdrawal, but the patient died 2 to 3 months later.

Evidence reviewed by Flor-Henry (9) suggests that the organization of the cerebral mood system in dextrals may be lateralized to the right hemisphere. We can now add that our case, as well as the four previously reported cases of mania secondary to localized cerebral impairment, demonstrates predominant right hemisphere dysfunction. As Flor-Henry has speculated (9), perception of our own movement, so necessary for biological survival, may be an important psychophysiological component in the generation of our moods. These case reports support a close association between motility and visuospatial defects and secondary mania.

For diagnosis and treatment, justified emphasis has been placed recently on dividing mania into functional (primary) versus organic (secondary) categories. However, the etiological distinction may be somewhat arbitrary. Dalen's study (10) of hospitalized manic patients showed that those patients who apparently lacked a hereditary predisposition had had a greater frequency of exposure to perinatal events that involved a risk of brain damage and a greater frequency of EEG abnormalities. Our patient, as well as other secondary manics, showed a typical time course and response to lithium. He manifested the classic pattern of remission without medication and periodic recurrence.

These reports suggest that it may be possible to develop an animal model of mania secondary to surgically or chemically induced right temporoparietal dysfunction. Development of this model would require awareness of a latency period between cerebral insult and the onset of mania.

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Haloperidol Excretion in Human Milk

BY RONALD B. STEWART, M.S., BARRY KARAS, AND PHILIP K. SPRINGER, M.D.

Information concerning the excretion of specific psychotropic drugs in human milk is limited, although much concern for the potential hazards has been expressed in the literature (1–4). Psychotropic drugs are used frequently during pregnancy to treat nausea and vomiting and after delivery to treat anxiety, depression, and, occasionally, psychotic episodes. In this paper we present a case report documenting excretion of haloperidol in human breast milk and discuss the implications of this finding.

Case Report

Ms. A, a 33-year-old woman, entered our hospital with the complaint, "I killed my baby." This, in fact, was not true. Five days before her hospitalization she reportedly engaged in bizarre behavior that consisted of screaming and lying on the floor. She was diagnosed at this time as suffering from a major depressive disorder with psychosis. Ms. A had experienced a brief psychotic episode 1 year earlier and another one 1½ years earlier.

Five weeks before her hospitalization Ms. A had given birth to a girl, and she had breast-fed the infant until she was admitted to the hospital. Ms. A wished to continue nursing her child after she was discharged so she expressed milk from her breast three or four times daily during the hospitalization to maintain lactation. Because of her psychotic symptoms we prescribed 20 mg/day of haloperidol and increased the dosage as needed.

We were unable to find any definitive information concerning the excretion of haloperidol in human milk, so we decided to collect samples of Ms. A's breast milk and measure it for levels of haloperidol. Ms. A expressed the milk by hand; we collected samples in vacutainer blood tubes and then froze them. Concentrations of haloperidol were measured at the National Psychopharmacology Laboratory in Knoxville, Tenn., by using a gas chromatograph equipped with a nitrogen detector.

After 6 days of haloperidol therapy with a mean daily dose of 29.2 mg, 5 ng/ml of haloperidol were detected in Ms. A's breast milk. This level was obtained 11 hours after her haloperidol dose. On day 12, 9 hours following a 12-mg dose, her breast milk levels were 2 ng/ml. From day 13 to 19 we prescribed 7 mg/day of haloperidol (2 mg in the morning and 5 mg in the evening). We reduced the dose to 2 mg/day on day 20, and the next day we discontinued the drug. Concentrations of haloperidol levels in the breast milk were not detectable 3 days after the last 7-mg dose. These breast milk levels are similar to the therapeutic plasma levels that have been observed in pharmacokinetic studies in man (5).

Discussion

To our knowledge, this is the first report of haloperidol excretion in human breast milk. One study showed that levels of haloperidol in the milk of lactating cows and ewes were approximately 65% of those levels found in the animals' serum (6).

The pharmacokinetics of haloperidol have been extensively studied in man and animals (5, 7, 8). Haloperidol is eliminated primarily by metabolism in the liver and no active metabolites are produced. Bioavailability of haloperidol after oral administration is estimated to be 60%, and a serum half-life from 12.6 hours to 22.0 hours has been observed. Serum concentrations of haloperidol reach a steady-state level after approximately 6 days of treatment, and levels may vary up to tenfold in individuals given the same dose. Steady-state serum levels of haloperidol were shown to range from 3 ng/ml to 20 ng/ml in a study of 11 patients who were each given 6 mg/day of haloperidol (5).

Levels of haloperidol in the breast milk of our patient were 5 ng/ml after an average dose of 30 mg/day and 2 ng/ml following a 12-mg dose. This is a very small concentration of haloperidol in the milk. An infant normally ingests from ½ to 1½ liters of milk daily (9). Using this approximation, we calculated that an infant could receive a maximum of 0.0075 ng/day of haloperidol. The effect of these small concentrations of haloperidol on an infant is not known. Therefore, physicians should carefully assess the need for antipsychotic drug therapy in a mother nursing her infant.

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Oculogyric Dystonia in Tardive Dyskinesia

BY HENRY A. NASRALLAH, M.D., NICHOLAS J. PAPPAS, AND RAYMOND R. CROWE, M.D.

Dystonia is an acute extrapyramidal side effect of neuroleptic drug treatment. In one study (1) it was found that approximately 10% of the patients receiving neuroleptic drugs developed dystonia, which is usually associated with such high-potency neuroleptics as haloperidol and the piperazine phenothiazines. Dystonia usually occurs shortly after neuroleptic drug ingestion: 90% of patients experience dystonia within the first 4 days of drug treatment (1), or after an increase in drug dosage. It is characterized by tonic contraction of any skeletal muscle or muscle group, commonly involving the face, mouth, neck, and extraocular muscles in a painful and frightening oculogyric dystonic reaction. Dystonia usually occurs in young males and mainly in the initial phase of neuroleptic treatment.

We report here the appearance of an oculogyric dystonic reaction in a chronically medicated patient with severe tardive dyskinesia of several years' duration. This was our first observation of these symptoms in a patient with tardive dyskinesia, and to our knowledge such an occurrence has not been reported. Because the mechanism of dystonia and tardive dyskinesia is hypothetical and rather vague, the coexistence of these two symptoms and the circumstances leading to the coexistence may shed some light on the underlying neuropharmacological correlates of the symptoms.

Case Report

Mr. A, a 42-year-old single man, was admitted to the hospital for management of severe buccolingual tardive dyskinesia, which he had had for several years. It was gradually interfering with his speech, eating, drinking, and even breathing. He had been diagnosed as suffering from chronic paranoid schizophrenia at age 22, when he was first treated for psychosis. Mr. A was repeatedly hospitalized over the next 20 years, with the same diagnosis, and was maintained almost continuously on neuroleptics—chlorpromazine, thioridazine, trifluoperazine, fluphenazine, and haloperidol. Buccolingual dyskinesia was first noted at age 30. At age 40 Mr. A was hospitalized for the first time for management of his tardive dyskinesia, mainly severe mouth opening and tongue movements. He failed to show any improvement with a test dose of 2 mg of i.v. physostigmine, a cholinergic agonist, but he showed partial improvement with 1250 mg/day of sodium valproate, a γ-aminobutyric acid agonist, without a neuroleptic. However, Mr. A became psychotic again and manifested many first-rank symptoms; he was placed on 800 mg/day of thioridazine with good results.

During the current hospitalization, Mr. A received 800 mg/day of thioridazine and continued to suffer from severe tardive dyskinesia. He was given a trial of up to 40 mg/day of diazepam and improved somewhat for about 2 weeks. Diazepam was then gradually discontinued, and we decided to give him a trial of reserpine. However, Mr. A asked that another neuroleptic be substituted because thioridazine had made him impotent for the past several months. Thioridazine was gradually discontinued and 15 mg/day of trifluoperazine was started; we increased the dose to 50 mg/day within 5 days. Mr. A's involuntary movements did not change during this transition, and .1 mg of reserpine t.i.d. was started. Three days later Mr. A suddenly developed an oculogyric dystonic reaction at night; that day he had received trifluoperazine, 10 mg orally t.i.d. and 20 mg h.s., and reserpine, .25 mg orally t.i.d. and .75 mg h.s. Mr. A's eyes were fixed in an upward gaze and his neck was stiff, but he continued to have his buccolingual involuntary movements. Immediately 50 mg of i.v. diphenhydramine were given to Mr. A; he had prompt relief of his dystonic symptoms and, according to him, a transient subjective relief of his dyskinetic movements. During the next 36 hours Mr. A had two mild recurrences of upward gaze and still neck, which were promptly relieved with 25 mg–50 mg of i.v. diphenhydramine. He continued to take 50 mg/day of trifluoperazine for the next 3 weeks and up to 6 mg/day of reserpine, with no further dystonic reaction and with partial improvement of his tardive dyskinesia symptoms.

Discussion

The mechanism of neuroleptic-induced dystonias is still unknown, and there is no animal model of dys-

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tonia. However, due to the efficacy of anticholinergic drugs in treating dystonia, it has been suggested that dystonia may be due to the sudden decrease in dopaminergic activity and the relative increase in cholinergic activity following the dopamine-blocking effects of neuroleptics. Reversal of dystonia with dopamine agonists such as methylphenidate (2) and apomorphine (3) also supports this hypothesis. However, other drugs such as caffeine (4), diazepam (5), meperidine (6), and barbiturates (7) have been reported to relieve dystonia as well. Hypocalcemia and hypomagnesemia have also been shown to predispose patients to dystonia (8).

Another hypothesis by Marsden and associates (9) proposes that dystonia may be due to an increase, rather than a decrease, in dopamine activity due to the initial and transient compensatory increase in dopamine synthesis and turnover at the beginning of neuroleptic therapy. Support for this view also comes from Garver and associates (10). They found that dystonia appeared after the red blood cell concentration of a single dose of neuroleptic had peaked and was decreasing, possibly reflecting a decreased blockade of dopamine receptors.

The possibility that dystonia may be due to increased dopamine activity is of particular significance in this patient because his dystonia was superimposed on tardive dyskinesia. It is also believed that tardive dyskinesia is caused by dopamine hyperactivity and relative cholinergic hypoactivity (11) due to dopamine receptor supersensitivity secondary to a chronic dopamine receptor blockade by neuroleptics. Is it possible, then, that dystonia is the acute, transient, extrapyramidal manifestation of increased dopamine hyperactivity through a rebound increase in dopamine release? Is it possible that at the same time tardive dyskinesia is the chronic, persistent extrapyramidal manifestation of increased dopamine hyperactivity through the increased number of dopamine receptors, i.e., hypersensitivity? If so, then could acute dystonia early in the course of neuroleptic treatment predict future predisposition to tardive dyskinesia?

In our patient the addition of reserpine to trifluoperazine 2 days before the dystonic reaction is consistent with Marsden's hypothesis of dopamine hyperactivity preceding dystonia. Reserpine is a neuroleptic that releases dopamine until the neuronal stores are depleted. Thus during the first 2 days our patient took reserpine, there was probably a massive increase in synaptic dopamine concentrations in the presence of dopamine receptor hypersensitivity, which was only partially blocked by trifluoperazine. This could have resulted in a sudden, exceptionally high dopamine activity above that which exists, hypothetically, in tardive dyskinesia. This sudden surge in dopamine hyperactivity may have allowed the dystonic symptoms to emerge against the background of tardive dyskinesia.

Finally, reserpine given alone is known to produce various extrapyramidal side effects but not dystonia, even when reserpine is given in high dosages (9). On the other hand, neuroleptics that block dopamine receptors can produce dystonia even in very small dosages. This suggests that a dopamine receptor blockade may be an important step in the neurophysiological mechanism of dystonia. It would be quite important to note whether reserpine given with or without a neuroleptic that blocks dopamine receptors, such as trifluoperazine in our patient, may have a differential effect on dystonia and/or tardive dyskinesia.

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Family Experiences of Self-Mutilating Patients

BY JACQUELINE CARROLL, CHARLES SCHAFFER, M.D., JAMES SPENSLEY, M.D., AND STEPHEN I. ABRAMOWITZ, PH.D.

Parental deprivation is commonly noted in the childhood histories of self-mutilating patients (1). Feelings of isolation or separation anxiety often trigger self-mutilation (2–4), and some patients seek physical or sexual contact when tension mounts.

Recently, investigators have linked physical abuse during childhood to later episodes of self-mutilation (4–6). In two cases Green found a relationship between physical abuse and self-destructive behavior in children (6, 7). Roy (5) also found that more self-mutilating patients recalled excessive physical punishment in childhood than control subjects did.

The outward expression of anger may be severely restricted or punished in the families of self-mutilating patients, so that as children these individuals learned to turn their aggressive feelings inward (8). Other childhood histories of self-mutilators note eating problems (2, 3), accident-prone behavior (3, 7), family violence (1), and open displays of sex and aggression by the parents (4).

Most of the early data on the family backgrounds of self-mutilators are based on clinical impressions and case studies. Interview-based research has focused on present behavioral features, population characteristics, and management issues. Through a structured interview we explored aspects of early experience implicated but not yet systematically investigated in the self-mutilation literature.

Method

Our study included 14 self-mutilating patients and 14 nonmutilating, nonsuicidal control subjects from the same public psychiatric population. All self-mutilating patients reported at least one deliberate act of self-inflicted injury that was nonsuicidal in intent, and most had a history of several similar incidents. Their mean age was 28.1 years, with a range of 21–41 years. Six self-mutilators were inpatients (nonpsychotic at admission). Ten of the 14 self-mutilators were women, reflecting the sex ratio reported for this population (9). The control group was matched for age, inpatient/outpatient status, and sex.

Two of us (J.C. and C.S.) interviewed the subjects (one of us is a third-year clinical psychology graduate student, the other a third-year resident in psychiatry). We elicited information about aspects of behavior and history noted in the literature as relevant for self-mutilating, suicidal, or violent patients and nominally categorized responses.

Results

The results of Fisher exact probability tests indicated whether the criterion variables were more characteristic of the self-mutilating patients than of the control subjects. We found significant between-group differences for major separations before age 10, family violence, physical abuse, and anger prohibition by the family. Differences were not significant for birth order, sexual abuse, symptoms of triad for violence, accident proneness, eating problems, socioeconomic class, or family psychiatric history. In every instance, however, there was a trend consistent with a clinical formulation about self-mutilation.

Major separations from parental figures occurred more frequently in the childhood histories of self-mutilating patients. More self-mutilators than control subjects lost at least one parent before the age of 10 ($p<.05$).

Other coded data less amenable to reliable classification suggested a disparity in self-mutilators' parental relationships. Self-mutilating patients described a good relationship with one parent and a sharply negative relationship with the other more often than control subjects did.

Violent behavior or physical fighting occurred more often in the families of self-mutilators than in the families of control subjects. Two self-mutilating patients and 3 control subjects reported some family violence, while 10 self-mutilating patients and no control subjects reported excessive family violence. Self-mutilating patients were disproportionately represented among those who reported either some or excessive violence in the home during childhood ($p<.01$).

We also found a strong relationship between physical abuse by parents and self-mutilation. Four self-mutilating patients and 1 control subject recounted some or moderate abuse, while 8 self-mutilating patients and no control subjects described excessive physical abuse. Self-mutilating patients reported physical abuse strikingly more often than control subjects did ($p<.005$).

Sexual activity with an older family member tended to occur more often in the childhood histories of self-mutilators. Three female self-mutilating patients and 1

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female control subject experienced some sexual abuse, while 2 female self-mutilating patients and no control subjects described excessive sexual abuse.

The self-mutilating patients differed from the control subjects in regard to how anger was handled at home. Thirteen self-mutilating patients and 2 control subjects stated that expressions of anger were not allowed in their families, 1 self-mutilating patient and 9 control subjects stated that expressions of anger were sometimes allowed, and 3 control subjects stated there were no prohibitions against expressing anger in their families. More self-mutilating patients than control subjects reported strong restriction of anger in their families ($p<.005$).

Discussion

The higher frequency of major separations from parents experienced by self-mutilating patients as children supports the notion that separation anxiety and the threat of abandonment are significant themes. This is consistent with reports that a threatened or actual separation from an important relationship can precipitate self-mutilation (2–4).

The high incidence of family violence reported by the self-mutilating patients suggests disharmony in parental as well as child-parent relationships. The sheer extent of the family violence supports the theory that intrafamilial hostility precipitates the self-mutilating syndrome. Another precipitating factor was physical abuse during childhood, which empirical research has only recently related to mutilation (5, 6).

All but one of the self-mutilating patients were abused as children, either physically or sexually; prohibitions against expressing angry feelings may have led these individuals to seek other ways of coping with intense feelings. A conflictual formulation consistent with the data is that parental hostility fosters development of a punitive superego that, under stress, overwhelms ego defenses and triggers self-mutilation. Typically, self-mutilation is followed by a sense of calm, relief, and decreased tension and thus may promote a reintegration of ego defenses. The lack of maternal handling in early childhood reported by self-mutilating patients (3) also suggests a preconflictual component. Self-mutilators may come to associate physical abuse with contact that satisfies the need for physical touching and attention; the need for contact may override the unpleasant sense of pain or the self-mutilators may associate pain with need satisfaction and thus not experience it as pain.

We can also view self-mutilation in terms of a self theory, in which the impulsive self-destructive act becomes an attempt to overcome intense "depletion anxiety," i.e., fear of fragmentation or destruction of the self. Narcissistic patients who have been deprived of gratifications required for the development of a cohesive self may exhibit such behavior (10).

We recommend that researchers define the self-mutilating syndrome more precisely and conduct studies that incorporate nonmutilating suicidal control subjects and methods of subclassifying self-mutilating patients according to the type and the extent of self-injury.

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Tinnitus in Imipramine Therapy

BY JOHN RACY, M.D., AND E. ANN WARD-RACY, M.D.

Tinnitus (ringing in the ears) is a well-known side effect of certain medications (1, 2), but it is rarely reported in psychotropic drug use. The 1979 *Physician's Desk Reference* lists tinnitus as the last among neurologic side effects of tricyclics, yet we found no mention of it in any of the standard reference books on psychopharmacology (3–7). Our thorough search of the literature failed to reveal a single reference. In a report comparing two tricyclics, Melitracen and imipramine, Biros and associates (8) mentioned tinnitus as a side effect of Melitracen but not of imipramine. (Melitracen has not been marketed and is currently unavailable.) In 1960 Barker and associates (9) reported two cases of transient deafness in patients receiving imipramine.

Although the results of our literature search would lead us to recommend dropping tinnitus from the formidable list of tricyclic antidepressant side effects, our recent experience with four patients suggests otherwise.

Case Reports

Case 1. Ms. A, a 37-year-old woman, came to the clinic with manifestations of neurotic depression. She gave no indication of psychosis or any suggestion of a schizophrenic disorder. Ms. A had never received psychoactive drugs. Her therapist (E.A.W.-R.) prescribed imipramine, 50 mg/day. After 1 week of this regimen, Ms. A reported that ringing in her ears had started 2 to 3 days after she had begun taking imipramine. The tinnitus was so unpleasant that Ms. A did not want to continue treatment. When Ms. A's therapist persuaded her to continue taking the imipramine at the lower dose of 25 mg/day, her tinnitus stopped. Her clinical state improved, and she was maintained on the same dosage.

Case 2. Rose, a 15-year-old girl, exhibited predominantly depressive symptoms, with some elements suggestive of a schizophrenic disorder. Her therapist (E.A.W.-R.) prescribed imipramine on a graduated regimen of up to 150 mg/day to treat her depression. When Rose's imipramine dose reached 150 mg/day, she complained of ringing in her ears. Her medication was then reduced to 125 mg/day and the tinnitus disappeared. She responded favorably and has been maintained on that dosage.

Case 3. Ms. B, a 30-year-old woman, had had marital conflicts and suffered from severe anxiety and depression. Although she exhibited no evidence of psychosis, Ms. B was highly suspicious and concerned about people who might eavesdrop on her therapy sessions. At a later point her therapist (J.R.) prescribed imipramine. Ms. B responded partially to 100 mg/day and her therapist determined that she needed more medication. When he increased her dosage to 150 mg/day, Ms. B began to complain of nausea and a buzzing sound in her ears. When he reduced her medication to 100 mg/day, Ms. B's nausea and tinnitus subsided, but she again became depressed. Her therapist attempted to find a therapeutic level, increasing Ms. B's dosage by 10-mg increments. At 120 mg/day Ms. B's tinnitus and nausea returned. She achieved clinical improvement with 110 mg/day and suffered no further tinnitus.

Case 4. Mr. A, a 31-year-old man, exhibited low self-esteem, anxiety, anger, and progressive depression, with some borderline features. His therapist (J.R.) prescribed imipramine during the course of his psychotherapy. Its efficacy in relieving Mr. A's depression remained uncertain. While receiving 150 mg/day of imipramine Mr. A complained of distinct ringing in his ears. When the medication was reduced to 75 mg/day, the ringing was still present but somewhat milder. Eventually Mr. A's therapist discontinued imipramine and substituted desipramine at a dosage of 100–150 mg/day. Mr. A's tinnitus did not return and he found the medication helpful.

Discussion

Tinnitus may not be as rare a side effect of tricyclic therapy as would appear from the literature. The fact that our patients were able to continue with and profit from such therapy (either on a reduced dose of imipramine or with a substitute) suggests that this side effect is not a serious impediment or contraindication to tricyclic use. Tinnitus is very distressing to patients and must be controlled. The ringing in the ears described by our patients was typical and bore no resemblance to hallucinations. We found that reducing the dosage of imipramine was usually sufficient to control the tinnitus.

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Amitriptyline in Human Breast Milk and the Nursing Infant's Serum

BY TED F. BADER AND KARIN NEWMAN

To our knowledge, there have been no reports documenting the amount of amitriptyline found in the breast milk of women taking amitriptyline chronically or the amount of amitriptyline in the serum of their nursing infants. In this paper we describe such a patient and document the excretion of amitriptyline and its metabolite, nortriptyline, in human breast milk. A serum measurement was also taken of the nursing infant, and no amounts of amitriptyline or nortriptyline were found.

Case Report

Ms. A, a 36-year-old mother of one child, had been taking amitriptyline for 10 years to control her depression. Her usual dose, 150 mg/day was decreased to 100 mg/day when her second pregnancy was discovered. She continued this dose for the 7 months before the birth of her child, a girl weighing 2660 grams. Because Ms. A had suffered severe postpartum depression after the birth of her first child and she desired to breast-feed this child, it was decided that she should continue taking the lower dose of amitriptyline, 100 mg/day h.s.

In order to determine if the drug or its metabolite was accumulating in the mother's breast milk or the infant's serum, two sets of measurements were made to screen for detectable quantities of amitriptyline and nortriptyline in the breast milk (table 1). The first set of measurements was taken 6 weeks after Ms. A had begun breast-feeding her infant. When levels of amitriptyline and nortriptyline were discovered in her breast milk, a second set of measurements was repeated 11 days later; this time the infant's serum levels were also measured. Plasma samples were collected into heparinized vacuum blood collection tubes; care was taken not to allow the serum to come in contact with the stoppers. Breast milk samples were collected and stored in a plastic container. The serum and breast milk samples were stored under refrigeration at 4° C until measured. If samples were to be analyzed later than 3 days after being drawn, the serum was extracted and frozen at −20° C. All samples were analyzed within 10 days. Measurements of drug levels were performed by gas-liquid chromatography mass spectrometry as described by Biggs (1). This method of measurement is sensitive to plasma levels as low as 10 ng/ml. After analysis of the data, known amounts of amitriptyline were added to the breast milk samples to determine whether it was possible to completely recover the drug from the breast milk; the drug was recovered and our method of measurement verified. Ms. A gave informed consent for the study.

TABLE 1
Concentrations of Amitriptyline and Nortriptyline in Mother's Serum and Breast Milk[a] and Infant's Serum

Source	Hours Since Drug Ingestion	Concentrations (ng/ml) Amitriptyline	Nortriptyline
First test			
Mother's serum	15	141	86
Breast milk	16	151	59
Second test			
Mother's serum	14	83	59
Breast milk	14	135	52
Infant's serum		0	0

[a]The mother received 100 mg/day of amitriptyline h.s.

Discussion

In a comprehensive review Ayd (2) reported that there were no published reports on the excretion of nortriptyline and only one published report on the secretion of amitriptyline in human breast milk. In a study by Eschenhof and Rieder (3) no detectable amounts of amitriptyline were found in the breast milk of six women after they received a 25-mg or 50-mg dose of amitriptyline; however, the method of measurement used in the study was sensitive only to 100 ng/ml.

The amounts of amitriptyline and nortriptyline were approximately equal in our patient's breast milk and serum, which indicates that these drugs are probably passed from serum to breast milk in a passive manner.

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Equal levels of imipramine and desipramine in serum and breast milk have also been reported (4), even though the patient had taken imipramine and desipramine for only 16 days.

Our finding of undetectable amounts of tricyclic in the nursing infant is reasonable. If we assume the infant received a liberal intake of breast milk, say 1000 cc/day, and if we look at the highest amount of amitriptyline concentrated in Ms. A's breast milk, 151 ng/ml, the baby would ingest only 150 μg/day or 50 μg/kg per day. This figure is still much smaller than the mother's dose of 1.5 mg/kg of amitriptyline.

On the basis of this clinical study it was recommended to the mother that she continue breast-feeding her child if she so desired. A more general recommendation cannot be made from one case report. Nonetheless, in the absence of other data, this report represents preliminary reassurance to nursing mothers because amitriptyline did not accumulate in one nursing infant.

In order to make a rational decision about the use of drugs during breast-feeding, it is not sufficient to find detectable but nonpharmacological amounts of a drug in breast milk. The sine qua non of such a decision must rest upon taking measurements in many nursing infants whose mothers are receiving the drug. This case report is an initial step in gathering such knowledge about amitriptyline.

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Psychopharmacological Precautions in the Right to Refuse Medication

BY ROY B. LACOURSIERE, M.D.

The right of involuntarily treated mental patients to refuse medication is being progressively established and delineated (1–4). Although this right may enable a patient to refuse any medication, I will discuss primarily in this paper the psychotropic medications neuroleptics, antidepressants, and lithium. In general this right states that psychotropic medication cannot be forced upon a nonconsenting person being involuntarily treated for psychiatric problems except in an emergency, adjudication of incompetency, or sanction by a properly constituted reviewing body. A right to refuse medication applies with even more force to voluntarily treated persons.

If one accepts a right to refuse psychotropic medication, the responsibility that this right entails also has to be accepted. The following discussion identifies several psychopharmacological precautions, and therefore responsibilities, that need to be considered in the exercise of this right to refuse medication.

Many psychopharmacological issues have been addressed in the legal cases and other publications on the right to refuse psychotropic medication, including discussions on the efficacy of such medication and its adverse effects, such as tardive dyskinesia. However, other important psychopharmacological issues have not received much attention and need to be considered by the courts, mental health bar, and treating personnel. Mental health personnel are more aware of the precautions that need to be taken when using psychotropic drugs, but a reminder may encourage such professionals to keep these precautions before our legal colleagues.

Three issues relating to psychotropic drugs are discussed below: the therapeutic time lag that occurs from the time drug therapy is initiated until therapeutic effects develop; the deterioration time lag that occurs from the time drugs are discontinued until symptom recurrence or exacerbation may be noted in previously drug-responsive patients; and the medical effects (excluding tardive dyskinesia) that occur after abrupt medication withdrawal.

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Therapeutic Time Lag

Some patients will improve adequately without psychotropic medication, but for many others neuroleptics, antidepressants, or lithium will be part of the optimum therapeutic recommendation; sometimes more than one of these agents will need to be prescribed. There is a considerable time lag before the optimum therapeutic effects of these medications are achieved. For instance, although neuroleptics can have immediate sedating effects, their optimum antischizophrenic effects on hallucinations, delusions, and disordered thinking may take several weeks. Significant effects from antidepressant medication may take 2–3 weeks to develop, and lithium may not be optimally effective for 1 week or longer. Therefore, persons who refuse such medication, then begin to take it at a later date should not be expected to respond rapidly, except in the limited ways discussed above.

This therapeutic time lag could also mean that a severely suicidally depressed person who was initially a candidate for antidepressant medication but exercised his or her right to refuse the medication might later become a more appropriate candidate for ECT, especially by the time consent for treatment was obtained from the court or other reviewing body. Under these circumstances ECT would be a more rapid and safe treatment in the patient rather than having the patient wait an additional 2 weeks or more for significant response to antidepressant medication. Generally, the exercise of the right to refuse medication will, for many patients, prolong the period of dangerousness to self and/or others and the length of hospitalization.

Deterioration Time Lag

When patients who have been responsive to psychotropic medication then exercise their right to refuse further medication, they will often have an analogous time lag before psychiatric deterioration occurs—if it does occur. The deterioration time curves are less predictable than the therapeutic time curves. A few schizophrenic patients deteriorate seriously and dangerously within a few days after neuroleptic withdrawal, but others function adequately for several months or longer, or even indefinitely (5, 6). Similar time lags occur when patients who are responsive to the medication are withdrawn from antidepressants and lithium, although the percentage of these patients who deteriorate may not be as high as the percentage of schizophrenic patients who stop taking neuroleptics and deteriorate.

These time lags obfuscate our ability to assess the therapeutic and long-term legal effects of decisions made by individuals, courts, or reviewing bodies to exercise the right to refuse psychotropic medication. Patients who show no immediate dangerousness to themselves and/or others or other deleterious effects after their medication is withdrawn may show such effects during the following days or weeks. By then the patient may have been discharged.

This does not mean, of course, that patients cannot be withdrawn from psychotropic medication, but it does mean that precaution should be used by the courts and others in doing so and in assessing the effects of such medication withdrawal in this context of the right to refuse medication. The 1978 case of *Rennie v. Klein* (4) is highly illustrative in this regard. Concomitant with Mr. Rennie's changing acquiescence and refusal to use medication and the court's related interventions, his behavior fluctuated widely and he was at times seriously disturbed and dangerous.

Medical Effects of Abrupt Withdrawal from Psychotropics

A variety of medical symptoms can occur in the few days following the abrupt withdrawal of neuroleptics from patients who have taken them for several weeks or longer; highly sensitive patients may have withdrawal symptoms after only 1 or 2 weeks of medication. I will not discuss tardive dyskinesia because it has already received considerable attention in the literature on the right to refuse medication. Symptoms that are of concern here include nausea and vomiting, stomach pains, restlessness, insomnia, and headaches. These withdrawal symptoms can occur after neuroleptics are discontinued whether or not antiparkinsonian agents are taken concomitantly and are simultaneously and abruptly withdrawn. In a number of studies withdrawal symptoms occurred in 18%–75% of patients after neuroleptics were stopped, with symptoms occurring on the average in about 50% of the patients (7). Although the symptoms are often mild to moderate in severity and usually resolve without sequelae, they can be very severe (7, 8).

Medical withdrawal effects are more clearly established for neuroleptics and for antiparkinsonian agents that are often taken concomitantly with neuroleptics, but they also occur with tricyclic antidepressants (9, 10). Such effects have not been of concern after the abrupt withdrawal of lithium.

The courts, the mental health bar, and treating personnel must use precaution when dealing with previously medicated patients who are now exercising their right to refuse medication and are being removed from tricyclic antidepressants, neuroleptics, and antiparkinsonian medications, which are often taken concomitantly with neuroleptics. It is recommended that such medication be withdrawn gradually over 1 or 2 weeks and that withdrawal symptoms be treated when indicated, e.g., antacids for stomach irritation.

Discussion

In this paper my primary purpose has not been to enter into the legal or psychiatric arguments for or against the right of voluntarily and involuntarily treated persons to refuse psychotropic medication un-

der certain circumstances. Rather, my discussion focused on precautions that should be taken in this context because psychotropic medications have a therapeutic time lag, deterioration time lag, and can cause medical effects on abrupt withdrawal. If we include these precautions in our considerations on this right to refuse medication, a little less human misery may be produced.

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A Case Report of Anorexia Nervosa and Gaucher's Disease

BY MILTON K. ERMAN, M.D., AND GEORGE B. MURRAY, M.D.

Anorexia nervosa is a disorder of body image and eating habits seen most frequently in adolescent females. Research diagnostic criteria for the syndrome include refusal to maintain a minimal normal body weight, weight loss of at least 25% of original body weight, distorted body image, intense fear of becoming obese, absence of known medical or psychiatric illness that could account for the weight loss, and amenorrhea in females (1).

Gaucher's disease is an inherited metabolic disorder that occurs most often in Jewish families and is characterized by a deficiency of the lysosomal enzyme glucocerebrosidase (2). Three subtypes have been described. The most common is Type 1 or adult non-neuronopathic Gaucher's disease. It produces hepatosplenomegaly, pancytopenia, and erosion of the cortices of the long bones, has a variable course, and may remain undiagnosed until late in life. Type 2, the infantile neuronopathic form, leads to multiple developmental abnormalities, usually apparent by the sixth or seventh month of life, with a mean survival of nine months (2). Juvenile Gaucher's disease, or Type 3, is characterized by multiple central nervous system abnormalities, including mental deterioration, behavior disorders, psychoses, and abnormal EEGs. Although the disease generally manifests itself in childhood, some adults with this subtype have been reported (2).

We report a patient whose Gaucher's disease initially manifested itself with psychiatric symptoms. To our knowledge, this is the first adult case reported in which investigation of an apparent psychiatric illness led to the diagnosis of Gaucher's disease.

Case Report

Mr. A, a 28-year-old single white Jewish man, was referred to the Massachusetts General Hospital for an evaluation of unexplained weight loss and weakness in his left arm and leg. He reported a weight loss of 22.5 kg during the 6 months before his admission.

The admission physical examination revealed that he weighed 41.4 kg, showed marked cachexia, had lanugo, and had a resting pulse of 55 beats/minute. Hematologic abnormalities included a hematocrit of 24.8% and a platelet count of 50,000. Shortly after Mr. A was admitted, it was noted that he had peculiar attitudes toward food, and psychiatric consultation was requested.

Mr. A requested dietetic foods, professed a dislike for carbohydrates, and was concerned that people were trying to trick him into eating foods containing sugar. He was excessively involved with food preparation and spent 2 hours rearranging a lunch tray while eating virtually nothing. His food was found in wastebaskets; he expressed astonishment that

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this could have happened. Mr. A denied enjoying losing weight, but he was able to date the onset of his weight loss to a period when he became concerned with his appearance and began dieting and exercising, as well as decreasing his alcohol intake. He developed a distorted body image without awareness that his weight loss had become a serious problem. He reported periods of overactivity but denied bulimia or induced vomiting.

Mr. A's food avoidance behavior, distorted body image, and increased activity were suggestive of anorexia nervosa. However, his age, sex, lack of history of an eating disturbance, and successful and independent lifestyle during the preceding 10 years were atypical.

Although the hematologic abnormalities were presumed to be secondary to anorexia, a bone marrow biopsy was performed and revealed lipid-laden histiocytes. Elevation of acid phosphatase, hepatomegaly, and radiographic evidence of osteopenia and cortical thinning of the femur were also noted. Further histochemical studies showed that the foam cells in the marrow contained iron and were PAS (periodic acid-Schiff) positive, which is characteristic of true Gaucher's cells. The diagnosis of Gaucher's disease was firmly established when serum glucocerebrosidase levels were found to be approximately 10% of normal values (3).

Neurological and psychological abnormalities were observed. Neurologic examination revealed definite weakness in the left upper extremity, with questionable abnormalities in the lower extremities. Electromyography (EMG) showed a left radial nerve palsy and mild generalized motor neuropathy in the left upper extremity. The computerized tomographic (CT) scan revealed significant enlargement of the lateral and third ventricles and excessive prominence of the Sylvian fissures and convexity sulci. On the Wechsler Adult Intelligence Scale (WAIS), he obtained a verbal score of 119 and a performance score of 106 with marked subtest scatter.

Mr. A initially resisted a strict, behaviorally oriented high caloric regimen, but with increased cooperation he gained 10.5 kg in 21 days and was discharged with a weight of 50.7 kg. No psychotropic medications were used in Mr. A's treatment; while an inpatient, he was seen daily in supportive psychotherapy.

Discussion

Psychiatric consultation was requested because of the patient's suspected psychogenic weight loss. Multiple symptoms of anorexia nervosa were present, but Mr. A's complaints of weakness, objective evidence of left upper extremity weakness, abnormal findings on EMG, CT scan, and psychological testing, as well as dysarthria pointed to a central nervous system disturbance.

Neil and associates described a family in which the diagnosis of adult-onset Gaucher's disease was followed years later by psychosis and neurologic abnormalities (4). Of the three cases reported, two had generalized weakness and two had large WAIS subtest scatter and verbal-performance differences. One patient had an abnormal CT scan, but all three had abnormal EEGs. Other reports have described adults whose neurological symptoms developed long after the discovery of visceral and bone involvement (5) and weight loss and muscle wasting in the juvenile form of the disease (6).

The argument could be made that Mr. A had two illnesses, anorexia nervosa and coexistent Gaucher's disease. While there is no way to completely rule out this possibility, it is unlikely for at least two reasons. First, the traditional rule of parsimony says it would be most unusual for two such rare diseases to manifest themselves in one individual, especially since a link between adult-onset Gaucher's disease and subsequent psychiatric illness has already been established. Second, the atypical nature of his illness and his rapid improvement with treatment militates against the diagnosis of primary anorexia nervosa.

This case illustrates that adult-onset Gaucher's disease may manifest itself as psychiatric illness. Although it is a rare syndrome, Gaucher's disease should be considered when a behavioral disturbance or psychosis is seen in conjunction with weakness, neurologic abnormalities, or abnormal EEGs or CT scans. While it is unlikely that Gaucher's disease will often manifest itself as anorexia nervosa, it should be recognized as a medical disorder that may appear with psychiatric symptoms.

This case also confirms the need for a rigorous medical workup of patients with atypical presentations of classic psychiatric illnesses. Such situations may actually represent typical presentations of unusual illnesses; awareness of this fact may be a necessary prerequisite in making a difficult or obscure diagnosis.

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Acute Hyperkalemia During Lithium Treatment of Manic Illness

BY FREDERICK C. GOGGANS, M.D.

Disturbances of the serum potassium level have not been associated with the use of lithium carbonate in man (1). However, one study (2) showed that there were significant increases in the serum potassium levels of manic-depressive patients during the first 3 months of lithium therapy, even though the increases were within normal physiological limits (3.5–5.0 mEq/liter). The following case report describes a patient who suddenly developed hyperkalemia during the treatment of acute manic illness with lithium.

Case Report

Ms. A, a 67-year-old woman, was admitted to the hospital for the second episode of a psychotic illness. Her symptoms were sleeplessness, hyperactivity, loquaciousness, expansive mood, and paranoid delusions. I diagnosed her as suffering from manic-depressive illness and began a trial of lithium.

Ms. A had no history of medical problems and took no medications before this admission. A physical examination made at the time of her admission revealed no abnormalities. Before lithium was administered, a laboratory evaluation revealed the following: a normal complete blood count; sodium, 142 mEq/liter; potassium, 4.6 mEq/liter; chloride, 102 mEq/liter; bicarbonate, 31 mEq/liter; BUN, 16 mg/100 ml; creatinine, 1.2 mg/100 ml. Her ECG showed a sinus rhythm with a rate of 65 beats/minute; the PR interval was .16; QRS, .08; QT, .36. There was no evidence of any conduction abnormality or previous myocardial infarction. While Ms. A was taking 1200 mg/day of lithium, her serum lithium level was 1.2–1.5 mEq/liter; her manic symptoms decreased markedly on this dose, but her paranoid delusions persisted. During her third week of treatment 10 mg/day of haloperidol were added to her regimen, and her paranoid delusions disappeared.

Haloperidol was discontinued during the fourth week, and she was maintained on 600 mg/day of lithium; her lithium serum levels were 1.0–1.1 mEq/liter. At the end of the fourth week, a routine SMA-6 was as follows: sodium, 138 mEq/liter; potassium, 5.7 mEq/liter; chloride, 109 mEq/liter; bicarbonate, 31 mEq/liter; BUN, 15 mg/100 ml; and creatinine, 1.1 mg/100 ml. Repeat measurements the following morning showed these values: sodium, 136 mEq/liter; potassium, 6.2 mEq/liter; chloride, 103 mEq/liter; bicarbonate, 30 mEq/liter; BUN, 14 mg/100 ml; and creatinine, 1.2 mg/100 ml. This sudden appearance of hyperkalemia was confirmed when a third potassium level, obtained immediately after the morning's results were returned, was 6.2 mEq/liter. An ECG done just before that blood specimen was taken showed sinus bradycardia (55 beats/minute). The PR interval was .16; QRS, .10; and QT, .36. An incomplete right bundle branch block pattern appeared, and the T waves were slightly peaked. Although Ms. A was otherwise asymptomatic for hyperkalemia, she was treated with intravenous glucose, insulin, and oral kayexalate. Lithium was continued. Her serum potassium level fell to 4.4 mEq/liter after 4 hours. Ms. A's serum potassium levels 1, 3, and 7 days later were 4.8 mEq/liter, 4.7 mEq/liter, and 4.8 mEq/liter respectively; the last reading was taken immediately before she was discharged. Her ECG returned to its admission appearance when the potassium level returned to the normal range, and a final tracing on the day of her discharge was also identical to her admission ECG.

Ms. A was discharged and instructed to continue taking 600 mg/day of lithium; her serum lithium level was 1.0 mEq/liter. Three weeks after her discharge, Ms. A's serum potassium level had risen to 5.6 mEq/liter, and her lithium level was 1.0 mEq/liter. Lithium was discontinued and her serum potassium level fell to 4.5 mEq/liter within 1 week. Ms. A's serum potassium level remained in the normal physiological range. Her psychiatric status remained stable for 4 months following her discharge. Then she developed a hypomanic episode, which was treated successfully with haloperidol on an outpatient basis. No cardiac arrhythmias or potassium abnormalities were associated with that episode or its treatment.

Discussion

Ms. A had acute hyperkalemia that was asymptomatic except for the ECG abnormalities described above. These abnormalities were associated with her hyperkalemic state and disappeared following corrective treatment, which suggests that the ECG abnormalities were secondary effects of hyperkalemia. Bradycardia, cardiac conduction abnormalities, and peaked T waves are all commonly associated with hyperkalemia (3).

The differential diagnosis of acute hyperkalemia involves distinguishing pseudohyperkalemia, which is secondary to prolonged tourniquet time or hemolysis before potassium measurement, from true hyperkalemia. This distinction may be made by repeated potassium level determinations or by demonstrations of ECG changes consistent with hyperkalemia. Among the causes of true hyperkalemia are metabolic acidosis, renal insufficiency, Addison's disease, hyporeninemic hypoaldosteronism, periodic hyperkalemic paralysis, drug effect (usually secondary to potassium-sparing diuretics, such as triamterene or spironolac-

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tone), excess potassium ingestion, and endogenous release associated with cell injury. However, none of these causes was identified in Ms. A, despite a thorough review of her history with the internal medicine consultation service.

I then considered the possibility that lithium may have caused Ms. A's acute hyperkalemia. With the assistance of the Lithium Information Center at the University of Wisconsin, I made a search of the literature on lithium and hyperkalemia but found no reported case of acute hyperkalemia associated with the clinical use of lithium. Baldessarini and Lipinski reported that ECG changes resembling those of hyperkalemia have been reported in animals. The lithium levels required to induce these changes, however, are extraordinarily high, above 10 mEq/liter, and unlikely to be encountered clinically (4). McKusick also demonstrated in experimental animals that raising the serum lithium level above 10 mEq/liter by intravenous infusion produced a consistent series of ECG changes indistinguishable from those of hyperkalemia (5). He noted that under those conditions lithium caused the elevation of serum potassium levels, probably through direct displacement of the ion from the cell. He concluded that the ECG changes observed were most likely secondary to changes in the ratio of intracellular to extracellular potassium. At levels of lithium used clinically in man, the effect of lithium on ECGs usually resembles those effects associated with hypokalemia. The usual explanation of these abnormal ECG readings has been related to the theory that lithium replaces potassium within the myocardial cell (6). Nevertheless, this replacement has not been associated with changes in the serum level of potassium or with ECG changes resembling hyperkalemia.

Significant increases in serum potassium levels within the normal range have been reported during uncomplicated treatment of manic-depressive illness with lithium. It is not unreasonable to suspect, therefore, that Ms. A's acute hyperkalemia may have been related to lithium therapy. This conclusion is made more tenable by the fact that Ms. A's hyperkalemia returned while she was taking lithium, but it disappeared after lithium was stopped. A possible mechanism for this effect would be that lithium, as it enters the cell, may disturb the electrochemical equilibrium across the cell membrane and increase potassium conductance. A subsequent loss of potassium into the extracellular fluid may lead to hyperkalemia in some patients.

The association of hyperkalemia with the use of lithium at therapeutic levels is particularly important in light of the recent study of Shopsin and associates (7). They reported that there was a 3.8% incidence of sudden death in 105 patients established on maintenance lithium. Their explanation of this unexpectedly high rate of sudden death focused on the hereditary predisposition of the patients who died, but an alternative explanation of these sudden cardiac deaths could be the acute development of hyperkalemia. Further studies seem indicated to detect the prevalence and the effects of hyperkalemia in patients treated with lithium.

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Suppression of Plasma Cortisol in Depressed Patients by Acute Intravenous Methadone Infusion

BY PHILIP W. GOLD, M.D., IRL EXTEIN, M.D., DAVID PICKAR, M.D., ROBERT REBAR, M.D., RICHARD ROSS, M.D., PH.D., AND FREDERICK K. GOODWIN, M.D.

A cardinal biochemical finding in major depressive illness is activation of the hypothalamic-pituitary-adrenal (HPA) axis. This activation is manifested both by an overall increase in the daily 24-hour production rate of corticosteroids as well as disruption of the circadian pattern of corticosteroid secretion (1). Most patients with HPA activation also fail to adequately suppress corticosteroid secretion following dexamethasone administration (2).

The etiology of HPA activation in depression is unclear. It has been suggested that this activation is related to altered central biogenic amine neurotransmitter function (3–5). These neurotransmitters have been shown in experimental animals to modulate the secretion of corticotropin-releasing factor (CRF), a hypothalamic peptide that stimulates pituitary adrenocorticotropin (ACTH) release, which in turn stimulates adrenal steroid production (6). It has been demonstrated recently that a variety of neuropeptides may also influence CRF and/or ACTH release and hence corticosteroid secretion. Among these peptides are the endogenous opiates (7), which, like the biogenic amines, have been postulated to play an important role in regulating mood.

To explore the possible interface between endorphinergic and HPA function, we present here a report that, to our knowledge, represents the first study examining the effects of exogenous opiate administration on HPA function in patients with a major depressive disorder.

Method

Four patients who were mildly to moderately depressed and who met the standard Research Diagnostic Criteria (8) for admission to the NIMH clinical research units at the NIH Clinical Center were chosen to receive intravenous methadone. Each patient was drug-free for at least 3 weeks before our study and was given nothing by mouth after midnight the day before infusion of methadone. At 8:00 a.m. a polyethylene catheter was placed in each patient's antecubital vein and 5 baseline samples were drawn every 15 minutes from 9:00 a.m. to 10:00 a.m. At 10:00 a.m. each patient received 5 mg of methadone i.v. in 1 cc of sterile saline given over 1 minute. Additional blood was drawn until 1:00 p.m. at 15, 30, 45, 60, 90, 120, and 180 minutes after the infusion. In accordance with the usual diurnal pattern of cortisol secretion, we sought to control for the expected fall in cortisol levels between 10:00 a.m. and 1:00 p.m. by repeating the procedure on a separate day, except this time placebo (1 cc of sterile saline) was infused instead of methadone. Because methadone has an extended half-life, 5 baseline samples were also drawn in one patient between 9:00 a.m. and 10:00 a.m. the day following methadone infusion to evaluate the possibility of a sustained effect of methadone on cortisol excretion. All plasma samples were assayed for cortisol by radioimmunoassay. Baseline values for each subject were calculated utilizing the average of the 5 samples drawn between 9:00 a.m. and 10:00 a.m. before the infusions.

Results

There was no significant difference in the mean (±SE) of the average baseline levels between the methadone infusion day (102.9±14.3 ng/ml) and the placebo day (92.5±25.1 ng/ml). Methadone induced a progressive decline in cortisol secretion that was maximal at the time the last data point was obtained, 3 hours after the infusion. By this time, cortisol had fallen from the mean average baseline of 102.9±14.3 ng/ml to a mean of 26.0±5.8 ng/ml; this decline represents a mean decline of 76.9±12.1 ng/ml or a mean percentage fall of 73.4±2.8. On the placebo day a progressive decline in cortisol secretion was also maximal 3 hours after infusion. Thus, cortisol fell from a mean average baseline of 92.5±29.1 ng/ml to a mean of 63.8±14.8 ng/ml 3 hours after saline infusion. This decline represented a mean fall of only 28.7±8.7 ng/ml or a mean percentage fall of only 29.7±7.9. The mean percentage fall of cortisol ($p<.01$) and the mean fall of cortisol ($p<.02$) were significantly greater following methadone infusion than they were on the placebo day.

We noted a sustained suppression of cortisol secretion 24 hours after methadone infusion in the patient who was examined for this the following day. The mean baseline cortisol level of this patient was 124.67±28.76 ng/ml, which fell substantially to 39.0

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ng/ml 3 hours after infusion and remained suppressed at essentially this level (44.67±2.6 ng/ml) between 8:00 a.m. and 10:00 a.m. the next day.

Discussion

Endogenous opiates interact with HPA function in several important ways. For example, a common precursor prohormone peptide contains the sequence of both ACTH and β-endorphin and is thought to be the source of both peptides (7). Furthermore, in experimental animals, ACTH and β-endorphin are secreted in equimolar amounts concomitantly by a single anterior pituitary cell type and are each regulated identically by specific excitatory, including CRF, and inhibitory stimuli (7). In human subjects, heroin addicts have been reported to show diminished basal ACTH and cortisol levels, which has been interpreted to suggest that opiates, like ACTH, exert negative feedback effects on CRF and/or ACTH secretion (9).

In this study, methadone infusion induced a marked acute suppression of cortisol secretion. This suppression presumably reflects negative feedback effects exerted on CRF and/or ACTH secretion via opiate receptors located at hypothalamic and pituitary levels (10). Other mechanisms for methadone-induced suppression of cortisol secretion are also possible, including direct suppression at the adrenal level, but this possibility seems unlikely, especially in view of the ACTH suppression noted in narcotic addicts.

It has been clearly established that the HPA responds to feedback inhibition by corticosteroids at both hypothalamic and pituitary levels. Compared to normal subjects, depressed patients often show a relatively transient suppression of corticosteroid levels following administration of the synthetic glucocorticoid dexamethasone (2). This response of early "escape" to the standard dexamethasone suppression test implies an elevation in the set-point for feedback inhibition of CRF and/or ACTH in the presence of high circulating corticosteroid activity. If methadone suppresses cortisol levels via feedback inhibition of CRF and/or ACTH, it may be possible to devise a methadone suppression test, analogous to the dexamethasone suppression test, to evaluate the set-point for feedback inhibition exerted by opiates on the HPA.

It should be noted that methadone, like dexamethasone, has an extended half-life, so the extent as well as the duration of HPA suppression following methadone could be meaningfully evaluated. Although the one patient reported here showed no "escape" of cortisol secretion 24 hours after methadone administration, the possibility of early "escape" of cortisol suppression following methadone infusion in more severely depressed patients remains to be evaluated. Thus, it is proposed that the development of a methadone suppression test could be utilized to explore the possible relationship between endogenous opiate function and HPA activation in depressed patients.

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LETTERS TO THE EDITOR

This section contains reactions to Journal *articles, statements of opinion, brief research findings or ideas, short case descriptions, requests for information, etc. Letters should be submitted in duplicate to the Editor, who makes all decisions regarding publication. Letters must be typed double spaced throughout and should not contain more than 500 words and 5 pertinent references. Criticisms of published articles will automatically be sent to the author(s) for response. Letters will be edited for clarity and conformance with* Journal *style. We regret that we cannot inform writers of the disposition of letters or return those which are not printed.*

From the South African Department of Health

SIR: I am disappointed by the lack of scientific objectivity shown in the Report of the Committee to Visit South Africa (November 1979 issue). South African psychiatrists have respected APA because they believed that it published only objective reports.

The committee was not invited to do, nor did, an in-depth study of the policy of separate development (apartheid) but claimed, "We found that apartheid has a destructive impact on the families, social institutions, and the mental health of black South Africans. We believe that these findings substantiate allegations of social and political abuse of psychiatry in South Africa."

The committee went out of its way to meet opponents of this country's political system and ignored or omitted any positive facts provided by the South African Medical and Dental Council, as well as the South African Nursing Council, who control all medical and nursing practitioners, irrespective of race. There are black elected members on these councils. The 1973 Mental Health Act and the 1977 Health Act apply to all sections of the population without any reference to race. The shortcomings that exist are not merely due to discriminatory legislation or South Africa's policies.

The committee claimed that black patients aged 40–60 years were allowed to die even when a diagnosis of treatable illness like bacterial pneumonia was made. This implies that treatment is withheld from black patients as a result of government policy. The committee did not state that each hospital it inspected had a dispensary and that a full range of medicines was available. Committee members selected files, studied them, and returned them without asking the staff to explain shortcomings in the documentation of a specific deceased patient.

Medical literature does substantiate the observation that psychiatric patients often have lower resistance to diseases than do individuals with better mental health. Since 1934 (Malzberg in New York State) researchers have commented on the high mortality rate among psychotic hospitalized patients. In a preliminary report Hofer and Raymond (1) noticed that the mortality in their psychiatric population, as far as cardiovascular and respiratory diseases were concerned, was of the same ratio as in the general population. Mental illness, however, has an effect on longevity. The mortality rate is modified and death comes at a younger age. Lehmann (2) described how poor communication between patient and staff could cause a patient with schizophrenia to suffer from a serious illness without complaining. A diagnosis may be made in the late stage of the illness, but the patient may still die despite treatment. A select APA committee should have kept these problems in mind.

I dispute the implication that the differences in our service standards result from malicious disregard for other human beings. The Department of Health provides physical facilities to each population group that are usually an improvement on the standard of living of the social environment from which most patients come. The services in these facilities are related to the cultural practices of the group accommodated. World Health Organization guidelines support this practice. The Alma-Alta Declaration on primary health care issued by the WHO in September 1978 supports "essential health care based on practical, scientifically sound and socially acceptable methods and technology made universally accessible to individuals and families in the community through their full participation and at a cost that the community and the country can afford." The committee has confirmed that no unacceptable methods and technology were used in caring for these patients. Committee members visited facilities in the most isolated areas of South Africa. The committee could not find patients who were detained without sound reason, although it did doubt the need to detain certain epileptic patients. However, it is the social aspect of the care that is brought into question. The difference in diet, clothing, and sleeping arrangements and other facilities can be explained by social and cultural differences. Unfortunately, our cultural groups have different skin colors and different levels of social development and therefore critics of our mental health services always refer to politics.

The committee failed to mention the Department of Health's recognition of social development and its policy that all future psychiatric hospitals will be of the same structural standard. The first facility for colored patients implementing this policy will be erected during 1980. Planning commenced in 1975.

Have I failed to convince your readers that shortcomings in a service do not result only from maliciousness or policies? If so, let me cite the following passage from the *New York Times* (3).

> The fetid odor of unclean bodies and the gray-blue haze of cigarette smoke hang like smog about the destitute clientele of the Men's Shelter on the Bowery in Manhattan . . . . Ragged men with vacant eyes sit in the "Big Room" on plastic chairs attached in rows. As many as 250 men will sit all night if there are no beds available in nearby flop houses. Some men will wash themselves . . . but many refuse. Others must be deloused or they cannot stay.

Should South Africans conclude that the democratic, capitalistic, and integrational policies of the United States caused such conditions in New York City? If we accept this direct causal relationship, is not the South African service, provided under the policy of separate development, more humane? Nowhere did the committee find hospital facilities of this standard. The worst of our black patient care is better than this.

Some statements in the report are blatantly incorrect. For instance:

"An extensive response (to the draft report) submitted by the Department of Health in effect denied every critical finding." The Department has not denied a single finding. But the biased and often unsubstantiated conclusions were questioned. The Department of Health offers anyone wishing to verify this a copy of the original reply submitted to APA.

"The Department of Health acknowledged that a patient had been murdered by a nurse." This statement is the result of the following written reply from the Department of Health: "A senior psychiatrically trained (black) male nurse was involved in a struggle after which the patient died. At a trial the nurse was charged with murder but found guilty of culpable homicide."

Dr. Stone, a medical-legal expert of the Harvard University Law Faculty, should know the difference between murder and culpable homicide and that the committee's statement was grossly misleading, if not false. As Chief of Psychiatric Services, I accompanied committee members on probably 80% of their visits, and overheard some of their discussions suggesting that they had found many examples contrary to their preconceived ideas of South African conditions. I reminded them of this. Dr. Stone replied that "some are comments taken out of context, some were made to avoid conflict or confrontation and for reasons of social tact at the time (A.A. Stone: personal communication to Dr. J. de Beer, Secretary for Health, South Africa, Sept. 12, 1979). This is a very significant statement. The committee has again tied "the apartheid tin to the tail of the psychiatric cat" (4).

The committee was elected with a strong political bias. The May 1978 issue of *Bottomline* (5) reported that "Dr. Jeanne Spurlock, Deputy Medical Director [of APA and a member] of the Black Psychiatrists of America was given the assignment of working with other interested persons to determine the names of the people to serve on the committee, and to develop evaluation procedures and instruments."

The "other interested persons" were identified a few paragraphs later—"The Black Psychiatrists of America have joined in an informal coalition with various anti-apartheid groups to call for the freedom and rights for black South Africans."

Bearing in mind that the committee said things for "reasons of social tact" when they were in South Africa, the reader must wonder how much the anti-apartheid political tirade was allowed to influence their report for reasons of social tact in America.

I trust your readers will evaluate the committee's report in the light of the background information I have provided.

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Dr. Spurlock and Associates Reply for the Committee to Visit South Africa

SIR: Dr. Henning was our host in South Africa. He was unfailingly gracious and courteous to our committee throughout our stay. We very much appreciate his many acts of personal kindness to us under circumstances that must have been as difficult for him as they were for us. We hope that his letter and our response will not obscure those sentiments.

Dr. Henning's role as Chief of Psychiatric Services requires him to supervise the facilities we surveyed. His letter therefore must be understood as an official response to a critical report. The APA had given the South African Department of Health and Dr. Henning the opportunity to react to a preliminary draft of its report. We carefully reviewed their detailed critique before preparing our final report. Dr. Henning's letter echoed the substance of the earlier critique: 1) The committee had no mandate and no basis for commenting on apartheid. 2) We gave inadequate attention to positive developments reported by governmental policies. 3) Based on Dr. Henning's understanding of conversations he heard and overheard, the committee distorted the results of its survey. 4) Finally, the letter also contained a new elaboration of the South African response to our findings about the needless death of patients. We will reply to each of these:

1) The invitation from South Africa's Secretary of Health stated that "the prime objective of the visit would be to investigate the accusations of the United Nations Committee against apartheid in regard to psychiatric services in South Africa." The U.N. committee's accusations were made to direct attention to the effects of apartheid. Unequivocal data on apartheid were present in every psychiatric facility and in every community we visited. The impact of apartheid was demonstrated in the discrepancy in appropriations for black, colored, Asian, and white patients. The impact of apartheid was also demonstrated in the difference in the quality of facilities and in the quality of care, which varied directly in relationship to the racial standards established by the government of South Africa.

2. During the 18 days devoted to the survey we were involved in activities jointly planned with South African officials on 16 days. We also independently met with some people who opposed South African policies. We had indicated before going to South Africa that we intended to do this since we did not want to be limited to sources selected by the South African government. We did not find it necessary to go out of our way to meet them. We did what we had planned to do.

3. Dr. Henning indicated, based on his memory of conversations which he quoted out of context, that he was unprepared for the criticisms of the report. He suggested that our report was written by people who are easily influenced by the situation in which they find themselves and that we changed our minds about what we had seen upon returning to America. Committee members met each night in South Africa to tape collective impressions of the institutions we saw and the people we met. Our judgments of these facilities were based on our findings and our group discussions at that time. Dr. Henning was appointed by then President Jack Weinberg to include persons of appropriate expertise. The South African government was informed in advance and agreed to participation of black psychiatrists in this survey.

4. Dr. Henning cited studies that might offer a rationalization for the mortality of psychiatric patients in South Africa. He cited these studies out of context, as he does our conversations with him. Lehmann, for example, specifically refers to crowded conditions and inadequate nursing attention as important considerations in the mortality of schizophrenic patients. Furthermore, the records of the patients who died demonstrated incontrovertible evidence of absent or improper examination, diagnosis, and treatment of treatable illness where death need not have occurred. Charts were chosen at times randomly, at times by hospital personnel, and at times as a block of charts that had been filed together. In the first hospital we visited, with a census of 3,400 patients, we were ushered into a storeroom where there were 375 charts of patients who had died in the previous year. All of these charts were accessible for review, and we chose among them. We paid particular attention to the most recent deaths. Contrary to Dr. Henning's comments, we did ask the staff at some hospitals to explain shortcomings in the reported treatment of a specific deceased patient. We got no further information despite the fact that staff scurried about to find other records. We believe that any objective observer who systematically examined the charts of patients who died in these facilities would confirm our findings. We hope the South African Department of Health will follow the positive recommendations we set out in our report and seek to avoid needless deaths rather than continuing to attempt to deny and rationalize what is happening.

JEANNE SPURLOCK, M.D.
CHARLES PINDERHUGHES, M.D.
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ALAN A. STONE, M.D., CHAIRPERSON
Washington, D.C.

More on the South African Report

SIR: It would not be unreasonable to conclude that the Report of the Committee to Visit South Africa (November 1979 issue) reflected the inequitable distribution of health care to different population sectors in South Africa. The unsatisfactory nature of South African psychiatric and medical services is no less reprehensible because it stems from centuries of sociopolitical conflict between indigenous peoples and settlers. However, critics should understand this history before resorting to knee-jerk assumption.

Many miseries of the human condition may be implicated marginally as deleterious to mental health, including the military occupation of territory by foreigners, as occurs on the left bank of the Jordan River, the hostile 30-year siege of Israel by its neighbors, and the Soviet Union's repressive totalitarianism. The current state of the art of psychiatry does not justify postulating causal connections between these unhappy situations and mental illness. Despite our instinctive revulsion, the offensiveness of the apartheid system alone does not necessarily warrant implicating it as an etiological factor in the genesis of mental illness. The committee's report mentioned no epidemiologic studies of the incidence of mental illness in various South African population groups.

I question the relevance of APA involvement in South African apartheid or in other issues that do not deal with direct perversion of the medical process by failure of a nation's physicians to conform to universally accepted norms of ethical conduct. The committee's survey included no data substantiating charges of that nature.

No society is free of inequity. It must be of meager consolation to an indigent resident of the South Bronx that, while he is not barred by statute from seeking admission to Chestnut Lodge or to Shepherd-Pratt, he is inevitably obliged de facto to seek lesser quality care in a public hospital. The opportunity of society's disadvantaged for equal access to health care in the United States does not substantially differ from opportunities existing in South Africa.

ALEXANDER M. DON, M.D.
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Dr. Stone Replies for the Committee to Visit South Africa

SIR: Dr. Don presented three criticisms of the report. Dr. Pinderhughes responds below to one of these criticisms, and I shall attempt to deal with the others. The strategy of Dr. Don's first criticism was to admit the "unsatisfactory nature" of medical and psychiatric services for nonwhites, to claim that all that is bad is a legacy of the history of South Africa, and to assert that South African physicians have not violated ethical standards. Therefore, the responsibility for what is "reprehensible" lies not with psychiatrists but within the sociopolitical fabric of history which must be understood rather than treated in a "knee-jerk critical" fashion.

I agree with Dr. Don that South Africa's problems are rooted in a history that needs to be understood. So are all human problems. But history, even history understood, cannot become an acceptable excuse for continuing injustices. The Committee to Visit South Africa made a good-faith effort to understand the historical perspective both from a white and a nonwhite perspective. But we also had a duty to make a judgment about the present treatment of nonwhite patients, and we offered suggestions for their better treatment in the future. We suggested feasible remedies for the continuing injustices. Contrary to Dr. Don's reading of the

report, we did find evidence that some physicians violated accepted norms of ethical conduct—they allowed patients to die by withholding routine medical care. There was no knee-jerk response in this. We were at great pains not to condemn all of the "nation's physicians," and we suggested measures to be taken by ethical and responsible physicians and officials in South Africa to monitor and reduce these and other violations of ethical standards.

The second criticism asserted that the disadvantaged in the United States are not substantially better off than those in South Africa. Dr. Don is simply wrong. The United States certainly has not overcome all of its own racial inequities, but by any objective measure nonwhites are substantially worse off in South Africa. The notion, let him who is without sin cast the first stone, is implicit in Dr. Don's criticism. This time-honored maxim surely must be taken seriously, but should it become the justification for a conspiracy of silence among responsible physicians? There can be no question that there has been political abuse of psychiatry and mistreatment of psychiatric patients in various parts of the world, including the United States. This abuse takes various forms and psychiatrists have played different roles ranging from active complicity to passive participation. APA must acknowledge such abuse and speak against it. Otherwise we will fall into a mindless and defensive guild mentality that is amoral. We must remedy our own failings but we must not remain silent until we can speak from a posture of self-righteous perfection. Dr. Don, who is South African-born and trained, may well understand much about South Africa and its history that the committee did not. However, nowhere in his letter did he indicate that he ever visited or evaluated the treatment facilities for nonwhites in his native country and came to different conclusions than did the committee.

ALAN A. STONE, M.D.
Boston, Mass.

Dr. Pinderhughes Replies for the Committee to Visit South Africa

SIR: Dr. Don stated in his letter, "Despite our instinctive revulsion, the offensiveness of the apartheid system alone does not necessarily warrant implicating it as an etiological factor in the genesis of mental illness." The committee believes that frequent and intense life stress is an important etiological agent and precipitating factor for mental and emotional disorders and sometimes for psychosomatic and somatic disorders. In South Africa the committee observed conditions of deprivation, loss, fear, conflict, depression, loss of freedom, discrimination, disruption of family, absence of protection, and frequent encounters with separation, illness, and death. The committee considered these conditions pathogenic, causing great stress to human systems and often depriving these systems of important support. Relationships have been demonstrated between life stress and both mental and physical illnesses and between loss of social support systems and the incidence of mental and physical illness. There is no reason to believe that factors which contribute to morbidity and death in the United States do not do the same in South Africa.

CHARLES A. PINDERHUGHES, M.D.
Bedford, Mass.

Psychotherapy Standards

SIR: I was excited by the attempt of Peter Buckley, M.B., Ch.B., and associates to investigate "Common Mistakes in Psychotherapy" (December 1979 issue). However, the lack of psychotherapy standards makes such an investigation difficult. The authors claimed that "wanting to be liked by the patient" was a mistake. This hypothesis is vague. One school would argue that wanting to be liked by the patient is appropriate and similar to trying to develop a therapeutic alliance with the patient. Others would argue that wanting to be liked by the patient reflects an unresolved developmental conflict in the therapist that must be resolved before therapy can be beneficial. "Wanting to be liked by the patient" must be clarified, then the clarified hypothesis must be scientifically shown to help the outcome of therapy. This would result in a standard for testing the skill of the therapist. Unfortunately, this task has yet to be performed.

The authors considered "premature interpretation" as another mistake. Is an interpretation made in the first session premature? The authors failed to clearly define their criteria. This is also true for "overuse of intellectualization." How did they define intellectualization and what were the criteria for overuse? Much work must be done before we can confidently investigate the area of common mistakes in psychotherapy. I suspect that the authors labeled as mistakes the approaches of the residents that differed from those of the supervisors.

I feel that psychoanalytic psychotherapy is uniquely therapeutic for many situations. Unfortunately, this is still a bias and not a scientifically proven fact. Psychoanalytic hypotheses must be defined in a form that can be scientifically tested. This has not yet been done and is the major weakness in previous attempts to test the validity of psychoanalytic psychotherapy.

BRUCE ROBERTS, M.D.
Chicago, Ill.

Dr. Buckley and Associates Reply

SIR: We share Dr. Roberts' bias that psychoanalytic psychotherapy is uniquely therapeutic for many clinical situations. There is more scientific evidence for this than Dr. Roberts acknowledged. Malan (1) and Luborsky and associates (2) reported evidence favoring the use of dynamic psychotherapy in treating psychosomatic conditions. Research conducted at the Menninger and Tavistock Clinics has provided evidence for the efficacy of psychoanalytic psychotherapy in treating neuroses and character disorders (3, 4).

We agree with Dr. Roberts that psychoanalytic hypotheses must be scientifically tested and that this, along with the issue of patient selection, (i.e., which patients do best with which therapy) remains a central task for contemporary psychotherapy research.

In our study the supervising clinicians who rated the frequency of residents' "mistakes" were all very experienced psychotherapists. We felt that they possessed sufficient clinical experience to judge empirically the beginning therapists' conduct of treatment and what the root causes of badly conducted treatment were. These were individual judgments although the results were grouped for the purpose of data analysis.

We agree that the identification of specific therapeutic factors (including elements of technique) in psychoanalytic psychotherapy is another pressing question for psychotherapy research. We feel that studies similar to that of Strupp and Hadley (5) but with longer treatment courses are urgently needed.

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PETER BUCKLEY, M.B., CH.B.
TOKSOZ B. KARASU, M.D.
EDWARD CHARLES
Bronx, N.Y.

Affective Flattening and Emotional Blunting

SIR: The attempts by Nancy C. Andreasen, M.D., in "Affective Flattening and the Criteria for Schizophrenia" (July 1979 issue) and Richard Abrams, M.D., and Michael A. Taylor, M.D., in "A Rating Scale for Emotional Blunting" February 1978 issue) to discriminate the affective expressions of patients with schizophrenia from those of patients with affective disorders were thought provoking pieces of research. If these major categories of psychosis can eventually be discriminated from one another on the basis of affect, much useful clinical and possibly biological research could follow.

However, at least in Dr. Andreasen's paper, the assumption was made that emotional blunting and affective flattening are synonymous. Hinsie and Campbell (1), in defining disturbances of affectivity, state that "it is probably the incongruity between the affect displayed and the verbal productions of the patient that is more characteristic of schizophrenia than any other change in affect." Nondelusional depressed patients, in my experience, do not reveal this incongruity between the content of their verbal productions and its accompanying affective expression. Their affect expresses the same pessimism, helplessness, and hopelessness that is readily apparent in the content of their speech. This bland congruent affect is best described as flat. (This is true even of many delusionally depressed patients.) Many patients with schizophrenia, on the other hand, do not match their affect with their speech content. Thoughts of murder, fears of possible homosexual assault, or ideas of being controlled by people or machines are often accompanied by a bland, matter-of-fact affect. This bland incongruent affect is best described as blunted. When these same thoughts are accompanied by giggles or smiles, the affect is termed inappropriate.

The work of these psychiatrists is important for a more complete understanding and discrimination of the major psychoses. However, the assumption that emotional blunting and affective flattening are synonymous must be clarified so that future researchers will not become trapped in semantic confusion.

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KENNETH R. SILK, M.D.
Ann Arbor, Mich.

Dr. Andreasen Replies

SIR: Dr. Silk is quite right in pointing out that variability in the use of terms to describe psychiatric symptomatology creates a major obstacle to clear communication between clinicians and researchers. Not only do some distinguish between "blunting" and "flattening," but distinctions are often made between "emotion" and "affect." The distinction between flattening and blunting, based on congruence of the affect, is an interesting one, but it is by no means universally accepted. His definition of "incongruent affect" is quite similar to my definition of "inappropriate affect," while his "inappropriate affect" corresponds to what I would call "silly affect," which I see as a subtype of inappropriate affect. For me, the difference between blunting and flattening is quantitative rather than qualitative, with flattening being more severe and blunting less severe. I have no doubt that many other *Journal* readers define these terms quite differently than either Dr. Silk or I do.

Neither of us is necessarily right; it is difficult to develop criteria for determining "rightness" when it comes to defining terms. From the linguist's point of view, the concept of "rightness" does not apply. Words mean whatever the majority of users take them to mean. In science, however, the usual criteria are the reliability of the definition and its clinical usefulness. I hope that eventually, through the use of these criteria and through careful research, a consensus will be reached concerning the meaning of common terms. Although Dr. Silk and I do not agree on our definitions, we certainly do agree that our current semantic confusion should be reduced if not ended.

NANCY C. ANDREASEN, M.D., PH.D.
Iowa City, Iowa

Drs. Abrams and Taylor Reply

SIR: We view emotional blunting as an impairment of emotional life that may be manifested in various aspects of thinking, feeling, and behaving. For this reason we have included among the 16 items of our scale each of the phenomena Dr. Silk describes, without assigning preeminence to any single feature.

We believe that Dr. Andreasen's phrase "affective flattening" is coterminous with "emotional blunting" as we use it, although she specifically limited her rating scale items to directly observable behaviors. We agree with Dr. Andreasen that some depressed patients may show emotional blunting

and that the assessment of this feature in the diagnosis of schizophrenia should be done after excluding patients with diagnosable affective disorder.

RICHARD ABRAMS, M.D.
MICHAEL A. TAYLOR, M.D.
Chicago, Ill.

Minorities and Psychiatric Hospitalization

SIR: In "Ethnic Density and Psychiatric Hospitalization: Hazards of Minority Status" (December 1979 issue) Judith Godwin Rabkin, M.D., raised some important issues about the vulnerability entailed in minority status.

While doing research in Malaysia from 1966 to 1968 I examined the relative rates of hospitalization of Malays and Chinese Malaysians, the two major ethnic groups, in districts where they were relative minorities. Like Dr. Rabkin, I found that when these patients came from a district where they had been in the minority, they were overrepresented in the mental hospitals (1).

Among Dr. Rabkin's plausible explanations for the effect of low ethnic density and psychiatric hospitalization is "decreased support in times of difficulty, such as limited availability of sympathetic understanding, and helpful compatriots." My finding that the readmission rates more strongly reflected the relative minority status for both Malays and Chinese Malaysians than even the overall admission rates did provides evidence for this "support system" hypothesis. As we know from experience in community psychiatry, the presence or absence of a support system, whether psychiatric or interpersonal, is one of the determinants of rehospitalization.

Other explanations of Dr. Rabkin's hypothesis are exposure to others, absence of social buffers, mobility, and feelings of alienation. In another study of Malaysians I found that "fringe" type or transitional communities bear a higher risk for mental (and social) pathology (2). Such communities may be squatter settlements or semiurban villages that attempt to maintain rural traditions and practices while being bombarded by the influences of urban life.

One factor not clearly delineated by Dr. Rabkin is the greater tendency of a minority group to extrude a deviant member as an embarrassment. The majority community might not react that way in regard to one of its members. Dr. Rabkin alluded to this by asking, "Are blacks . . . more likely to be arrested in predominantly black communities and to be hospitalized in white communities?" The difference between arrest and hospitalization is the difference, usually, between externally imposed sanctions in case of arrest and internally imposed extrusion in case of hospitalization.

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JOSEPH HARTOG, M.D.
San Francisco, Calif.

Questioning Nonphysician Examiners in Psychiatric Education

SIR: I have worked in a variety of psychiatric settings—for 9 years in Czechoslovakia under Communist dictatorship, for 2 years in Austria under a democratic government, and for 11 years in the United States. In "Required Educational Objectives in Community Psychiatry" (Feburary 1979 issue) James H. Shore, M.D., and associates wrote that "psychiatric residents will be given a mini-Board examination at the end of their third year of community psychiatry rotation, which will include written examination following the ABPN national Board format and a one-hour oral examination. The oral examination will involve two examiners—a psychiatrist and a nonphysician mental health professional. This will evaluate the trainee's ability to apply his knowledge." Nonphysician professionals should never be allowed to examine or evaluate (and thereby control) a physician in his or her area of expertise. Once such a precedent is established the nonphysician mental health professional may not always be fully qualified. Psychiatry, as a medical subspecialty, could be in this way controlled not only by nonphysician mental health professionals but by others who pretend to be mental health professionals, e.g., religious and political fanatics, or U.S. citizens serving as foreign agents.

In Europe I spoke to psychiatric nursing assistants, *pflegers*, on the territory of the former German Third Reich. From 1939 to 1945, these *pflegers* were ordered to kill "untreatable" psychiatric patients by causing asphyxia through continuous application of ECT. The patient's psychiatrist had to stand by because politicians, "specialists in mental health," gave orders to physicians.

I also worked in Czechoslovakia where psychiatrists had to make false psychiatric diagnoses for political dissidents. Some psychiatrists may have done this in the hope that dissidents might receive more humane treatment in psychiatric hospitals than in labor camps. Nevertheless, nonphysicians gave the orders.

To my knowledge, no one has ever suggested that a surgical resident be examined by a nonphysician professional in surgery. Whether the idea of Dr. Shore and associates is well intended or an attempt to improve the reputation and gain the support of the nonphysician professionals and paraprofessionals in mental health remains unclear. The only situation in which a physician's professional competency and knowledge should be examined by a nonphysician is when the physician is a defendent standing before a judge and jury in a legal and democratic court or when the physician is testifying as a witness.

GEORGE O. KRIZEK, M.D.
Washington, D.C.

Dr. Shore and Associates Reply

SIR: Dr. Krizek criticized our curriculum for psychiatric residents in community psychiatry by focusing on the political implications of nonphysician control by totaljtarian governments of psychiatric treatment. We strongly concur with his concern for this abuse in any country by anyone, physician or nonphysician. However, psychiatric education in this country has a long tradition of interdisciplinary influence, exemplified by the faculty staffing pattern of most depart-

ments of psychiatry with representatives from psychology, nursing, social work, sociology, and anthropology. In many capacities these nonphysician mental health professionals participate in the education and evaluation of psychiatric residents under the authority and supervision of the psychiatry departments. Community psychiatry, in particular, requires physicians' skills in collaborating with other health professions. The participation of these colleagues in the training and evaluation of residents is a valuable educational experience for trainees and faculty alike. We do not delegate the authority for trainee evaluation. Nonphysician mental health professionals participate with us in a collaborative oral examination.

We disagree with the position that community mental health should be directed exclusively by psychiatrists. Note the current distribution of mental health administrators by discipline. At issue here are the unique role and skills of the community psychiatrist in the context of an interdisciplinary work setting. Our curriculum model realistically addressed that issue without diffusing the physician's role in community mental health. No psychiatrist should abuse his or her special responsibility, whether directed to do so by a physician or a nonphysician supervisor.

JAMES H. SHORE, M.D.
J. DAVID KINZIE, M.D.
JOSEPH D. BLOOM, M.D.
Portland, Ore.

Recognizing Geriatric Sexuality

SIR: The medical, nursing, and allied professional staff of any geriatric home or institution would agree that their objectives are to meet the physical, psychosocial, and spiritual needs of the residents. In regard to physical needs, I question the adequacy of provisions made for the sexual needs of the residents.

A prime prerequisite for the sexual experience is privacy. Residents' rooms, in practice, are not locked; most do not have locks, allowing the entrance of personnel at any time. It is difficult for residents to find the privacy they may desire to satisfy their own sexual needs.

The normalcy of the continued sexual drives of residents in their seventh, eighth, and even ninth decades should be stressed to all personnel. Staff members should regard a male resident who continues to read *Playboy* or an 86-year-old woman who is found crying because she is lonely for a mate as seriously as they would regard younger patients.

As health care professionals, we must recognize that sex, as a pleasure, a physical body function, and a need should not be denied an individual on the basis of age. It is incumbent upon us to teach others, including family members, accordingly.

MARTHA M. WILLIS, R.N.
Montreal, Que., Canada

Dermatosis and Lithium Therapy

SIR: We noted with interest the clinical report entitled, "Lithium Carbonate and Psoriasis," by Dwight L. Evans, M.D., and Willis Martin, M.D. (October 1979 issue). We wish to report another case of a dermatological complication with lithium carbonate use.

Ms. A, a 22-year-old woman with a 5-year history of bipolar affective illness, was treated with lithium carbonate. She had had no previous dermatological condition and no family history of any skin disorder. She had not previously taken lithium. Her serum lithium levels, determined biweekly, ranged from 0.55 mEq/liter to 0.65 mEq/liter. During the second week of lithium therapy Ms. A developed an acneiform rash in the perioral area that spread to both of her cheeks and later to her forehead. Cheilosis was observed at the corners of her mouth. Her condition gradually worsened over several weeks. She was referred to a dermatologist, who prescribed systemic antibiotics—tetracycline and then minocycline. In addition, he prescribed a topical compound of lincomycin and hydrocortisone in a Cetaphil lotion base. After 2½ weeks of this therapy Ms. A's condition had not improved. A gradual decrease in lithium dosage produced slight improvement. However, it was not until lithium had been discontinued for 3 weeks that Ms. A's dermatosis disappeared completely.

Ms. A's condition seems to resemble cases previously reported by Kusumi (1). We hope this report will generate further interest in the possible causal association between lithium carbonate and dermatological conditions.

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NEIL ALDOROTY, M.D.
WILLIAM R. LEVINE, M.D.
Wichita, Kans.

Psychopharmacologic Norms in Blacks and Whites

SIR: "A Pilot Study of Racial Differences in Erythrocyte Lithium Transport" by Samuel Okpaku, M.D., Ph.D., and associates (January 1980 issue) was a laudable departure from reports that are spin-offs from studies primarily aimed at other questions. The fact that previous observations of such differences have been incidental findings may have contributed to the failure of researchers to follow up their preliminary results.

More than ten years ago Overall and associates studied response patterns to antianxiety, antidepressant, and antipsychotic medications in a collaborative effort involving 372 patients in 5 Veterans Administration hospitals. They found statistically significant differences between the responses of blacks and whites, independent of social class disparities, and concluded that "race is an important additional source of difference in drug response that should be taken into account in future research" (1). Henry and associates reported similar findings in their investigation of 240 patients who had received antianxiety and antidepressant agents (2). Raskin and Crook studied 555 white and 159 black depressed patients in a multihospital study (1 state hospital, 2 private institutions, and 2 general hospitals). Black patients had a better response to medications, especially early in treatment (3). In Ziegler and Biggs' study (4) black subjects exhibited a higher mean plasma level of nortriptyline than white subjects did. Ziegler and Biggs observed that this may explain racial

difference in clinical response. Differences in lithium erythrocyte transport may have similar clinical consequences. For example, optimal plasma lithium levels for the treatment of mania are much lower in Japanese patients than in American patients (5).

Major racial differences in the action and efficacy of psychotropic medications would probably have been evident in clinical practice. But the above examples do raise the possibility of subtle variations in efficacy, steady-state plasma levels, therapeutic windows, and toxicologic norms that may act as compounding factors in research based on racially mixed groups of patients. Such subtle variations may also be important in the management of individual patients. Thus the information that depressed black women respond better to chlorpromazine than to imipramine, while the reverse is true of depressed black men (3), may be valuable in "resistant" cases.

Further research seems indicated. While prospective studies are ideal, retrospective analyses of completed investigations are also useful and have the added advantage of avoiding the Hawthorne effect, in which the act of observation significantly affects the perception of dependent measures.

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Corrections

There was an error in "Toward a Rational Pharmacotherapy of Depression," by Stephen L. Stern, M.D., A. John Rush, M.D., and J. Mendels, M.D., in the May 1980 issue. The last full sentence on page 547 should read, "For example, Glassman and associates (47) reported antidepressant response to imipramine in 13 of 14 patients. . . ." The staff regrets this error.

On page 447 of the April 1980 issue, in "Unmasking Masked Depression in Children and Adolescents," by Gabrielle A. Carlson, M.D., and Dennis P. Cantwell, M.D., the data in table 3 are incorrect. The correct table appears below.

TABLE 3
Depressive Symptoms in 61 Children Grouped According to *DSM-III* Diagnosis

	Primary Affective Disorder (N=14)		Secondary Affective Disorder with Behavior Disorder (N=9)		Behavior Disorder (N=27)		Secondary Affective Disorder with Other Disorder (N=5)		Anorexia Nervosa (N=6)		
Symptom	N	%	N	%	N	%	N	%	N	%	Significance[a]
Dysphoric mood	10	71	6	67	6	22	4	80	2	33	p<.005
Low self-esteem	9	64	8	89	12	44	4	80	1	17	p<.005
Anhedonia	7	50	7	78	10	37	4	80	0		p<.005
Fatigue	9	64	5	56	5	19	2	40	4	67	n.s.
Somatic complaints	9	64	4	44	8	30	5	100	2	33	p<.05
Suicidal ideation	12	86	9	100	10	37	4	80	3	50	p<.001
Hopelessness	10	71	6	67	10	37	4	80	3	50	p<.05

[a]The children with secondary affective disorders were compared with children with no affective disorder (chi-square analysis).

BOOK REVIEWS

NANCY C. ANDREASEN, M.D., BOOK REVIEW EDITOR

Out in Inner Space: A Psychoanalyst Explores the New Therapies, *by Dr. Stephen A. Appelbaum. Garden City, N.Y., Anchor Press/Doubleday, 1979, 510 pp., $14.95.*

In the spirit of the Zen koan one definition of enlightenment goes like this: before enlightenment one looks at a tree and sees the tree, and after enlightenment one looks at a tree and sees the tree. Before Appelbaum began his odyssey he was a psychoanalyst with an unusually open and inquiring mind, and after six years of wandering the trails and byways of humanist psychology, the consciousness movement, and holistic medicine, he was a psychoanalyst with an unusually open and inquiring mind. But he wasn't the same man.

Out in Inner Space is a companion piece to Adam Smith's *Powers of Mind* (1) (reviewed in this journal in June 1977) in that both authors are mature and sophisticated observers with sound, well-developed points of view who ventured into a psychological netherworld so near and yet so far from the disparate activities that define the American psychiatric establishment. Both authors immersed themselves—literally and figuratively—in the waters of their subject matter. Appelbaum participated in "trial by fire" in the following "outer" therapies and concerns (some farther "out" than others): Esalen workshops, acupuncture, Gestalt groups, psychic healing exercises, shiatsu massage. Kirlian photography, biofeedback, Rolfing (all 10 sessions), post-Rolf structural patterning, the Alexander technique exercises, bioenergetics therapy, est, Silva mind control, and several different kinds of meditation. He even spent 14 months on a macrobiotic diet.

Appelbaum discusses the techniques and strategies he encountered and the underlying theory of each, when there was one, in terms of psychoanalytic theory. Here the book occasionally lags, although the effort is honest and pioneering. It is not inappropriate that Appelbaum discusses psychoanalysis in the context of "outer" therapies and attempts also to defend it from its critics, but often it appears that he takes the criticism more seriously than the critics. Humanistic psychology is not yet much committed to debates on theory; for the most part it is a universe where what counts is what works. To a surprising and challenging extent, many "out" things do work, and we who are "in" have no better frames for understanding those results than labels like transference cure, suggestion, and flight into health. It is useful to remind ourselves that the main use of psychoanalytic theory is to deal with data drawn from the analytic consulting room. Applications to nonclinical or atypical clinical situations are tricky at best. On the other hand, it is surely fascinating when Appelbaum compares the methods of psychoanalysis with those of yogic meditation and finds far more homologues than anyone could expect to find by sheer coincidence. Some deep undercurrents of connection must exist between the two, but what are they?

Like *Powers of Mind, Out in Inner Space* is more a travelogue than a scientific treatise. It is a thoughtful account of a return from nearby strange, mysterious, and evocative places. It is also an invitation. One cannot come away from the book without being intrigued and challenged. There is simply too much of obvious relevance to modern psychiatry and medicine to ignore; yet, in large measure, it is ignored. The paradox that challenged Appelbaum more than any he encountered on his journey was an attitude he confronted in a respected psychiatrist who was working with cancer patients. This man not only knew virtually nothing of the meditative therapy developed by Dr. O. Carl and Stephanie Simonton, he was resistant to hearing anything about it. To quote Appelbaum, here was "a celebrated authority [in cancer], recipient of thousands of government grant dollars, functioning in one of the country's major medical centers . . . content, if not complacent, at overlooking the possibilities for curing cancer, despite evidence from his own patients" (p. 370). On January 1, 1978, 45% of all of Simonton's cancer patients were alive more than twice as long as the national average for survival after the first diagnosis of cancer; this figure is made more impressive by the notation that every Simonton patient was judged medically incurable before coming to the Simontons. Appelbaum is obviously compelled by what he saw and learned in the outer space of psychic healing. He remains at work on research in this field and has a manuscript on that topic in preparation.

Appelbaum and Smith are of a small group of objective observers who have traveled in "outer space" and returned to us with the same message: it doesn't all make sense in the paradigm of our rational Western scientific world, but "something" is out there. It seems inevitable that that "something" will be studied and that the future of our psychological sciences will be shaped thereby. *Out in Inner Space* is a fine launching pad.

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JUSTIN SIMON, M.D.
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Psychiatry for Lawyers, revised ed., *by Andrew S. Watson, M.D. New York, N.Y., International Universities Press, 1978, 451 pp., $25.00.*

"The knowne certaintie of the law, is the safetie of all," wrote the venerable Sir Edward Coke. Dr. Andrew Watson, perhaps the dean of law and psychiatry in this country, is more than a little suspicious that, in their search for a secure certainty, lawyers (and law students, whom Dr. Watson is a master at educating) may miss the enthralling ambiguities of client behavior that make law practice so rewarding for those sensitive to them. His revised edition of *Psychiatry for Lawyers* is a lucid and graceful attempt to open up new vistas for us lawyers, who are probably characterologically predisposed to be better talkers than listeners. Dr. Watson

teaches us to watch and to listen, not only to our clients but to ourselves.

He educates us in the use of transference and countertransference, through examples that the lawyer can readily understand: the young attorney, just started at work with a large law firm, who has difficulties with his superior because he unconsciously confuses him with his dogmatic father and is therefore afraid to show the necessary initiative and independence, the young woman client who tearfully seeks a divorce that, as further investigation reveals, is more on her parents' agenda than her own. In a discursive style, which is the pattern of the book, Dr. Watson instructs us in the development and growth of the personality and about the ego and its defenses and takes us gently through stages from orality to senescence. This is not a "how to do it" book, a short primer for lawyers. It is a book to be chewed and digested. If the readers' experiences are anything like my own, they will recognize some part of themselves on every page, not always with comfort but assuredly with profit. Indeed, I fear that the title of the book may unduly restrict is circulation. It is the best general introduction to normal personality development for the layman that I have yet encountered. Friends to whom I have lent it have retained it for an unusually long time, so fascinating a mirror is it on themselves and their relationships. Perhaps Dr. Watson could be persuaded to bring this out in another edition as well, with much of the legal material excised. It should be titled *Psychiatry for Everyman*.

In not being a "how to do it" book, it is the ultimate "how to do it" book because it emphasizes that a glimpse into the workings of the human mind comes only after deep and quiet pondering. This book should be read anew each time one encounters a client with a complex human problem. *Psychiatry for Lawyers* is so rich a book that new realizations and nuances will glint out from each page, making the lawyer more sensitive to his or her own role and the situation before him. One of the great virtues of this book is the sets of problems and questions that are posed after each chapter, which give the more theoretical material preceding it a local habitation and a name. We encounter "The Case of the Murder for Magic," in which a Spanish-speaking farm laborer has signed a confession stating that he buried his victim under the floor of a chicken coop and later dug up the body, decapitated it, neatly cleaned the skull, and performed a voodoo ritual. We encounter a troubled adolescent—a "nice boy" who has been given "everything he wanted"—whose mother cannot comprehend why he was arrested for thievery while at college. Dr. Watson makes the at first inexplicable much more understandable, given a person's internal history and culture, and shows how a jury can honestly be brought to the same compassionate understanding.

Dr. Watson's range of knowledge and allusion help him convey the insights of his profession. A *New Yorker* cartoon, for example, makes vivid a very destructive closeness between mother and son. He also illustrates his ideas with references to *Cat on a Hot Tin Roof, Tea and Sympathy*, President Carter's *Playboy* interview, *The Diary of Anne Frank*, and ancient Egyptian philology, among others.

What is most impressive about Dr. Watson is his honesty and lack of pretense. A final chapter on the preparation of psychiatric expert testimony for the courtroom is the chief addition to the first edition. It is clear that Dr. Watson wants to play no games. He does not want his experts to befuddle a jury with descriptive labels but to lay out clearly, after thorough preparation, the etiology of a defendant's illness or condition, how the defendant came to be the human being who exploded in a certain way. Nor should the psychiatrist be coached overmuch by the defense lawyer: to do so would detract from the naturalness of his or her presentation. He is skeptical about psychiatry's capacity to predict behavior, despite the demands of many in the legal system for such an opinion. To eliminate controversy, he suggests that expert witnesses for both the prosecution and the defense examine the defendant simultaneously. He also thinks that the defendant should not decline to take the stand. Finally, Dr. Watson is very clear regarding what psychiatric testimony is about. It is to try to explain behavior, not to make moral judgments, "for psychiatrists are no more competent to make moral judgments than other members of society." Radical recommendations, perhaps, in this age of trial tactics, but such common sense.

MAURICE DEG. FORD, J.D.
Cambridge, Mass.

The Psychopath in Society, *by Robert J. Smith. New York, N.Y., Academic Press, 1978, 157 pp., $12.00.*

The author writes that theorists and diagnosticians have presented a wide variety of viewpoints on the etiology of the psychopath: genetic, developmental, psychologic, and sociologic. To these perspectives he adds another, that the psychopath can be conceived more appropriately as the logical extension, or perhaps the fantastic exaggeration, of what Western societies demand for a person to gain individual fame and fortune.

In developing his thesis Smith leans heavily on a concept of Machiavellianism formulated by Christie in 1964. He states that Machiavellianism has four characteristics:

> 1) A relative lack of affect in interpersonal relationships—manipulating should be enhanced by viewing others as objects. 2) A lack of concern with conventional morality—regarding lying, cheating, and deceit in general, manipulators should have a utilitarian view of interaction with others. 3) A lack of gross psychopathology—the manipulator is hypothesized to take a rational view of others and is in contact with objective reality. 4) Low ideological commitment—he is more interested in tactics to an end rather than inflexible striving for the idealistic goal.

A "Mach Scale" of 20 items was constructed to identify people with Machiavellian characteristics. The scale included such items as, "The best way to handle people is to tell them what they want to hear" and "Honesty is the best policy in all cases." The "Machiavel" would agree with the first and disagree with the second. This scale has been used in studies to discover personality correlates of the Machiavel. People who receive high Mach scores are manipulative, judge others as untrustworthy, do not concern themselves altruistically, and less readily enter into relationships to produce mutual solutions to problems. The individual with a high Mach score is also relatively unmoved by others and seems equally unaffected by his or her own beliefs and even

his own behavior, can successfully argue a position different from his own, is rated as less anxious by judges, and can look an accuser in the eye steadily while denying cheating. These traits are called the "cool syndrome." They share commonalities with the portrait of the psychopath painted by the theorists: manipulative, self-oriented, convincing, and distrustful.

Smith says that "the social philosophy basic to a given culture colors the process of diagnosing psychological problems." The American personality has undergone a change from the "Protestant ethic," with its emphasis on hard work, frugality, and prudence, to a new "social ethic," concerned with enjoyment and consumption and obsessed with selling and buying. A philosophy of the marketplace prevails in which selling one's personality promotes achievement.

The achieving personality is described as adventurous, self-confident, assertive, restless, ambitious, self-centered, shrewd, competitive, and individualistic. Superlatives are used to extol this personality as a superstar who has an obsession with display and bigness. The culture applauds and reinforces behavior that earns the label "psychopath."

> If the . . . culture requires projecting a good image while watching out for oneself, if it encourages pursuit of material pleasure and the merchandizing of people, then far from being a mask of sanity or a moral imbecile, the psychopath is the reasonable one and those of us who are trusting, reliable, and empathic are out of phase with reality.

This is an interesting point of view in a well-written book. I recommend it be read by all who are concerned with the psychopath.

PAUL E. HUSTON, M.D.
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Clinical Pharmacology of Psychotherapeutic Drugs, *by Leo E. Hollister, M.D. New York, N.Y., Churchill Livingston, 1978, 226 pp., $19.50.*

Over the past four to five years a plethora of monographs in the field of psychopharmacology has appeared. With the proliferation of so many books in this field, I wonder where the value of yet another monograph lies. *Clinical Pharmacology of Psychotherapeutic Drugs*, however, is truly a unique book. Perhaps its major strength lies in Dr. Hollister's ability to convey the knowledge of a man who has devoted over a quarter of a century of his professional life as a teacher, clinician, and researcher to clinical psychopharmacology. Dr. Hollister's ability to integrate these three areas of endeavor and to interweave the pharmacologic principles of psychotropic drugs in the context of treating the patient has resulted in a monograph that is clear, concise, and filled with factual information.

Chapter 1 is an overview of Dr. Hollister's approach to the use of psychotherapeutic drugs in the treatment of emotional illness. Although this is not a major philosophical treatise, Dr. Hollister discusses his approach to treatment, which includes recognition of mental illness, the utility of drug treatment, the benefits to be derived and the risks to be weighed when using psychotherapeutic drugs, and a conceptualization of the "ideal drug."

Chapter 2, "Antianxiety Drugs," begins with a brief discussion of the nature of anxiety, the chemical classes of anxiolytic drugs, pharmacologic action of specific drugs, pharmacokinetics, and metabolism, followed by a discussion of the clinical uses of these compounds with emphasis on dosage, therapeutic and side effects, and the general use of the antianxiety drugs. The section on pharmacokinetics and metabolism is extremely helpful in understanding the action of the benzodiazepine drugs and those characteristics which distinguish benzodiazepine drugs from one another. As an example, intramuscular diazepam and chlordiazepoxide are absorbed erratically due to the vehicle used in the manufacture of both these drugs. Thus, in a clinical situation where diazepam or chlordiazepoxide is to be used, the preferred route of administration would be orally, or intravenously if the parenteral route of administration is desired.

The section on Clinical Use of Anxiolytic Drugs is rich with practical suggestions for the clinician treating the patient for whom an anxiolytic drug would be prescribed. Dosage schedules, drug combination, duration of treatment, interactions, and side effects are some of the areas discussed. This format is basically carried out in each of the four subsequent chapters, "Hypnotic Drugs," "Antidepressant Drugs," "Antipsychotic Drugs," and "Lithium and Manic-Depressive Disorder." In each of the chapters Dr. Hollister carefully develops pharmacologic and pharmacokinetic principles, discussing plasma drug levels, metabolism, and kinetics, intertwining these principles into treatment concepts.

The understanding of the clinical use of antidepressant drugs is made clearer by inclusion of a discussion of metabolism, pharmacokinetics, and plasma levels. The practical considerations in the prescribing of tricyclics or monoamine oxidase (MAO) inhibitors in the treatment of depression is presented in such a fashion that the reader gains a full understanding of these drugs in respect to dosage, spectrum of activity, and side effects.

To review each of the remaining chapters would be redundant. Each chapter follows the format described above so that the reader can logically follow each chapter in the monograph and make comparisons between classes of drugs with relative ease.

In summary, *Clinical Pharmacology of Psychotherapeutic Drugs* is a concise, clear, erudite book. This monograph provides technical and clinical information and would be a valuable asset to medical students in their clerkship in psychiatry and clinical pharmacology, to the resident in psychiatry as well as any specialty where psychotropic drugs are used, and to the seasoned clinician and/or researcher.

BURTON J. GOLDSTEIN, M.D.
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Current Developments in Psychopharmacology, Vol. 5, *edited by W.B. Essman and L. Valzelli. New York, N.Y., SP Medical & Scientific Books (Spectrum Publications), 1979, 465 pp., $50.00.*

This volume consists of 10 invited papers representing important current developments in the field of neuropsychopharmacology. The search for a "language" to facilitate communication between basic scientists and clinicians is the leading theme of the book. Andreoli and Maffei review methods in collecting clinical data that meaningfully communicate.

to basic research, and Valzelli discusses some of the neurochemical contributions that have opened the possibility of communication between biochemical concepts and behavior.

Recognition that putative neurotransmitters may have a role in the regulation of normal and pathological behavior has stimulated research to explore whether substances such as histamine fulfill the criteria of neurotransmitters (the chapter by Schwartz and associates) and to develop techniques such as *d*-amphetamine-induced hypothermia that could render the effects of pharmacological agents on specific neurotransmitter functions accessible to investigation (the chapter by Yehuda). Shore discusses the effect of stress on neurotransmitter kinetics, with consideration of biochemical genetics, in relationship to the psychobiology of schizophrenia, and Burns and Mendels review the possible role of the neurotransmitter serotonin in affective disorders. In describing circadian rhythms in manic-melancholic disorders, however, Mellerup and Rafaelsen hypothesize that very rapid changes in mood indicate derangements of distribution rather than derangements of production of neurotransmitters, neurohormones, or other critical factors.

There is also a paper by Malick on the pharmacology of isolation-induced aggressive behavior and by Brain and Bowden on sex-steroid control of fighting in male mice. Rigter and Van Riezen review current developments in regard to the effect of neuropeptides in amnesia with special emphasis on the possibility that ACTH analogues may facilitate the retrieval process and vasopressin analogues may facilitate both memory consolidation and retrieval.

The authors included in this volume are prominent scientists and clinicians; their contributions are of high quality. The editors have done a commendable job in identifying topics that are of importance. However, it should be recognized that each paper in this volume is written for a different audience.

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Progress in Psychobiology and Physiological Psychology, Vol. 8, *edited by James M. Sprague and Alan N. Epstein. New York, N.Y., Academic Press, 1979, 334 pp., $35.00.*

This monograph might be considered of greatest interest to the neurophysiologist, to the behaviorist, to the neurologist, and, finally, to the psychiatrist, in that order. The topics covered in the seven chapters range from somewhat exotic areas of neuroanatomy to a variety of animal behavior experiments involving regulation of such basic functions as sleep, wakefulness, stress, appetite, suckling behavior, and mother-young relations. In all instances the chapter authors are well versed in their topic and write with commendable clarity and generality. Fortunately, the volume is not at all rendered obscure by the use of specialized terminologies.

The first chapter reviews the neuroanatomic and physiologic relationships of cortex and subcortex and presents some new ideas that would be of considerable interest to the anatomist and neurophysiologist. The second chapter reviews recovery mechanisms that are manifested following lesions of the visual cortex in the monkey. This is followed by a rather specialized chapter on neurophysiologic mechanisms operative during sleep that particularly emphasizes "PGO waves" and their occurrence in REM sleep. This is meant for the specialist.

Although the first three chapters could be interesting to neurologists, their psychiatric application must be regarded as minimal. However, the fourth chapter has to do with stress and arousal and reviews a psychoendocrine hypothesis to account for these phenomena. This chapter will be of considerable interest to both specialists in the field and those psychiatrists who wish some guidance and stimulation from basic research. The next chapter, on postprandial satiety, reviews mechanisms of appetite, surely of interest to those who treat the obese and who are concerned with burgeoning problems of fitness and overweight. Finally, there are two chapters that provide some basic information on the development of suckling behavior. Specifically, the last chapter concerns the interrelationships of mothers and their young.

In summary, the average psychiatrist will not find very much of interest or importance in this volume. However, for those with an academic inclination or those who have to present lectures on the more basic aspects of psychologic function there is some very significant and well digested information. Material in some of the later chapters would also form an excellent background for discussion of clinical syndromes with an organic or neuroendocrine bent, and the inclusion of such information would often add some interest and excitement to an exposition of what might be an otherwise rather humdrum clinical case.

REGINALD G. BICKFORD, M.B.B.CHIR.
La Jolla, Calif.

Research in the Psychobiology of Human Behavior, *edited by Eugene Meyer III and Joseph V. Brady. Baltimore, Md., Johns Hopkins University Press, 1979, 136 pp., $14.00.*

The seven papers that comprise this volume were presented as part of the Adolf Meyer Symposium on Psychobiology in 1976, organized to mark the centennial of the Johns Hopkins University and to honor the first chairman of the Henry Phipps Psychiatric Clinic and originator of the term "psychobiology." The editor's preface describes the great influence Adolf Meyer had on the practice of psychiatry in this country.

The text is a wide-ranging collection of reviews of the contributors' work in an area that can be broadly defined as psychobiology. For the most part, this work has been described elsewhere in greater detail and is now dated by three to four years. Nonetheless, if some of the chapter descriptions below sound unfamiliar and interesting, the reader might judge the book a worthwhile purchase.

Floyd Bloom starts with a description of some of his work on norepinephrine synapses in the brain, particularly those projecting from the locus coeruleus to cerebellar Purkinje cells and hippocampal pyramidal cells. He discusses electrophysiological, histochemical, biochemical, and behavioral evidence suggesting that the locus coeruleus may function as a system for attributing value to environmental signals and relates possible malfunction to perceptual inundation in schizophrenia. This section especially suffers from being dated by several years because there is no mention of newer pharmacological data such as the role of opiates in locus coeruleus function (1, 2). He concludes with a brief allusion to possible animal lesion models of depression.

Seymour Kety continues with his well-known work on the genetics of schizophrenia, using adoption records in Denmark. After a brief discussion of earlier twin studies (which found a concordance rate of about 40%–60% in monozygotic twins versus about 10% in dizygotic twins) and their limitations, he gives a fairly complete description of his own multifaceted studies of adoptive versus biological relatives of schizophrenic index cases. As shown in detailed tables, the overall incidence of schizophrenic illness in biological relatives was about 14% compared with about 3% in adoptive relatives. Kety eliminates possible uterine and early mothering influences by demonstrating an increased incidence of schizophrenic illness in paternal half-siblings of index cases compared with controls.

Edward Sachar discusses three approaches in the field of psychoneuroendocrinology: 1) studies of the effects of psychological stress on endocrine function and the benefits of being able to cope, 2) analysis of neuroendocrine regulation as a window on hypothalamic function in psychotic states, with emphasis on the hypersecretion of ACTH in depressive illness and the normal inhibitory effects of norepinephrine on ACTH, and 3) the use of hormonal responses to illuminate psychoactive drug effects, with emphasis on dopamine's inhibition of prolactin secretion and the effects of neuroleptics. The major exception to the correlations that are described in the last section was clozapine, which produced little or no extra prolactin secretion in spite of being an antischizophrenic drug.

Neal Miller's chapter, titled "Psychosomatic Effects of Learning," starts with a discussion of the accelerating pace of change, the stresses it generates, and their possible effects. He continues with experimental studies in rats demonstrating the beneficial effects of being able to anticipate or avoid shocks in terms of reducing stomach lesions, reducing corticosteroid levels, or elevating brain norepinephrine levels. In some experiments, pargyline or habituation to stress or to tetrabenazine appeared to protect rats from behavioral disruption produced by subsequent exposure to the stress of severe, unpredictable, inescapable electric shocks. After briefly considering the effects of stress on the cardiovascular and immune systems, Miller ends with a discussion of the conditioning of visceral responses and the use of biofeedback in combatting postural hypotension in paraplegic subjects.

John and Beatrice Lacey, in a somewhat difficult to follow chapter titled "Somatopsychic Effects of Interoception," present evidence in favor of their theory that baroreceptor discharge is inhibitory to sensorimotor activity and is facilatory to internalized cognitive abilities. The major evidence was the demonstration of time variation within the cardiac cycle of motor responses that gated further sensory input.

Martin Roth briefly describes his scheme for classifying affective disorders, emphasizing the importance of classification in spite of its neglect by Meyer and emphasizing the true heterogeneity of the conditions discussed. Although he states that the utility of classification comes from the ability to predict response to different forms of therapy, he makes no attempt to incorporate possible biochemical differences among patients into the classification.

The concluding chapter by Gerald Klerman considers the legacy of Adolf Meyer and how the Meyerian pluralistic approach and emphasis on early environment should be modified to incorporate greater use of statistics and adequate controls.

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Crazy Talk: A Study of the Discourse of Schizophrenic Speakers, *by Sherry Rochester and J.R. Martin. New York, N.Y., Plenum Press, 1979, 221 pp., $25.00.*

This experimental study of the discourse of schizophrenic speakers is a landmark in the interdisciplinary approach to cognition and language. At best, talk about talk introduces complexities that call for the use of a metalanguage. Talk about "crazy" talk adds a psychological dimension. The authors resolve this requirement with the high-powered lense of psycholinguistics, focused precisely on the particularities and operations of language that mediate coherence and meaning.

Crazy Talk introduces the notion of discourse failures that "occur when the listener cannot follow the speaker, even though the speaker is using familiar words in generally well-formed sentences." Talk familiar to the clinician as schizophrenic is illustrative of discourse failure. To approximate natural language use, the authors select context material extended in time and spread over a range of situations: the unstructured interview, narrative, and cartoon interpretation. The speakers were three groups of 10 subjects each: 10 normal voluntary subjects and 20 schizophrenic patients subdivided in accordance with an index of formal signs of thought disorder.

The study achieves a verifiable documentation of linguistic operations involved in the aberrant talk of schizophrenic speakers and, further, demonstrates significant differences in these operations correlated with subjects with and without thought disorders. The two principal linguistic methods introduced as exploratory instruments are cohesion analysis and the network system of reference and retrieval categories.

The procedural and experimental part of the analysis, which constitutes a major segment of this book, is presented in detail and accompanied by careful critique and evaluation. A very rewarding feature of this study is the evaluation of assumptions and hypotheses that have guided theoretical and experimental approaches to schizophrenic thought and language. This leads to a survey of the pertinent linguistic methodology of recent decades. In the theoretical justification for their experimental design, the authors provide an instructive exposition of linguistic concepts and methods.

The concluding section places the interpretation of findings within the original objective. With this as a point of departure, the authors engage the reader in a challenging consideration of theoretical implications and hypotheses; for example, language versus thought as the essential problem in thought disorder, evidence of lateralized dysfunction in schizophrenia, and thought disorder and diagnosis of schizophrenia.

This study provides a model for the transformation of impressionistic and intuitively perceived "crazy talk" into a

systematized, reasoned, verifiable, and predictable assessment of deviations in discourse.

MARIA LORENZ, M.D.
Belmont, Mass.

Dual-Career Marriage: Conflict and Treatment, *by David G. Rice, Ph.D. New York, N.Y., Free Press (Macmillan Publishing Co.), 1979, 185 pp., $12.95.*

Dr. Rice has written a lucid book that provides psychological understanding and clinical approaches in an area of our rapidly changing psychosocial system: marriages in which both husband and wife have a career. The strength of the book lies in its solid clinical base, frequently illustrated by process notes from couple therapy sessions from Dr. Rice's practice.

The book is rich with thoughtful assessments of the personality traits of spouses that suggest either a good or a bad prognosis for the marriage. As an example, the troublesome dynamics of vulnerability to an injury to self-esteem due to frustration of dependency needs suggest a bad prognosis. From his complex clinical data Dr. Rice abstracts three dynamic issues that carry the greatest emotional charge in dual-career marriages: 1) competition for power, 2) frustrated dependency needs, and 3) lack of adequate support and appreciation.

He details the problem of a dearth of successful models for dual-career marriages. Marriage that begins with only the husband working is studied with particular attention to the need for techniques of working out a new marital contract (analogous to the findings of Visher and Visher [1] with regard to "blended" families).

The author shows a special sensitivity to the different issues that face married career women: the need to integrate the roles of career person, spouse, and, often, mother. Dr. Rice documents the career woman's need for a role model, especially in a close woman relative or friend.

There are many useful technical suggestions in the chapters on marital therapy. Dr. Rice uses clinical case examples to illustrate the following techniques: careful assessment of how the couple use their time; task assignments, beginning with the easier tasks; priority of attention to affective issues, especially anger, before addressing the specific areas of conflict; and the values of different forms of cotherapy.

The focus of understanding is largely transactional, with attention to how the dual-career marriage magnifies unresolved role and dependency problems that derive from families of origin. There is little attention to the study of intrapsychic issues, such as oedipal dynamics and separation-individuation. Larger systems and structural issues are also neglected to some degree. For example, the role of members of families of origin in the couple's current relationship are rarely addressed.

Apparently, Dr. Rice and his colleagues do not routinely see the couple's children. This omission risks overlooking the important questions of whether the children have been triangulated into the marital conflict and the developmental impairment they may suffer as a result of the marital stresses.

Overall, however, this is a useful book for therapists working with dual-career couples and for the couples themselves.

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NORMAN I. MOSS, M.D.
Newton, Mass.

Psychopathology of Aging, *edited by Oscar J. Kaplan. New York, N.Y., Academic Press, 1979, 309 pp., $24.50.*

The editor defines the objectives of this book in his introduction:

> This book is a series of original essays on selected topics in geriatric psychiatry. It does not pretend to be encyclopedic in its coverage. However, it does seek to define the present frontiers in a number of significant areas.

How well does the book achieve this goal? The frontier persons, I suppose I must say, include veterans like Charles V. Ford and Lissy F. Jarvik on "Genetic Aspects of Psychopathological Disorders in Later Life," June E. Blum and Marcella Bakur Weiner on "Neurosis in the Older Adult," and some comparative newcomers like Robert H. Gerner, whose thorough review of "Depression in the Elderly" has 13 pages of references. Other chapters, such as "Acute, Reversible, Psychotic Reactions in Geriatric Patients" by Ivan N. Mensh, review work in the subject over the last decade.

Eugene Ziskind contributes an interesting chapter on "The Older Sociopath." He describes the six-year-old Gateways Research Project in which psychiatrists, psychologists, and social workers collaborated. This research, among other things, demonstrated defective laboratory conditioning of sociopathic subjects compared with control subjects.

Warren Breed and Carol L. Huffine write a thoughtful chapter on "Sex Differences in Suicide Among Older White Americans." They conclude that "differential socialization experiences will result in greater adaptability among girls in this society than among boys. These differences tend to be reinforced by typical adult role patterns of men and women and are ultimately reflected, we believe, in differential suicide rates" (p. 307). Some might disagree with their emphasis on adaptability as a major factor that depends, in turn, on socialization and developmental experiences. Such a concept appears too general to account for the well-known wish to die and its translation into weaker suicidal activity in physically ill elderly women compared with elderly men. The authors do not mention the observed fact that older women make more unsuccessful attempts at suicide than elderly men. Forced dependency through chronic ill health and self-hatred from feeling useless and a burden are readily observed in the elderly; presumably they affect men more than women. Here, adaptability as a factor seems less relevant.

Kaplan contributes an authoritative article on "Psychological Testing of Seniles." One of the minor irritations in the book is his persistent use of the undefined and pejorative adjective "senile" as a noun. He devotes considerable space to a description of his Psychological Abilities Scale for Seniles (PASS), evolved from a study he describes as "one of the most systematic and extensive ever undertaken on cogni-

tive functioning in seniles" (p. 60). Earlier in his introductory chapter he writes,

> Although it is unlikely that many cases [of late life dementia] will prove to be the result of some primary cause, such as slow-growing viruses or aluminum accumulation or genetic defect, a likely possibility is that a high percentage of the cases are of multiple causation and develop over a period of many years. This has led to the formulation of the Kaplan Cumulative Insult Theory, which postulates that some cases of senile dementia are the result of lifelong loss of cortical neurones through both "normal" aging and various pathological processes and events.

This begs the question, indeed.

Apart from occasional faults like these, Kaplan and his coauthors have drawn a helpful map of the frontiers in the psychopathology of aging. If some of them wander through treacherous swamps and fens, this is because our current knowledge is far too weak to allow us to draw them on firmer ground.

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Soviet Psychoprisons, *by Harvey Fireside. New York, N.Y., W.W. Norton & Co., 1979, 193 pp., $11.95.*

In the last few years there has been a flurry of books dealing with the Soviet misuse of psychiatry for political purposes. For example, Victor Nekipelov's *Institute of Fools* (1) describes the apex of such misuse—the Serbsky Forensic Psychiatry Institute, where he was briefly detained in 1973. Alexander Podrabindek, the young medical assistant who is in exile for publicizing the political use of psychiatry, has written a comprehensive account of the practice that tallies closely with an earlier report and analysis by Peter Reddaway and myself (2). Victims of the abuse have also detailed their experiences; particularly illuminating are those of Bukovsky and Plyuschch.

Fireside, a political scientist, has added to this expanding literature a brief, clearly written volume that brings the continuing "saga" up to date. There is a foreword by Zhores Medvedev, a distinguished geneticist who was himself detained for 19 days in the Kaluga Mental Hospital in 1970. His forcible admission was undoubtedly prompted by the political authorities who, realizing Medvedev's international reputation, resorted to psychiatric hospitalization in the hope that this would cause less unfavorable publicity than his arrest or deportation. The authorities miscalculated: a furor ensued both in the USSR and abroad, and Medvedev was quickly released. Despite Medvedev's gripping account of this episode (3), Western psychiatrists were somewhat incredulous in the early 1970s that their Soviet counterparts could have participated in such unethical practices. Since then the evidence of blatant and widespread abuse has become overwhelming. Now only a minority of Western psychiatrists agree with the Soviet defenders of their profession that the allegations of abuse are groundless and merely an "element in the cold war."

Fireside's book deals with several aspects of the abuse, including the motivations of the Soviet doctors involved, the victims, and opposition to the practices in the USSR and the West. The treatment of these topics is, however, rather brief and chiefly descriptive; little attempt is made at analysis. The second half of the book consists of several intriguing appendixes. Included is the "Manual on Psychiatry for Dissidents," a unique document co-authored by Bukovsky and Dr. Semyon Gluzman when they were interned for a short time in a labor camp together. The manual's chief purpose is to instruct human rights activists, would-be emigrants, religious dissidents, and others who have suffered at the hands of the KGB on how to deal with the psychiatrists who diagnose their "ideas of reformism" as evidence of severe mental illness. The manual also offers advice to the dissident on how to recant should he be unfortunate enough to be admitted to a mental hospital. The fact that there is a need for a manual of this kind is in itself an indictment of contemporary Soviet psychiatry.

Other appendixes are a summary of Podrabinek's book and personal accounts by three dissenters, Vladimir Gusarov, Oleg Solovyov, and Mikhail Kukobaka, of their psychiatric experiences. These accounts offer a vivid portrait of life inside both prison and civilian psychiatric hospitals.

It seems tragic that as we enter the 1980s there is still a need for books such as *Soviet Psychoprisons*. The story of Soviet abuse continues; dissidents are still detained in hospitals for nonmedical purposes, while those released live under the possibility of forcible readmission as long as they persist in their "political behavior." One hopes that the newly established Review Committee of the World Psychiatric Association, which is empowered to examine cases of abuse wherever they occur, will help to contribute to the closing of a shameful chapter in the history of psychiatry.

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Applied Hypnosis and Hyperempiria, *by Don E. Gibbons. New York, N.Y., Plenum Press, 1979, 202 pp., $18.50.*

Hypnosis has become an acceptable method of treatment. According to the foreword of this book, "During the past twenty-five years, more research has been conducted on hypnosis than in all the preceding years since Mesmer." The book is well written and, for the most part, adequately documented. It contains 13 chapters, some of which are only 1–3 pages in length. The induction protocols are detailed and clearly presented. There are practical suggestions for the deepening of hypnosis, the retrieving of repressed or forgotten material, improved creativity and performance, and terminating the trance.

The blurb on the inside of the front cover states that this book is an "entirely new approach" called "hyperempiria" (a hyperexperience)—"a mind expanding induction technique based upon heightened awareness originated by the author."

The methodology is neither "new" nor "mind expanding" (1). Therapeutic results will probably be the same whether the induction suggestions are passive and sleeplike or more active and suggesting a heightened sensory awareness. The hypnotic experience is more than a "believed-in imagination" (according to Sarbin). The external behavioral aspects of hypnosis are artifacts that depend on the patient's expectations, what he or she has learned about hypnosis, and the operator's cues (according to Orne). The hypnotist only sets the stage. It is the subject who is actually in control. The subject is not a passive agent but an active participant who uses the hypnotist for his or her own needs (2).

Since this book is another "how to do it" text, all subjective reports of what is going on in the subject have been excluded. Hypnosis, however, is a type of effortless, receptive attention, which occurs only in a special relationship with a trusted guide, whom the subject has selected at a particular time in his or her illness and endowed with the power to heal (to alleviate anxiety, expiate guilt, reduce pain, and expedite the recall of significant traumatic memories) (2).

Chapter 1 ("The Nature of the Induction Process") contains two significant historical errors. The first is that Puységur's patient, who became the prototype of "artifical somnambulism," was named Victor Race, not Victor Immanuel. This patient's sleeplike, "lucid" trance state was not "first manifested by people who were imitating the behavior of a retardate, who was too stupid to realize that he was supposed to go into convulsions (Mesmer's crisis) and went to sleep instead" (p. 5).

The author has altogether overlooked the historical significances of Puységur's momentous discovery of the "new" magnetism, which, as Ellenberger (3) indicated, "gave a new course to the evolution of 'animal magnetism.' . . . This discovery equals or exceeds the importance of Mesmer's work." The real founder of modern hypnosis is Puységur. It was from Victor Race that he learned that magnetism should not be used for public demonstrations (Victor became worse or refused to perform) but only for therapeutic purposes. Puységur also learned that the "pure crisis" (artificial somnambulism) involved not only the will of the magnetizer but also the rapport between the operator and his subject; this made Mesmer's theories scientifically preposterous. Puységur determined that Victor was brighter and more alert in the trance than in his normal state, that he had no memory of having been magnetized (spontaneous amnesia), and that he thought that he could diagnose his own and other patients' diseases and could prescribe treatment while in the trance.

The second error is the incorrect description of the classical Braid technique of hypnotic induction. Braid did not "swing" a bright shining object "back and forth" while giving the subject "a steady stream of suggestions to the effect that his eyes are becoming heavy" and "that he will want to close them" (pp. 12, 43).

Braid (4) wrote,

> Take any bright object, hold it about 8 to 15 inches from the eyes, at such position above the forehead as may be necessary to produce the *greatest possible strain* upon the eyes. . . . The patient *must* be made to understand that he is to keep his mind *riveted on the idea of that one object*.

Braid *never* used suggestions of eye heaviness. This type of induction was Liebault's contribution, which was taken over by Bernheim.

The author briefly describes various hypnotic models, including Orne's trance logic in the state of altered consciousness, Hilgard's neodissociation hypothesis, Coe and Sarbin's role-playing paradigm, Gill and Brenman's transference-regression theory, and Barber's social-psychological variables. It is Gibbons' opinion that "trance logic, rapport, transference, and disassociation phenomena are *all* products of suggestion, rather than the trance state itself." He takes no special position in regard to any of these models and states, "If usefulness rather than 'Truth' becomes the criterion by which various models are to be evaluated, it follows that models which initially appear to be quite different from one another need not be considered as mutually exclusive."

This "how-to-do-it" text will be of value to beginners, who need to learn what to say during the initial induction procedure. The book represents a start and requires that its reader learn more about the psychodynamics of the trance relationship and its meaning to the patient.

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Between Reality and Fantasy: Transitional Objects and Phenomena, *edited by Simon A. Grolnick, M.D., and Leonard Barkin, M.D., in collaboration with Werner Muensterberger, Ph.D. New York, N.Y., Jason Aronson, 1978, 555 pp., $30.00.*

This volume is substantially a memorial to the personality and work of the late Donald W. Winnicott. Winnicott's more than 40 years as a practicing pediatrician, during which he had countless rich opportunities for the observation of behavior in children, and his many years as a clinical psychoanalyst, during which he was able more systematically to explore the inner workings of the minds of not only children but also adults, put him in a unique position to explore and contribute to our understanding of the interplay between childhood experience and adult personality. Because of Winnicott's personality, however, his contributions tend to be more genial and inspirational than systematic or carefully elaborated. His genial inspirations rose out of the chaos of his own struggles to understand, a view that Winnicott was not slow to articulate.

As is so often the case with explorations of the fragile boundaries of any science, inspired forages must be followed by attempts of a more systematic and plodding nature to sort out the territory, to integrate what has been discovered with what is already known, and to tidy up the mess. *Between Reality and Fantasy* represents an effort to tidy up a bit, without carrying the impetus of Winnicott's contribution forward to any noteworthy extent. Much of the tidying up has to do with clarifying and specifying what precisely is meant by "transitional object" and "transitional phenomena." Winnicott used terms somewhat loosely, and there is a need

for clarifying the transitional usage from issues of pathological infantile fetishism on the one hand and problems of perception and apperception on the other. Questions in definition arise; for example, the extent to which and the manner in which the transitional object comes to replace the mother and whether it attains a greater valence for the child than does the person of the mother herself. An additional matter has to do with the extent of parental involvement in the choice of transitional object and in the emergence of the transitional phenomenon itself. These are all matters of degree and involve a spectrum of transitional phenomena that require further delineation and discussion.

An effort is made in several pieces to extend more deeply the empirical basis on which the transitional schema builds. This effort is carried on primarily by child analysts and psychiatrists and has immediate relevance to concerns about personality development. The attempts at theoretical integration seem somewhat more tenuous. Efforts are made to integrate Winnicott's insights with the work of Spitz, for example, and with early matters of physiological regulation in the interaction between mother and child. Some of this must be considered highly speculative, and a word of caution is indicated because the authors engaged in this speculative venture are not always clear as to what is actually a theoretical extension of Winnicott's formulations and what may be venturesome speculation.

One of the more important sections of the book is a series of clinical studies that attempt to apply Winnicott's notion of transitional phenomena to aspects of clinical work. Subjects covered include the "analyzing instrument," transferences, dreaming phenomena, self-object relations, obsessive-compulsive states, and particular forms of clinical manifestation such as friendships, attachments to cats, infantile fetishes, and delusions.

Following Winnicott's observation that transitional phenomena are the basis for the development of illusion, which includes the locus of cultural experience, there is a rather fascinating section on extensions of the transitional frame of reference to aspects of human creativity. Particular studies address themselves to Etruscan burial symbols, transitional language usage, and poetic imagery in the work of Gerard Manley Hopkins and Emily Dickinson. Particularly noteworthy and disappointing here is the lack of an examination of the role of transitional phenomena in religious experience.

Fortunately, and refreshingly I might add, dissenting voices who sing *extra choram* are included in the book. Anthony Flew's acute and telling critique of the inherent logic of Winnicott's formulations deserves thoughtful and careful reading as a helpful corrective to the sometimes too easy extension of the notions of transitional phenomena and illusion to broad and somewhat diffuse areas of human experience.

All in all, I consider this volume a substantial contribution to the psychoanalytic and psychiatric literature, one that brings a greater degree of substance and articulation to the often amorphous if intuitive and tantalizing formulations in Winnicott's work. Clinicians who admire Winnicott's contributions will find their thinking in many areas of application to be considerably deepened and amplified. Those who are drawn to try to integrate Winnicott's thinking in a more systematic or theoretical way will come away sobered but, I think, not discouraged.

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Working with the Impulsive Person, *edited by Howard A. Wishnie and Joyce Nevis-Olesen. New York, N.Y., Plenum Press, 1979, 184 pp., $19.50.*

Impulsivity has a long heritage in psychiatry. Many of the profession's pioneers, such as Freud, Fenichel, and Alexander, described impulsivity and acting out and wrote about impulsive characters; later workers, such as Greenacre and Johnson, shed light on the problems of acting out and the etiologies of antisocial impulsivity. In spite of the wealth of theoretical material, however, there is a paucity in the literature on the treatment of impulsive patients. Many clinicians are interested in the phenomenology of impulsivity but are disdainful of the treatment of these patients and stay away from them.

This volume contains papers on etiology, clinical approaches, the criminal justice system, and a historical overview. The tone of the book is psychodynamic, and most contributors spend time on intrapsychic mechanisms. The first three chapters deal with early childhood development, object relations theories, and criminologic considerations. The next group of papers are more therapeutically oriented and deal with alliances. There are two papers on drug abuse that have a theoretical flavor. Dr. Wishnie's own chapter on panic states summarizes some material presented in his previous fine book titled *The Impulsive Personality* (1). I enjoyed his vignettes and diagrammatic conceptualizations of impulsive behavior. A final section of the book is more forensic in nature and deals with legal decisions affecting violence and dangerousness.

The chapters are very uneven in quality. If I had my way, I would have published only the clinical approaches because we have been deluged with theory and need more practical management techniques. One omission from the book is any discussion of important developments in the neurophysiology of impulsivity. Monroe (2) and others have made important contributions toward the biologic understanding of impulsivity. Such work, together with the observations made by Dr. Wishnie, gives therapeutic insight into behaviors that otherwise appear perplexing and chaotic.

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Books Received

This list acknowledges the receipt of recent books. Books of particular interest to the readers of this journal will be reviewed as space permits, and copies of the reviews will be sent to the publishers. Books cannot be returned to the publishers.

Parent Education and Intervention Handbook, *edited by Richard R. Abidin, Ed.D. Springfield, Ill., Charles C Thomas, 1980, 569 pp., $24.50.*

Handbook of Adolescent Psychology, *edited by Joseph Adelson. New York, N.Y., John Wiley & Sons, 1980, 597 pp., $39.95.*

Minds in Many Pieces: The Makings of a Very Special Doctor, *by Ralph Allison, M D., with Ted Schwarz. New York, N.Y., Rawson, Wade, 1980, 206 pp., $10.95.*

The Psychopathology of Women, *by Ihsan Al-Issa. Englewood Cliffs, N.J., Prentice-Hall, 1980, 382 pp., $15.95; $7.95 (paper).*

Diagnostic and Statistical Manual of Mental Disorders, 3rd ed., *by the American Psychiatric Association. Washington, D.C., American Psychiatric Association, 1980, 481 pp., $25.00; $20.00 (paper).*

How to Expand your S.I.Q. (Social Intelligence Quotient), *by Dane Archer, Ph.D.; photographs by Arlene Burney and Rick Bender. New York, N.Y., M. Evans and Co., 1980, 164 pp., $9.95; $5.95 (paper).*

Anyone Can Have a Happy Child: The Simple Secret of Positive Parenting, *by Jacob Azerrad, Ph.D. New York, N.Y., M. Evans and Co., 1980, 179 pp., $8.95.*

Women Discover Orgasm: A Therapist's Guide to a New Treatment Approach, *by Lonnie Barbach. New York, N.Y., Free Press (Macmillan Publishing Co.), 1980, 229 pp., $20.00.*

Introduction to Clinical Psychology, *by Alan S. Bellack and Michel Hersen. New York, N.Y., Oxford University Press, 1980, 385 pp., $16.95.*

Psychoanalytic Explorations of Technique, *edited by Harold P. Blum, M.D. New York, N.Y., International Universities Press, 1980, 455 pp., no price listed.*

Loss: Sadness and Depression. Attachment and Loss, Vol. III, *by John Bowlby. New York, N.Y., Basic Books, 1980, 462 pp., $22.50.*

Progress in the Functional Psychoses, *edited by Robert Cancro, M.D., Lester E. Shapiro, M.D., and Martin Kesselman, M.D. New York, N.Y., SP Medical & Scientific Books (Spectrum Publications), 1979, 187 pp., $20.00.*

Sexual Anxiety: A Study of Male Impotence, *by Eric Carlton. Totowa, N.J., Barnes & Noble, 1980, 193 pp., $22.50.*

Behavioral Complexities, *by Rémy Chauvin and Bernadette Muckensturm-Chauvin; translated by Joyce Diamanti. New York, N.Y., International Universities Press, 1977, 257 pp., no price listed.*

The Annual for Psychoanalysis, Vol. VII, *by the Chicago Institute for Psychoanalysis. New York, N.Y., International Universities Press, 1979, 410 pp., no price listed.*

Urban Community Care for the Developmentally Disabled, *edited by Herbert J. Cohen, M.D., David Kligler, M.D., and Judith A. Eisler, M.S. Springfield, Ill., Charles C Thomas, 1980, 336 pp., $25.50.*

Survivors, Victims, and Perpetrators: Essays on the Nazi Holocaust, *edited by Joel E. Dimsdale, M.D. Washington, D.C., Hemisphere Publishing Corp., 1980, 456 pp., $19.50.*

Mental Illness in the United States: Epidemiological Estimates, *by Bruce P. Dohrenwend, Barbara Snell Dohrenwend, Madelyn Schwartz Gould, Bruce Link, Richard Neugebauer, and Robin Wunsch-Hitzig. New York, N.Y., Praeger, 1980, 157 pp., no price listed.*

Terminal Care: Friendship Contracts with Dying Cancer Patients, *by Loma Feigenberg, M.D. New York, N.Y., Brunner/Mazel, 1980, 272 pp., $19.50.*

Annual Review of Behavior Therapy and Practice, 1979: Commentary and Appraisal, with Selected Papers, *by Cyril M. Franks, Ph.D., and G. Terence Wilson, Ph.D. New York, N.Y., Brunner/Mazel, 1980, 760 pp., $27.50.*

Artful Scribbles: The Significance of Children's Drawings, *by Howard Gardner. New York, N.Y., Basic Books, 1980, 274 pp., $15.00.*

Psychotherapy: An Eclectic Approach, *by Sol. L. Garfield. New York, N.Y., John Wiley & Sons, 1980, 304 pp., $19.95.*

Alcoholism and Affective Disorders: Clinical, Genetic, and Biochemical Studies, *edited by Donald W. Goodwin, M.D., and Carlton K. Erickson, Ph.D. New York, N.Y., SP Medical & Scientific Books (Spectrum Publications), 1979, 294 pp., $25.00.*

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Psychosynthesis: Vital Therapy, *by Max L. Hutt. Oceanside, N.Y., Dabor Science Publications, 1977, 225 pp., $15.00.*

Freud and His Self-Analysis: Downstate Psychoanalytic Institute Twenty-Fifth Anniversary Series, Vol. 1, *edited by Mark Kanzer, M.D., and Jules Glenn, M.D. New York, N.Y., Jason Aronson, 1979, 301 pp., $25.00.*

On Sexuality: Psychoanalytic Observations, *edited by Toksoz B. Karasu and Charles W. Socarides. New York, N.Y., International Universities Press, 1979, 397 pp., no price listed.*

Introduction to Design and Analysis: A Student's Handbook, *by Geoffrey Keppel and William H. Saufley, Jr. San Francisco, Calif., W.H. Freeman & Co., 1980, 481 pp., $25.00; $15.00 (paper).*

Alienation in Perversions, *by M. Masud Khan. New York, N.Y., International Universities Press, 1979, 238 pp., no price listed.*

Directing Health-Care Volunteers, *by Nancy Kieffer. Oradell, N.J., Medical Economics Co., 1980, 245 pp., $12.95.*

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Surviving the Breakup: How Children and Parents Cope with Divorce, *by Judith S. Wallerstein and Joan Berlin Kelly. New York, N.Y., Basic Books, 1980, 336 pp., $18.50.*

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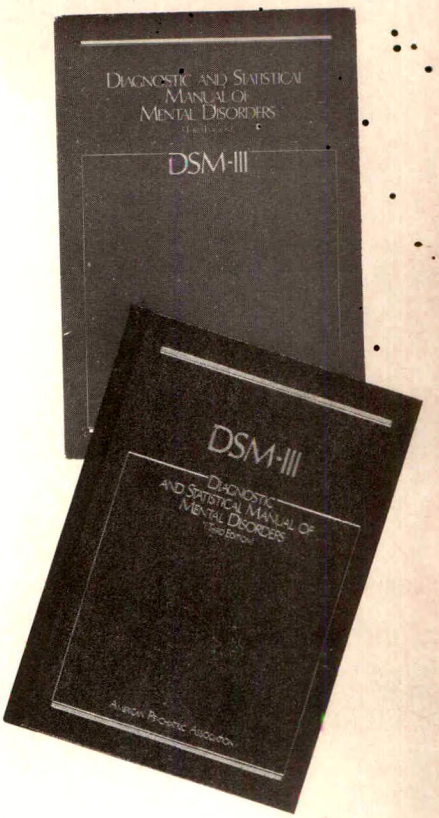
The Use of Massage in Facilitating Holistic Health, *by Robert Henley Woody, Ph.D., Sc.D. Springfield, Ill., Charles C Thomas, 1980, 120 pp., $11.75.*

Mental Retardation and Developmental Disabilities: An Annual Review, *edited by Joseph Wortis, M.D. New York, N.Y., Brunner/Mazel, 1980, 240 pp., no price listed.*

The Development of Self-Regulation Through Private Speech, *edited by Gail Zivin. New York, N.Y., John Wiley & Sons, 1979, 360 pp., no price listed.*

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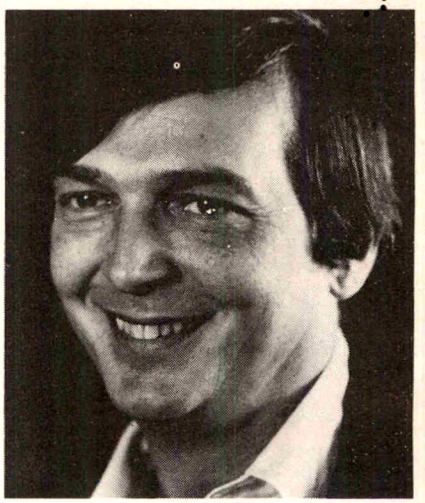
"Cardiopulmonary resuscitation training was one valuable asset that day. I got caught in some heavy machinery at the plant and blacked out.

"They told me later it took six guys to free me. I wasn't breathing. My heart stopped. One of the guys, Don Guarino, said, 'I gotta try anyway.'

"Thanks to his CPR training, he revived me – saved my life.

"Since that happened I realize that no employee is immune from heart attacks or accidents, and a few CPR-trained employees – maybe one for every 50 people – can make the difference between life and death."

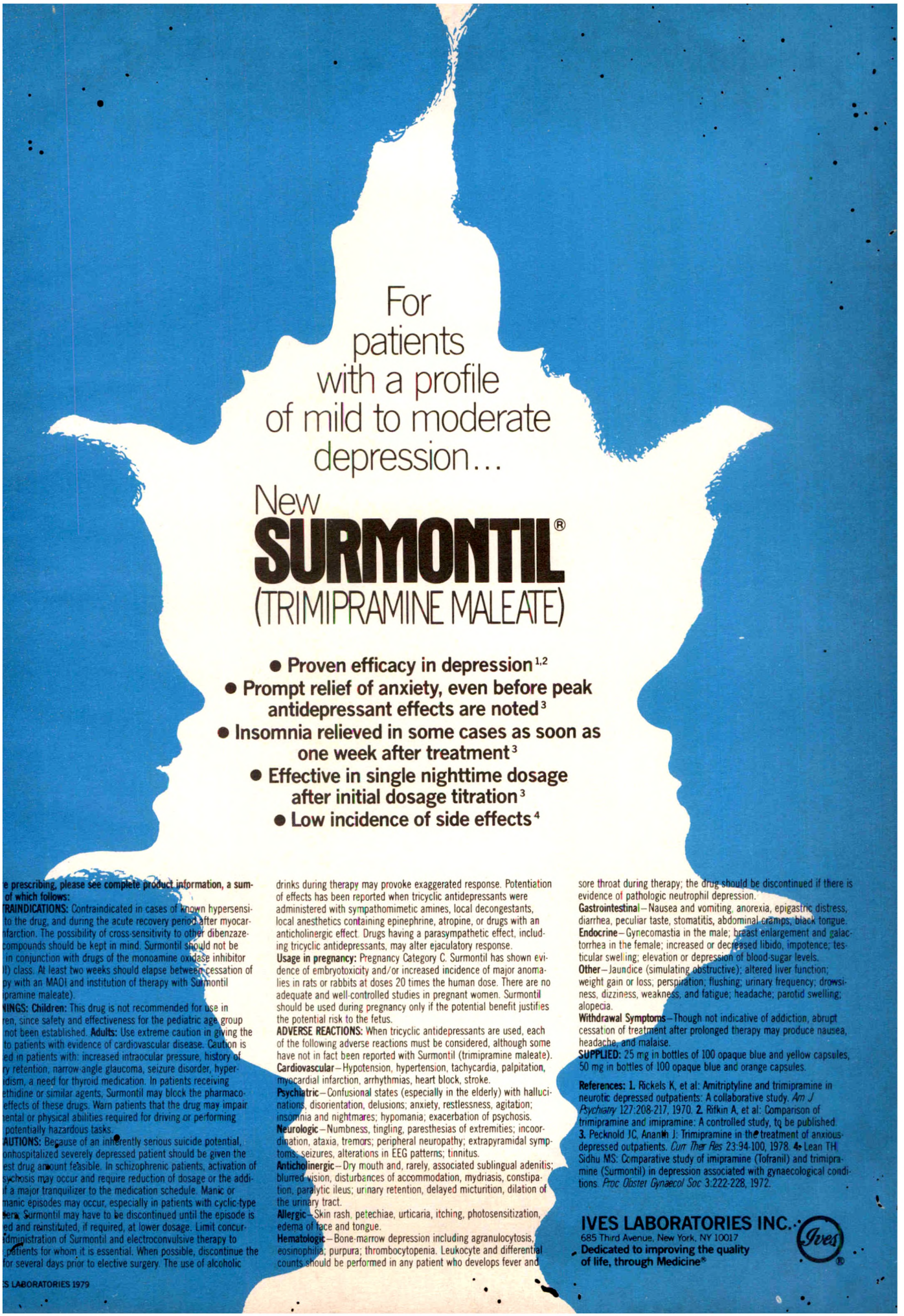
Call your Red Cross Chapter and find out about CPR training for your plant or office. CPR training is a valuable asset for any business.





It takes steady, trained hands to perform CPR.

American Red Cross



e prescribing, please see complete product information, a sum- of which follows:

TRAINDICATIONS: Contraindicated in cases of known hypersensi- to the drug, and during the acute recovery period after myocar- nfarction. The possibility of cross-sensitivity to other dibenzaze- compounds should be kept in mind. Surmontil should not be in conjunction with drugs of the monoamine oxidase inhibitor I) class. At least two weeks should elapse between cessation of py with an MAOI and institution of therapy with Surmontil ramine maleate).

NINGS: Children: This drug is not recommended for use in ren, since safety and effectiveness for the pediatric age group not been established. **Adults:** Use extreme caution in giving the to patients with evidence of cardiovascular disease. Caution is ed in patients with: increased intraocular pressure, history of ry retention, narrow-angle glaucoma, seizure disorder, hyper- idism, a need for thyroid medication. In patients receiving ethidine or similar agents, Surmontil may block the pharmaco- effects of these drugs. Warn patients that the drug may impair ental or physical abilities required for driving or performing potentially hazardous tasks.

AUTIONS: Because of an inherently serious suicide potential, onhospitalized severely depressed patient should be given the est drug amount feasible. In schizophrenic patients, activation of sychosis may occur and require reduction of dosage or the addi- f a major tranquilizer to the medication schedule. Manic or manic episodes may occur, especially in patients with cyclic-type hera. Surmontil may have to be discontinued until the episode is ed and reinstituted, if required, at lower dosage. Limit concur- dministration of Surmontil and electroconvulsive therapy to patients for whom it is essential. When possible, discontinue the for several days prior to elective surgery. The use of alcoholic drinks during therapy may provoke exaggerated response. Potentiation of effects has been reported when tricyclic antidepressants were administered with sympathomimetic amines, local decongestants, local anesthetics containing epinephrine, atropine, or drugs with an anticholinergic effect. Drugs having a parasympathetic effect, includ- ing tricyclic antidepressants, may alter ejaculatory response.

Usage in pregnancy: Pregnancy Category C. Surmontil has shown evi- dence of embryotoxicity and/or increased incidence of major anoma- lies in rats or rabbits at doses 20 times the human dose. There are no adequate and well-controlled studies in pregnant women. Surmontil should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

ADVERSE REACTIONS: When tricyclic antidepressants are used, each of the following adverse reactions must be considered, although some have not in fact been reported with Surmontil (trimipramine maleate).

Cardiovascular—Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric—Confusional states (especially in the elderly) with halluci- nations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurologic—Numbness, tingling, paresthesias of extremities; incoor- dination, ataxia, tremors; peripheral neuropathy; extrapyramidal symp- toms; seizures, alterations in EEG patterns; tinnitus.

Anticholinergic—Dry mouth and, rarely, associated sublingual adenitis; blurred vision, disturbances of accommodation, mydriasis, constipa- tion, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic—Skin rash, petechiae, urticaria, itching, photosensitization, edema of face and tongue.

Hematologic—Bone-marrow depression including agranulocytosis, eosinophilia; purpura; thrombocytopenia. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathologic neutrophil depression.

Gastrointestinal—Nausea and vomiting, anorexia, epigastric distress, diarrhea, peculiar taste, stomatitis, abdominal cramps, black tongue.

Endocrine—Gynecomastia in the male; breast enlargement and galac- torrhea in the female; increased or decreased libido, impotence; tes- ticular swelling; elevation or depression of blood-sugar levels.

Other—Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency; drowsi- ness, dizziness, weakness, and fatigue; headache; parotid swelling; alopecia.

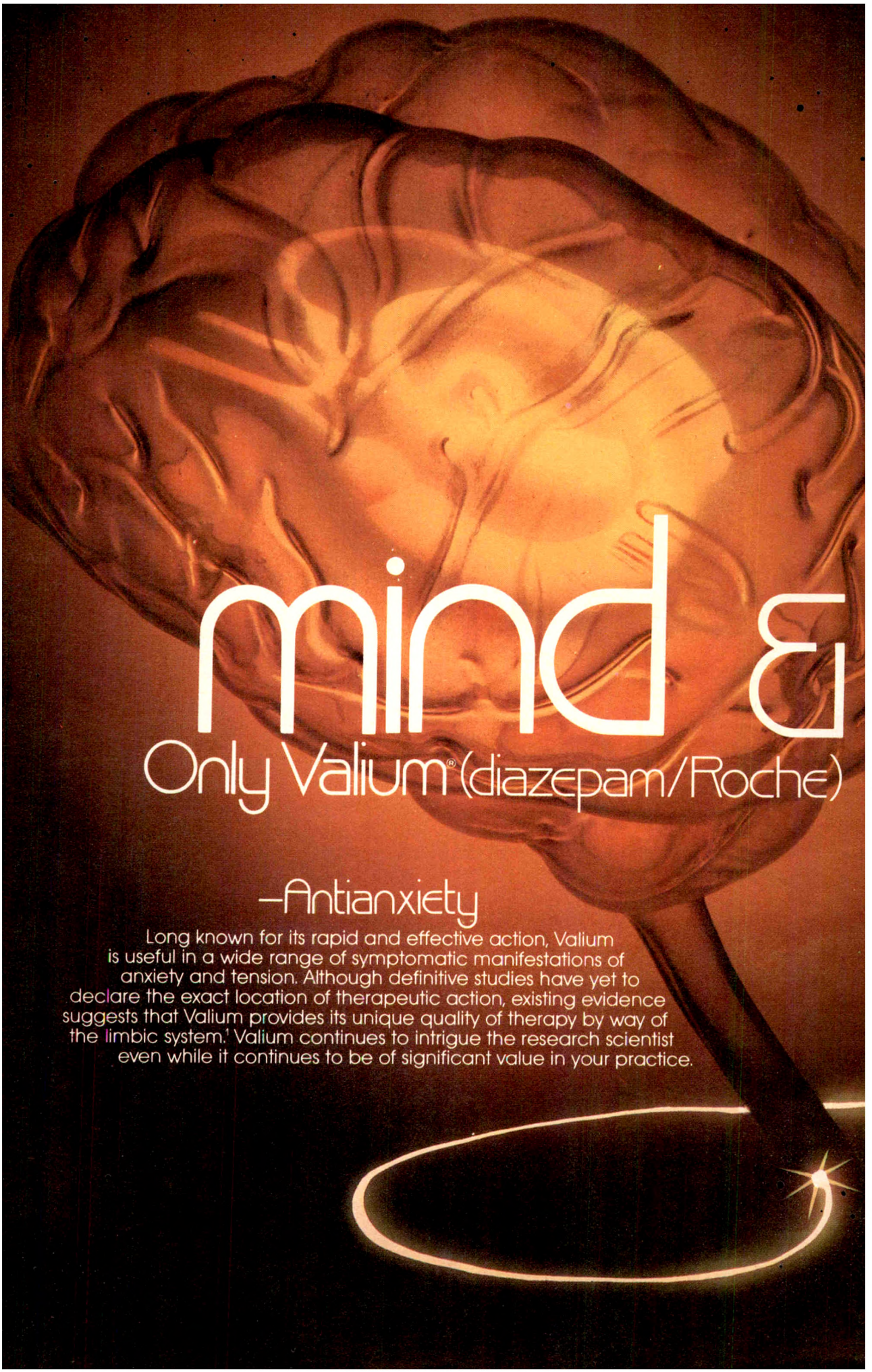
Withdrawal Symptoms—Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache, and malaise.

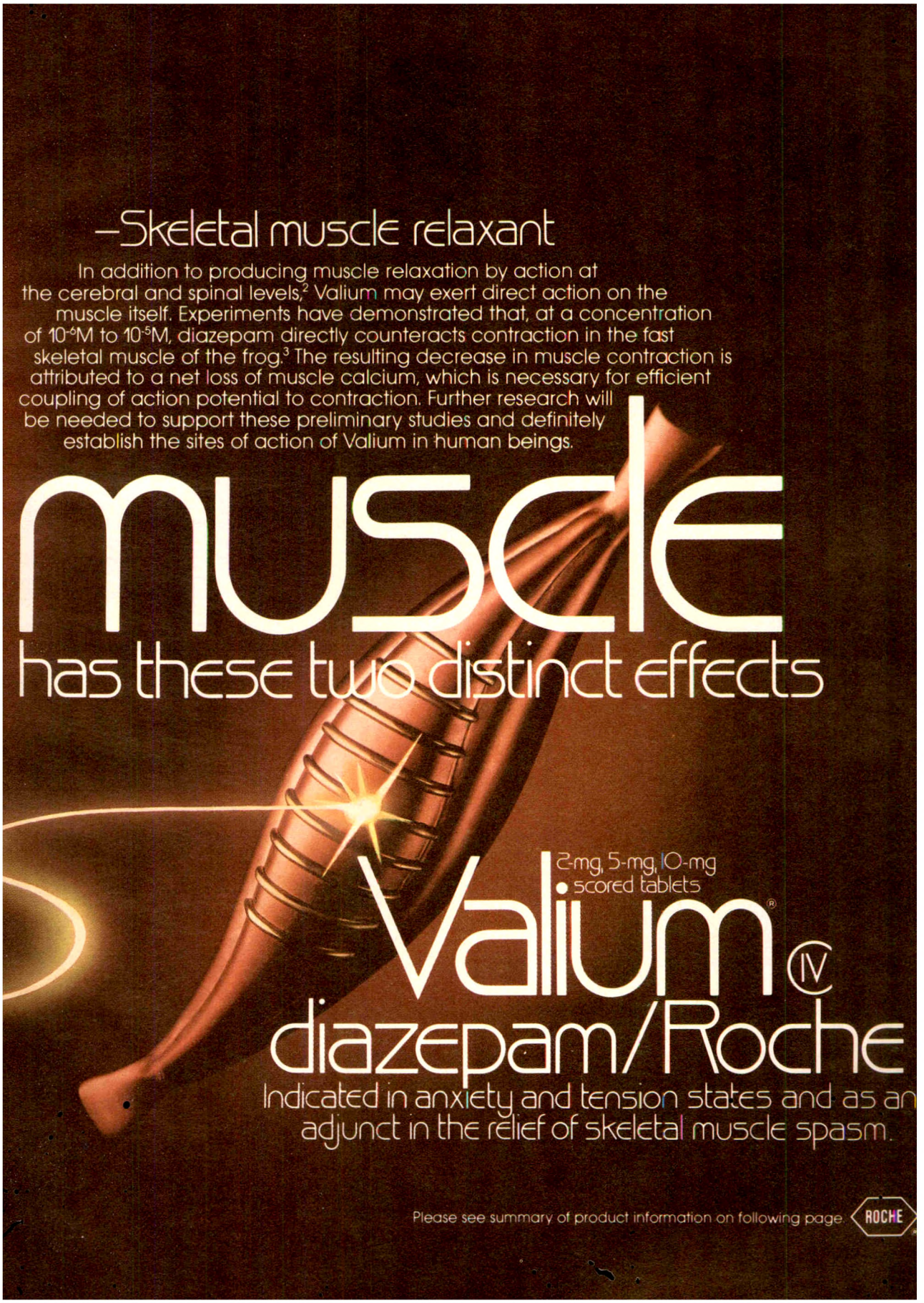
SUPPLIED: 25 mg in bottles of 100 opaque blue and yellow capsules, 50 mg in bottles of 100 opaque blue and orange capsules.

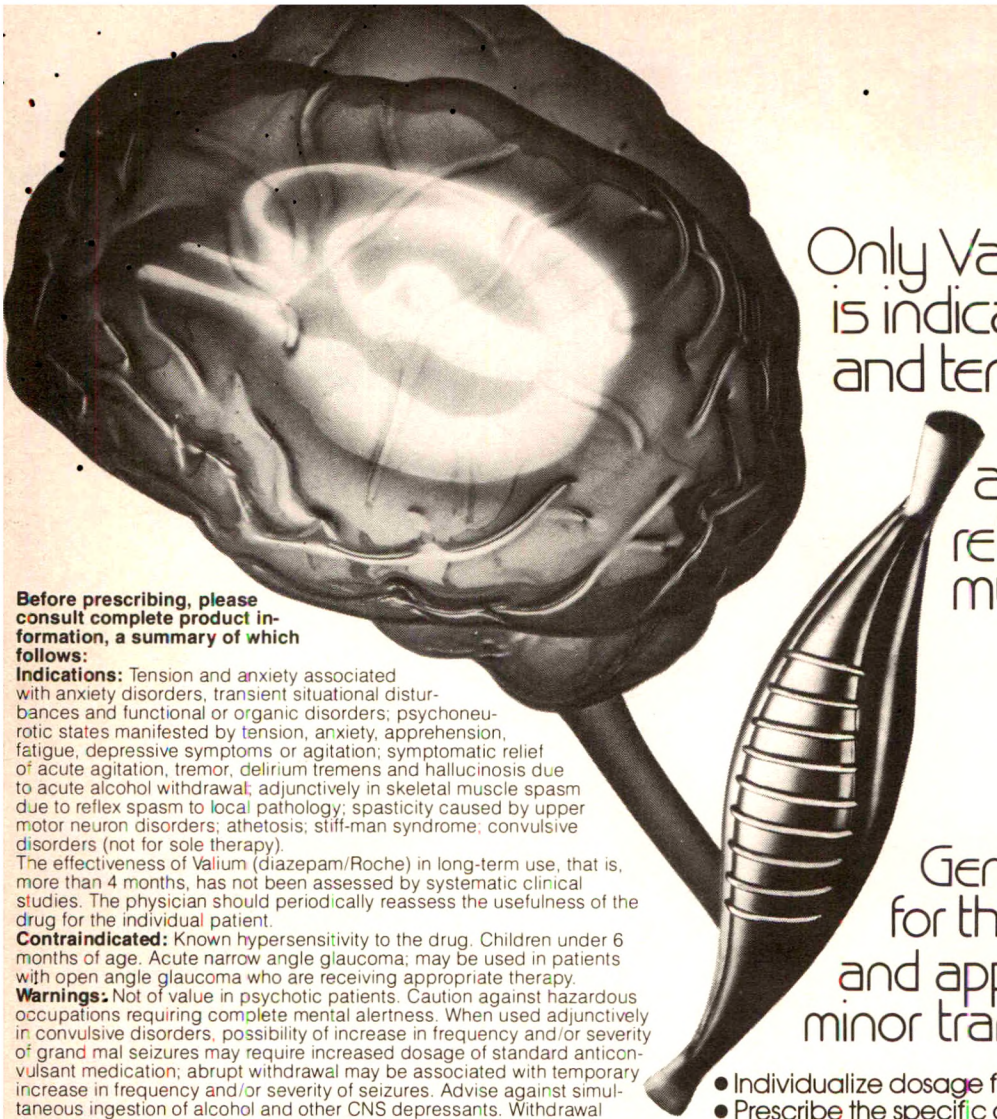
References: 1. Rickels K, et al: Amitriptyline and trimipramine in neurotic depressed outpatients: A collaborative study. *Am J Psychiatry* 127:208-217, 1970. **2.** Rifkin A, et al: Comparison of trimipramine and imipramine: A controlled study, to be published. **3.** Pecknold JC, Ananth J: Trimipramine in the treatment of anxious-depressed outpatients. *Curr Ther Res* 23:94-100, 1978. **4.** Lean TH, Sidhu MS: Comparative study of imipramine (Tofranil) and trimipra- mine (Surmontil) in depression associated with gynaecological condi- tions. *Proc Obstet Gynaecol Soc* 3:222-228, 1972.

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mind &
Only Valium® (diazepam/Roche)
–Antianxiety
Long known for its rapid and effective action, Valium is useful in a wide range of symptomatic manifestations of anxiety and tension. Although definitive studies have yet to declare the exact location of therapeutic action, existing evidence suggests that Valium provides its unique quality of therapy by way of the limbic system.' Valium continues to intrigue the research scientist even while it continues to be of significant value in your practice.


–Skeletal muscle relaxant
In addition to producing muscle relaxation by action at the cerebral and spinal levels,[2] Valium may exert direct action on the muscle itself. Experiments have demonstrated that, at a concentration of 10^{-6}M to 10^{-5}M, diazepam directly counteracts contraction in the fast skeletal muscle of the frog.[3] The resulting decrease in muscle contraction is attributed to a net loss of muscle calcium, which is necessary for efficient coupling of action potential to contraction. Further research will be needed to support these preliminary studies and definitely establish the sites of action of Valium in human beings.
muscle
has these two distinct effects
2-mg, 5-mg, 10-mg
• scored tablets
Valium®
diazepam/Roche
Indicated in anxiety and tension states and as an adjunct in the relief of skeletal muscle spasm.
Please see summary of product information on following page.
ROCHE



Only Valium® (diazepam/Roche) is indicated in anxiety and tension states and as an adjunct in the relief of skeletal muscle spasm

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety associated with anxiety disorders, transient situational disturbances and functional or organic disorders; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).
The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

Supplied: Valium® Tablets, 2 mg, 5 mg and 10 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available in trays of 10.

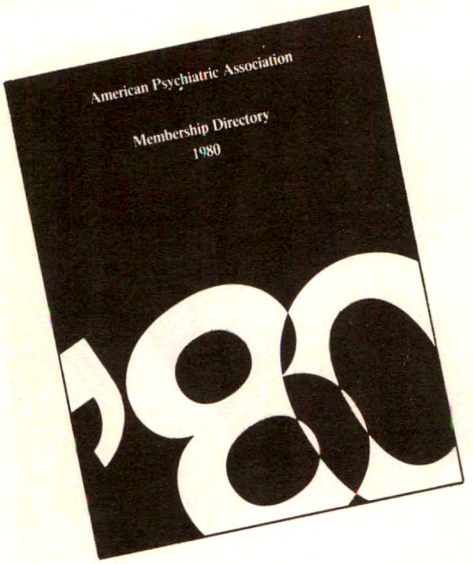
General guidelines for the prescribing and appropriate use of minor tranquilizers

- Individualize dosage for maximal beneficial effect.
- Prescribe the specific quantity needed until the next checkup period, schedule frequent, periodic reexaminations to monitor results of therapy.
- Establish treatment goals and gradually discontinue medication when these have been met.
- Avoid prescribing for individuals who appear dependency-prone or whose histories indicate the potential for misuse of psychoactive substances, including alcohol.
- Caution patients against engaging in hazardous occupations requiring complete mental alertness such as operating machinery or driving.
- Advise patients against the ingestion of alcoholic beverages while undergoing therapy with minor tranquilizers.
- Counsel patients to follow label directions, keep medication out of children's reach, and dispose of unused or old medication.
- Caution patients against giving medication to others.
- Avoid abrupt cessation of extended therapy by tapering dosage before discontinuing medication.

1. Brazier MAB, Crandall PH, Walsh GO: *Exp Neurol* 51:241-258, Apr 1976
2. Naftchi NE, Lowman EW: The effect of diazepam on presynaptic inhibition. Scientific exhibit at the meeting of the American Academy of Family Physicians, Las Vegas, Nev, Oct 10-13, 1977
3. deGroof RC, Bianchi CP: *Fed Proc* 37:787, 1978

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The latest listing of APA members (as of January 31, 1980) has just been published. Unlike previous editions, the 1980 Membership Directory, largely at the urging of President Dr. Donald G. Langsley, contains telephone numbers as well as names, addresses, membership class and membership status. A new format provides easy access to the alphabetical and geographical listings.

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Help cure cancer write now.

Thanks to your help, the tide is beginning to turn. The past few years have brought new discoveries in chemotherapy.

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Slow-Release Lithium Carbonate 300 mg.

Before prescribing, see complete prescribing information in Rowell literature. The following is a brief summary.

WARNING
Lithium toxicity is closely related to serum lithium levels, and can occur at doses close to therapeutic levels. Facilities for prompt and accurate serum lithium determinations should be available before initiating therapy.

Indications: Treatment of manic episodes of manic-depressive illness. Maintenance therapy prevents or diminishes the intensity of subsequent episodes in manic-depressive patients with a history of mania.

Warnings: Lithium should generally not be given to patients with significant renal or cardiovascular disease, severe debilitation or dehydration, sodium depletion, or to patients receiving diuretics.

Lithium therapy has been reported in some cases to be associated with morphologic changes in the kidneys.

Caution patient and family to watch for diarrhea, vomiting, tremor, mild ataxia, drowsiness, or muscular weakness as signs of lithium toxicity, and to discontinue therapy and contact a physician should they occur. Patients receiving combined therapy with lithium and an antipsychotic should be monitored closely for early evidence of neurologic toxicity and treatment discontinued promptly if such signs appear. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery).

Lithium should not be used in pregnancy, especially during the first trimester, unless potential benefits outweigh possible hazards.

Not recommended for children under 12.

Precautions: Lithium tolerance is greater during the acute manic phase and decreases when manic symptoms subside.

Lithium therapy may lead to sodium depletion. Normal diet (including salt) and adequate fluid intake (2500-3000 ml) must be maintained, at least during initial stabilization period. Protracted sweating or diarrhea can decrease tolerance; in such cases, administer supplemental fluid and salt.

Each tablet contains 40 mg of sodium chloride, equivalent to 15.7 mg of sodium.

Sweating, diarrhea, and concomitant infection with elevated temperatures may require temporary reduction or cessation of dosage.

Adverse Reactions: Mild to moderate toxic reactions may occur at serum lithium levels from 1.5 to 2.5 mEq/L, and moderate to severe reactions at levels from 2.0 to 2.5 mEq/L. Fine hand tremor, polyuria, and mild thirst may occur during initial therapy and persist. Transient and mild nausea and general discomfort also appear during initial therapy. These effects usually subside with continued treatment or temporary reduction or cessation of dosage. If persistent, discontinue dosage.

Diarrhea, vomiting, drowsiness, muscular weakness, and lack of coordination may be early signs of toxicity and may occur at levels below 2.0 mEq/L. At higher levels, ataxia, giddiness, tinnitus, blurred vision, and a large output of dilute urine may be seen. Serum levels above 3.0 mEq/L may produce a complex clinical picture, involving multiple organs and systems. Serum levels should not exceed 2.0 mEq/L during acute phase.

The following reactions appear to be related to serum lithium levels, including levels within the therapeutic range: Neuromuscular—tremor, muscle hyperirritability (fasciculations, twitching, clonic movements of whole limbs), ataxia, choreo-athetotic movements, hyperactive deep tendon reflex; Central Nervous System—blackout spells, epileptiform seizures, slurred speech, dizziness, vertigo, incontinence of urine or feces, somnolence, psychomotor retardation, restlessness, confusion, stupor, coma; Cardiovascular—cardiac arrhythmia, hypotension, peripheral circulatory collapse; Gastrointestinal—anorexia, nausea, vomiting, diarrhea; Genitourinary—albuminuria, oliguria, polyuria, glycosuria; Dermatologic—drying and thinning of hair, alopecia, anesthesia of skin, chronic folliculitis, exacerbation of psoriasis, xerosis cutis; Autonomic—blurred vision, dry mouth; Thyroid Abnormalities—euthyroid goiter and/or hypothyroidism (including myxedema) with lower T_3 and T_4. I^{131} uptake may be elevated; EEG Changes—diffuse slowing, widening of the frequency spectrum, potentiation and disorganization of background rhythm; EKG Changes—reversible flattening, isoelectricity or inversion of T-waves; Miscellaneous—fatigue, lethargy, transient scotomata, dehydration, weight loss, tendency to sleep.

Reactions unrelated to dosage include: transient EEG and EKG changes, leukocytosis, headache, diffuse nontoxic goiter with or without hypothyroidism, transient hyperglycemia, generalized pruritus with or without rash, cutaneous ulcers, albuminuria, worsening of organic brain syndromes, excessive weight gain, edematous swelling of ankles or wrists, thirst or polyuria, sometimes resembling diabetes insipidus, and metallic taste. A single case of a syndrome resembling Raynaud's has been reported.

Dosage and Administration: *Acute Mania*—900 mg b.i.d. or 600 mg t.i.d. (1800 mg per day) usually will provide serum lithium levels ranging between 1.0 and 1.5 mEq/L. Serum levels should be determined twice per week until serum level and clinical condition have been stabilized.

Long-Term Control—900 mg to 1200 mg per day in two or three divided doses usually will maintain serum lithium levels at 0.6 to 1.2 mEq/L. Serum lithium levels should be monitored at least every two months.

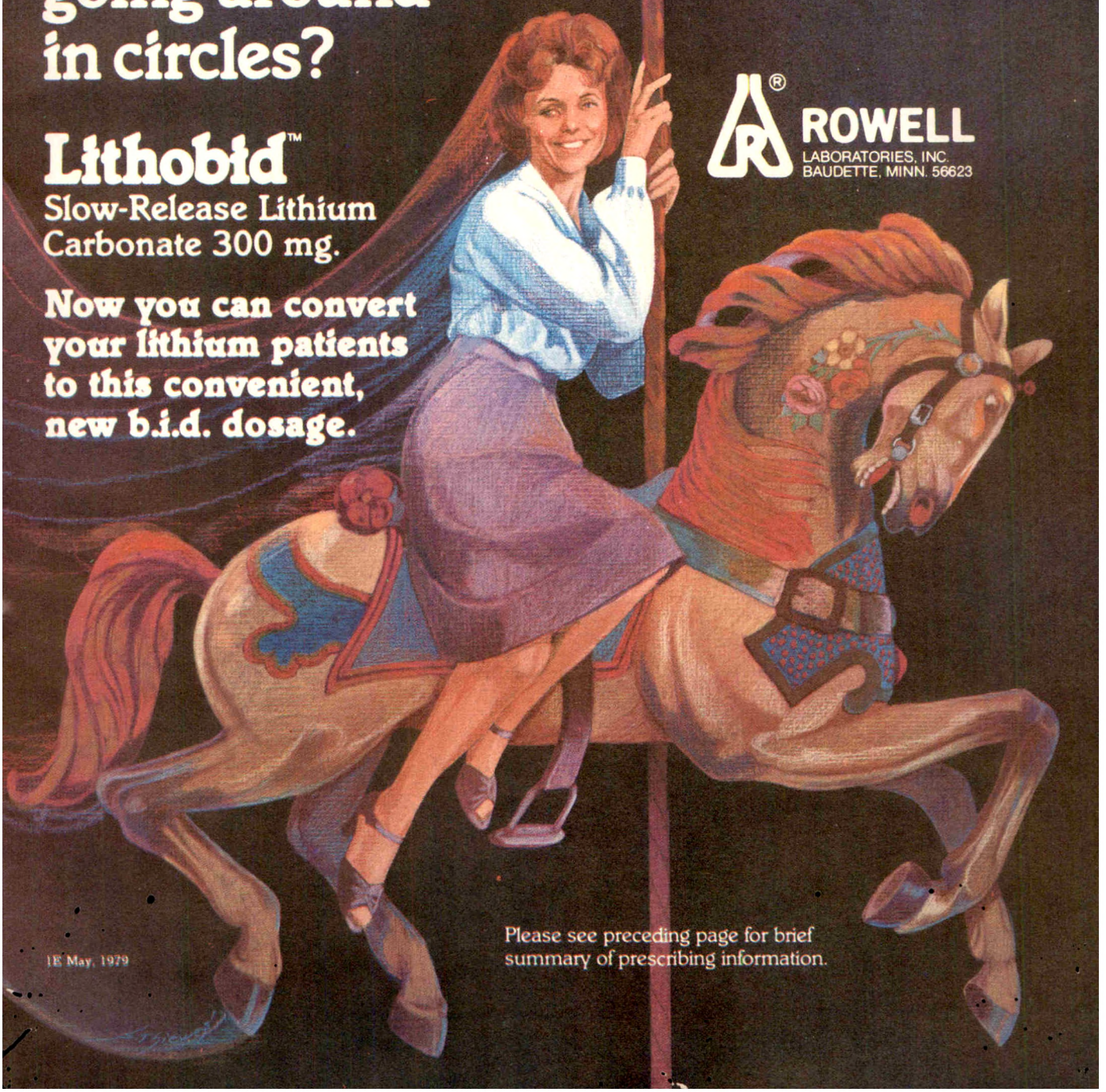
How Supplied: 300 mg peach-colored tablets, imprinted "ROWELL 7514" in red, are supplied in bottles of 100 and 1000.

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Do lithium dosage schedules keep your patients going around in circles?

Slow-Release Lithobid enables twice-a-day dosing as a practical route to better patient compliance. Patients on conventional lithium dosage forms can be converted to new Lithobid at the same daily dose, divided b.i.d. All patients on long-term lithium maintenance therapy will appreciate the convenience of this reduced dosage frequency.

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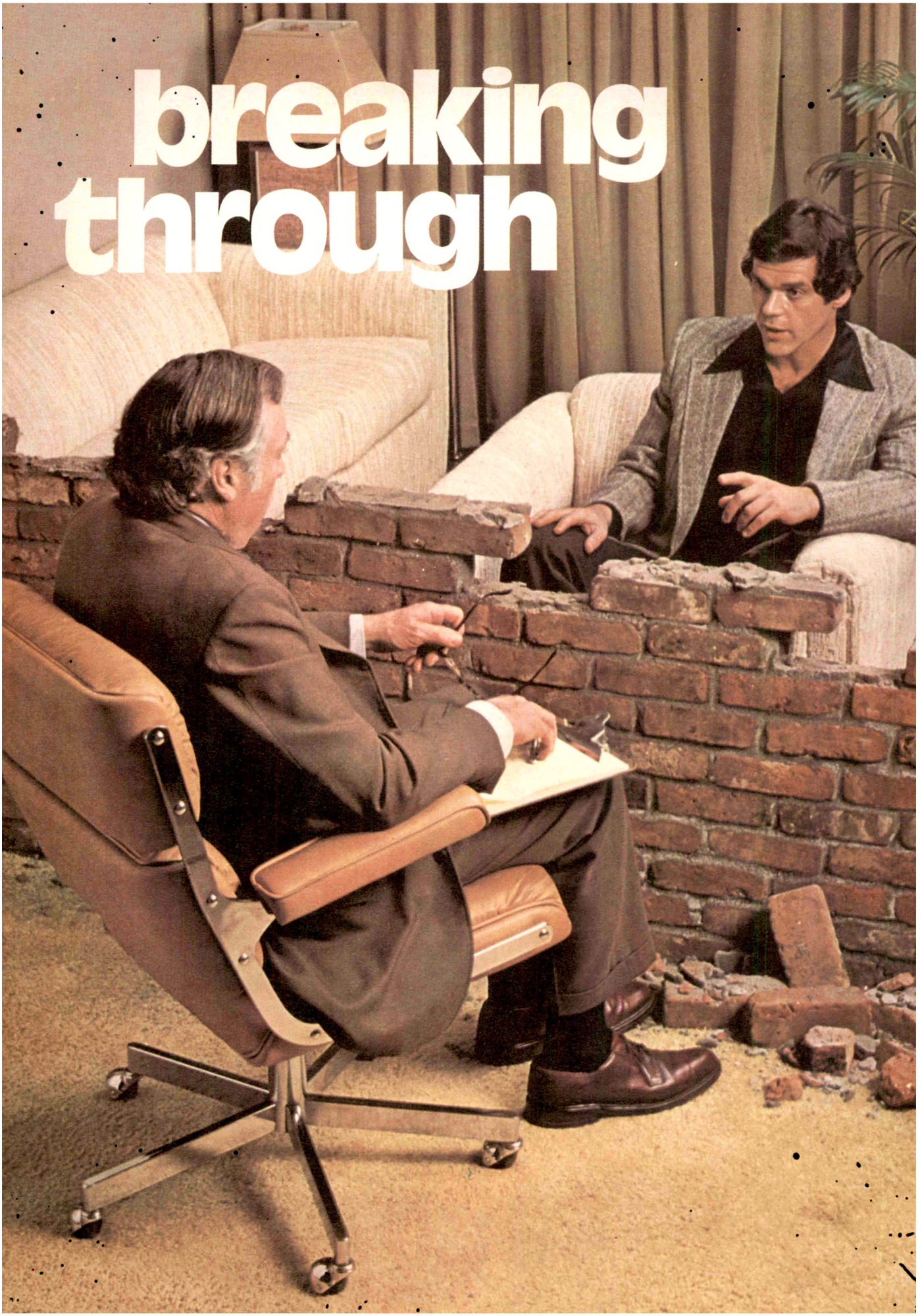
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Slow-Release Lithium Carbonate 300 mg.

Now you can convert your lithium patients to this convenient, new b.i.d. dosage.

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Please see preceding page for brief summary of prescribing information.

1E May, 1979


breaking
through

In marked agitation with depression symptomatic relief may make the patient more accessible and responsive.

Rapid relief of marked agitation in many patients
The tranquilizer component alleviates symptoms of agitation and anxiety within a few days, without apparent dulling of mental acuity. Hypnotic effects from the tranquilizer component appear to be minimal, particularly in patients permitted to remain active. However, TRIAVIL may impair mental and/or physical abilities required for performance of hazardous tasks.

Highly effective antidepressant action
The antidepressant component relieves symptoms of depression such as poor concentration and feelings of hopelessness as well as early morning awakening; adequate relief of symptoms may take a few weeks or even longer.

Helps break barriers to psychotherapy
As symptoms are relieved, many patients often communicate more effectively, become more cooperative, and are able to return to normal daily activities.

More prescribing convenience
There are now *five* tablet strengths of TRIAVIL for ease of dosage adjustment. For many patients with agitation and depression, you can now initiate therapy with one TRIAVIL® 4-50, containing 4 mg perphenazine and 50 mg amitriptyline HCl, b.i.d. The regimen is simple, economical, and may well enhance patient compliance.

Treatment with TRIAVIL—a balanced view:
TRIAVIL is contraindicated in CNS depression from drugs, in the presence of evidence of bone marrow depression, and in patients hypersensitive to phenothiazines or amitriptyline. It should not be used during the acute recovery phase following myocardial infarction or in patients who have received an MAOI within two weeks. Patients with cardiovascular disorders should be watched closely. Not recommended in children or during pregnancy. TRIAVIL may impair mental and/or physical abilities required for performance of hazardous tasks and may enhance the response to alcohol. Antiemetic effect may obscure toxicity due to overdosage of other drugs or mask other disorders. The possibility of suicide in depressed patients remains until significant remission occurs. Such patients should not have access to large quantities of the drug. Hospitalize as soon as possible any patient suspected of having taken an overdose.



For marked agitation with depression

dual-action **Triavil**®

containing perphenazine and amitriptyline HCl

MSD
MERCK SHARP & DOHME

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Please see following page for a brief summary of prescribing information.

More dosage strengths than any other formulation containing a tranquilizer and an antidepressant

dual-action TRIAVIL®

containing perphenazine and amitriptyline HCl

Available:
TRIAVIL® 2-25: Each tablet contains
2 mg perphenazine and 25 mg amitriptyline HCl.
TRIAVIL® 2-10: Each tablet contains
2 mg perphenazine and 10 mg amitriptyline HCl.
TRIAVIL® 4-50: Each tablet contains
4 mg perphenazine and 50 mg amitriptyline HCl.
TRIAVIL® 4-25: Each tablet contains
4 mg perphenazine and 25 mg amitriptyline HCl.
TRIAVIL® 4-10: Each tablet contains
4 mg perphenazine and 10 mg amitriptyline HCl.

CONTRAINDICATIONS: Central nervous system depression from drugs (barbiturates, alcohol, narcotics, analgesics, antihistamines); evidence of bone marrow depression; known hypersensitivity to phenothiazines or amitriptyline. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred from such combinations. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with TRIAVIL. Therapy should then be initiated cautiously with gradual increase in dosage until optimum response is achieved. Not recommended for use during acute recovery phase following myocardial infarction.

WARNINGS: TRIAVIL should not be given concomitantly with guanethidine or similarly acting compounds since TRIAVIL may block the antihypertensive action of such compounds. Use cautiously in patients with history of urinary retention, angle-closure glaucoma, increased intraocular pressure, or convulsive disorders. Dosage of anticonvulsive agents may have to be increased. In patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely. Tricyclic antidepressants, including amitriptyline HCl, have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time, particularly in high doses. Myocardial infarction and stroke have been reported with tricyclic antidepressant drugs. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Not recommended in children or during pregnancy.

PRECAUTIONS: Suicide is a possibility in depressed patients and may remain until significant remission occurs. Such patients should not have access to large quantities of this drug.

Perphenazine: Should not be used indiscriminately. Use with caution in patients who have previously exhibited severe adverse reactions to other phenothiazines. Likelihood of some untoward actions is greater with high doses. Closely supervise with any dosage. The antiemetic effect of perphenazine may obscure signs of toxicity due to overdosage of other drugs or make more difficult the diagnosis of disorders such as brain tumor or intestinal obstruction. A significant, not otherwise explained, rise in body temperature may suggest individual intolerance to perphenazine, in which case discontinue.

If hypotension develops, epinephrine should not be employed, as its action is blocked and partially reversed by perphenazine. Phenothiazines may potentiate the action of central nervous system depressants (opiates, analgesics, antihistamines, barbiturates, alcohol) and atropine. In concurrent therapy with any of these, TRIAVIL should be given in reduced dosage. May also potentiate the action of heat and phosphorous insecticides. There is sufficient experimental evidence to conclude that chronic administration of antipsychotic drugs which increase prolactin secretion has the potential to induce mammary neoplasms in rodents under the appropriate conditions. There are recognized differences in the physiological role of prolactin between rodents and humans. Since there are, at present, no adequate epidemiological studies, the relevance to human mammary cancer risk from prolonged exposure to perphenazine and other antipsychotic drugs is not known.

Amitriptyline: In manic-depressive psychosis, depressed patients may experience a shift toward the manic phase if they are treated with an antidepressant. Patients with paranoid symptomatology may have an exaggeration of such symptoms. The tranquilizing effect of TRIAVIL seems to reduce the likelihood of this effect. When amitriptyline HCl is given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. Paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs.

Caution is advised if patients receive large doses of ethchlorvynol concurrently. Transient delirium has been reported in patients who were treated with 1 g of ethchlorvynol and 75-150 mg of amitriptyline HCl.

Amitriptyline HCl may enhance the response to alcohol and the effects of barbiturates and other CNS depressants.

Concurrent administration of amitriptyline HCl and electroshock therapy may increase the hazards associated with such therapy. Such treatment should be limited to patients for whom it is essential. Discontinue several days before elective surgery if possible. Elevation and lowering of blood sugar levels have both been reported. Use with caution in patients with impaired liver function.

ADVERSE REACTIONS: Similar to those reported with either constituent alone.
Perphenazine: Extrapyramidal symptoms (opisthotonus, oculogyric crisis, hyperreflexia, dystonia, akathisia, acute dyskinesia, ataxia, parkinsonism) have been reported and can usually be controlled by the concomitant use of effective antiparkinsonian drugs and/or by reduction in dosage, but sometimes persist after discontinuation of the phenothiazine.

Tardive dyskinesia may appear in some patients on long-term therapy or may occur after drug therapy with phenothiazines and related agents has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw. Involuntary movements of the extremities sometimes occur. There is no known treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms. It is advised that all antipsychotic agents be discontinued if the above symptoms appear. If treatment is reinstituted, or dosage of the particular drug increased, or another drug substituted, the syndrome may be masked. Fine vermicular movements of the tongue may be an early sign of the syndrome. The full-blown syndrome may not develop if medication is stopped when lingual vermiculation appears.

Other side effects are skin disorders (photosensitivity, itching, erythema, urticaria, eczema, up to exfoliative dermatitis); other allergic reactions (asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions); peripheral edema; reversed epinephrine effect; hyperglycemia; endocrine disturbances (lactation, galactorrhea, gynecomastia, disturbances of menstrual cycle); altered cerebrospinal fluid proteins; paradoxical excitement; hypertension, hypotension, tachycardia, and ECG abnormalities (quinidine-like effect); reactivation of psychotic processes; catatonic-like states; autonomic reactions, such as dry mouth or salivation, headache, anorexia, nausea, vomiting, constipation, obstipation, urinary frequency or incontinence, blurred vision, nasal congestion, and a change in pulse rate; other adverse reactions reported with various phenothiazine compounds, but not with perphenazine, include grand mal convulsions, cerebral edema, polyphagia, pigmentary retinopathy, photophobia, skin pigmentation, and failure of ejaculation.

The phenothiazine compounds have produced blood dyscrasias (pancytopenia, thrombocytopenic purpura, leukopenia, agranulocytosis, eosinophilia); and liver damage (jaundice, biliary stasis).

Pigmentation of the cornea and lens has been reported to occur after long-term administration of some phenothiazines. Although it has not been reported in patients receiving TRIAVIL, the possibility that it might occur should be considered.

Hypnotic effects, lassitude, muscle weakness, and mild insomnia have also been reported.

Amitriptyline: Note: Listing includes a few reactions not reported for this drug, but which have occurred with other pharmacologically similar tricyclic antidepressant drugs and must be considered when amitriptyline is administered. *Cardiovascular:* Hypotension; hypertension; tachycardia; palpitation; myocardial infarction; arrhythmias; heart block; stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth; blurred vision; disturbance of accommodation; increased intraocular pressure; constipation; paralytic ileus; urinary retention; dilatation of urinary tract. *Allergic:* Skin rash; urticaria; photosensitization; edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis; leukopenia; eosinophilia; purpura; thrombocytopenia. *Gastrointestinal:* Nausea; epigastric distress; vomiting; anorexia; stomatitis; peculiar taste; diarrhea; parotid swelling; black tongue. Rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido; elevated or lowered blood sugar levels. *Other:* Dizziness, weakness; fatigue; headache; weight gain or loss; increased perspiration; urinary frequency; mydriasis; drowsiness; alopecia. *Withdrawal Symptoms:* Abrupt cessation after prolonged administration may produce nausea, headache, and malaise. These are not indicative of addiction.

OVERDOSAGE: All patients suspected of having taken an overdosage should be admitted to a hospital as soon as possible. Treatment is symptomatic and supportive. However, the intravenous administration of 1–3 mg of physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage of physostigmine should be repeated as required particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine. On this basis, in severe overdosage with perphenazine-amitriptyline combinations, symptomatic treatment of central anticholinergic effects with physostigmine salicylate should be considered.

J8TR32 (DC6613215)

For more detailed information, consult your MSD Representative or see full Prescribing Information. Merck Sharp & Dohme, Division of Merck & Co., INC., West Point, Pa. 19486.

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PREVENTING MENTAL ILLNESS

Efforts and Attitudes

A new publication of The Joint Information Service of the American Psychiatric Association and the Mental Health Association

Raymond M. Glasscote; *Eleanor Kohn, Allan Beigel, Merrill F. Raber, Nancy Roeske, Bonnie A. Cox, James R. Raybin, Bernard L. Bloom*

There has long been interest in preventing mental illness, but no great emphasis on financing necessary research and pilot programs. Dramatic discoveries of nutritional, toxic, and infective causes of certain psychoses were made long ago and led to their prevention, but prevention of schizophrenia, mania, sociopathy, and biological depression, among other major disorders, requires further knowledge.

In recent years faith has grown that programs supporting those experiencing heavy stress and privation will provide benefits increasing the likelihood that they will remain functional, productive, and well. This volume provides a balanced, fairminded, and realistic explication of present possibilities and reasonable expectations. It explores a variety of viewpoints and describes in significant detail a variety of prevention-motivated programs for a variety of vulnerable people and provides such outcome information as is presently available.

1980. 138 pp., Casebound.

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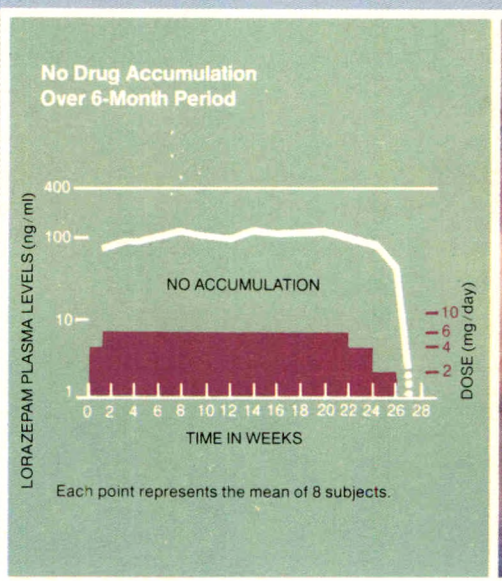
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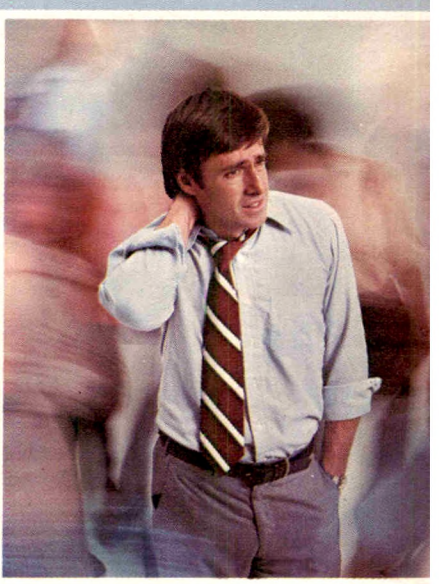
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Pharmacokinetics

Ativan (lorazepam) is eliminated more rapidly than other benzodiazepines; generates no clinically active metabolites. The half-life of free lorazepam is about 12 hours; steady-state blood levels are attained in 2-3 days. (Comparable data for diazepam: 20-50 hours and at least 7-10 days.) Ativan shows no evidence of accumulation, even when given in high doses for as long as 6 months. (The pharmacokinetic profile of a drug can define such characteristics as absorption, distribution, metabolism and elimination but cannot, at present, be directly related to its therapeutic effectiveness.)

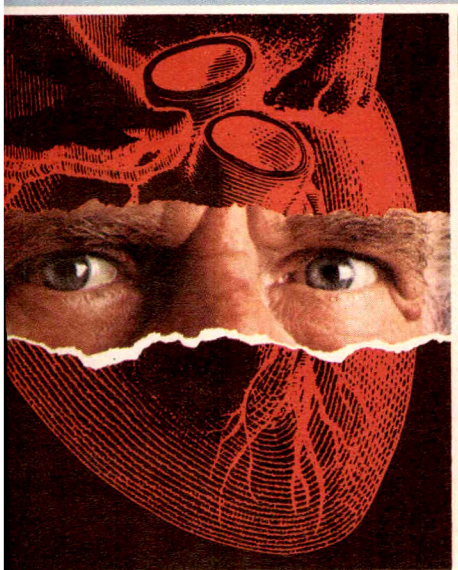
Anxiety neuroses

In many nationwide studies involving thousands of patients with anxiety neuroses, Ativan (lorazepam) consistently provided significant relief of anxiety, tension, agitation and irritability as measured by standard Hamilton, Global (physician rated) and 35-Item (patient self-rated) scales.

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They're all about the same, aren't they? Until recently, that seemed to sum up the prevalent attitude about the benzodiazepines. After all, they all work similarly and all are effective anxiolytics. With seven of these compounds now available, however, differences have begun to emerge. Foremost among them: different metabolic pathways, which distinguish the relatively short-acting Ativan (lorazepam) from long-acting diazepam, clorazepate and chlordiazepoxide. At the same time, your choice can be made with confidence in the clinical record of Ativan, which confirms its value in anxiety neuroses and in significant anxiety associated with functional or organic disorders, as well as in older patients.

Consider the record of Ativan:



Anxiety in cardiovascular disorders

Ativan (lorazepam) has been specifically evaluated and found effective in seven common protocol, double-blind studies involving 423 patients (211 on Ativan) whose anxiety was related to organic and functional cardiovascular disorders. The cardiovascular component has not, of course, been shown to be significantly benefited by such therapy.



Anxiety in gastrointestinal disorders

So far, nine common protocol, double-blind studies of Ativan have focused on anxious patients with functional or organic gastrointestinal complaints (457 patients, 234 on Ativan). Ativan was clearly effective in reducing the anxiety of these patients. The gastrointestinal component has not, of course, been shown to be significantly benefited by such therapy.



Anxiety in the aging patient

Because its simple metabolism is not readily impaired with advancing age, and accumulation is not likely to present a problem, Ativan is a good choice for older patients. Those who have trouble swallowing solid medication appreciate the small Ativan tablet, which is tasteless and disintegrates within seconds in water or fruit juice.

Ativan® (lorazepam) for Anxiety

See important information on following page.

Brief Summary of Prescribing Information

Indications and Usage: Symptomatic relief of anxiety, tension, agitation, irritability and insomnia associated with anxiety neuroses and transient situational disturbances; anxiety associated with depressive symptoms and as a treatment of symptoms of anxiety if such symptoms are a significant feature of functional or organic disorders, particularly gastrointestinal or cardiovascular.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients on lorazepam not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation.

Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions.

Observe usual precautions with impaired renal or hepatic function.

Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component.

Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day. No effect dose was 1.25mg/kg/day (approximately 6 times the maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatric patients requires caution and frequent monitoring for symptoms of upper G.I. disease.

Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug.

In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Overdosage: In management of overdosage with any drug, bear in mind that multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring of vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

Ativan®(IV) for Anxiety
(lorazepam)

Dosage: **Individualize for maximum beneficial effects. Increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; insomnia due to anxiety or transient situational stress, 2-4mg h.s.**

How Supplied: **0.5, 1.0 and 2.0mg tablets.**

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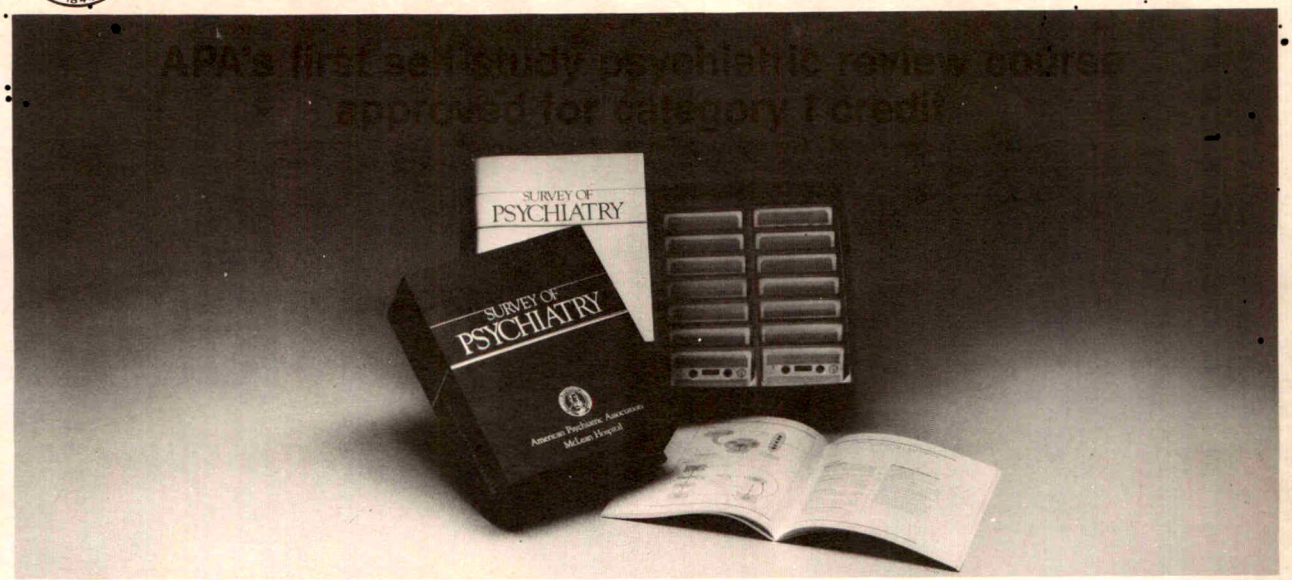
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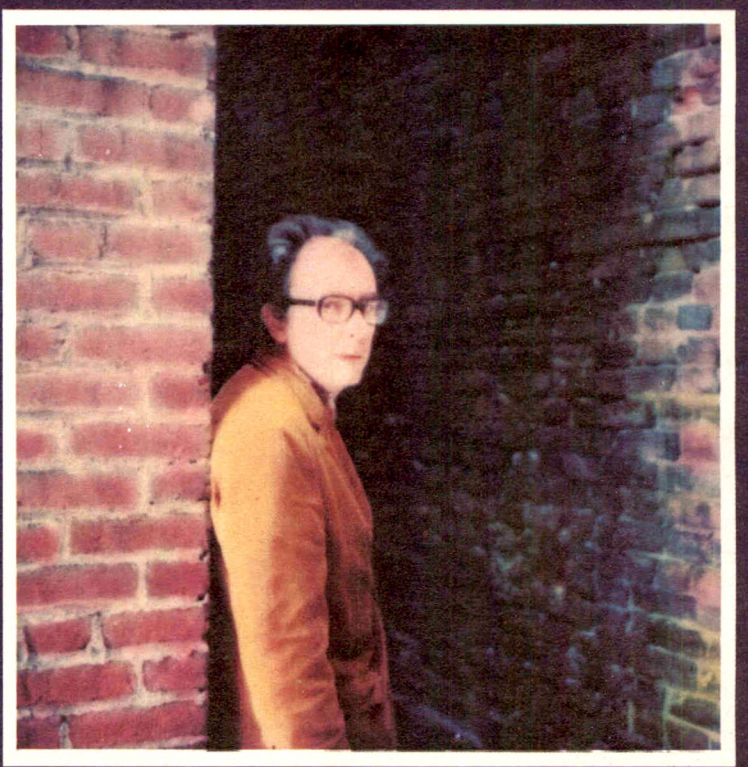
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In fact, no other antipsychotic agent has demonstrated significantly greater overall effectiveness and significantly fewer adverse effects than 'Stelazine'.

'Stelazine'. A first choice for therapy.

Before prescribing, see complete prescribing information in SK&F literature or PDR. The following is a brief summary.

Indications

Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Effective: For the management of the manifestations of psychotic disorders.

Possibly effective: To control excessive anxiety, tension and agitation as seen in neuroses or associated with somatic conditions.

'Stelazine' has not been shown effective in the management of behavioral complications in patients with mental retardation.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose or greatly depressed states due to C.N.S. depressants; blood dyscrasias; bone marrow depression; liver damage.

Warnings: Generally avoid using in patients hypersensitive (e.g., blood dyscrasias, jaundice) to any phenothiazine. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery), especially during the first few days' therapy.

Additive effect is possible with other C.N.S. depressants, including alcohol.

Use in pregnancy only when necessary for patient's welfare.

Precautions: Use cautiously in angina. Avoid high doses and parenteral administration when cardiovascular system is impaired. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Prolonged administration of high doses may result in cumulative effects with severe C.N.S. or vasomotor symptoms. If retinal changes occur, discontinue drug. Agranulocytosis, thrombocytopenia, pancytopenia, anemia, cholestatic jaundice, liver damage have been reported.

Patients on long-term therapy, especially high doses, should be evaluated periodically for possible adjustment or discontinuance of drug therapy.

Adverse Reactions: Drowsiness, dizziness, skin reactions, rash, dry mouth, insomnia, amenorrhea, fatigue, muscular weakness, anorexia, lactation, blurred vision. Neuromuscular (extrapyramidal) reactions: motor restlessness, dystonias, pseudoparkinsonism, persistent tardive dyskinesia.

Other adverse reactions reported with Stelazine (trifluoperazine HCl, SK&F) or other phenothiazines: Some adverse effects are more frequent or intense in specific disorders (e.g., mitral insufficiency or pheochromocytoma).

Grand mal convulsions; altered cerebrospinal fluid proteins; cerebral edema; prolongation and intensification of the action of C.N.S. depressants, atropine, heat, and organophosphorus insecticides; nasal congestion, headache, nausea, constipation, obstipation, adynamic ileus, inhibition of ejaculation; reactivation of psychotic processes, catatonic-like states; hypotension (sometimes fatal); cardiac arrest; leukopenia, eosinophilia, pancytopenia, agranulocytosis, thrombocytopenic purpura; jaundice, biliary stasis; menstrual irregularities, galactorrhea, gynecomastia, false positive pregnancy tests; photosensitivity, itching, erythema, urticaria, eczema up to exfoliative dermatitis; asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions, peripheral edema; reversed epinephrine effect; hyperpyrexia; a systemic lupus erythematosus-like syndrome; pigmentary retinopathy; with prolonged administration of substantial doses, skin pigmentation, epithelial keratopathy, and lenticular and corneal deposits. EKG changes have been reported, but relationship to myocardial damage is not confirmed. Discontinue long-term, high-dose therapy gradually. NOTE: Sudden death in patients taking phenothiazines (apparently due to cardiac arrest or asphyxia due to failure of cough reflex) has been reported, but no causal relationship has been established.

Supplied: Tablets, 1 mg., 2 mg., 5 mg. and 10 mg., in bottles of 100; in Single Unit Packages of 100 (intended for institutional use only); Injection, 2 mg./ml.; and Concentrate (intended for institutional use only) 10 mg./ml.

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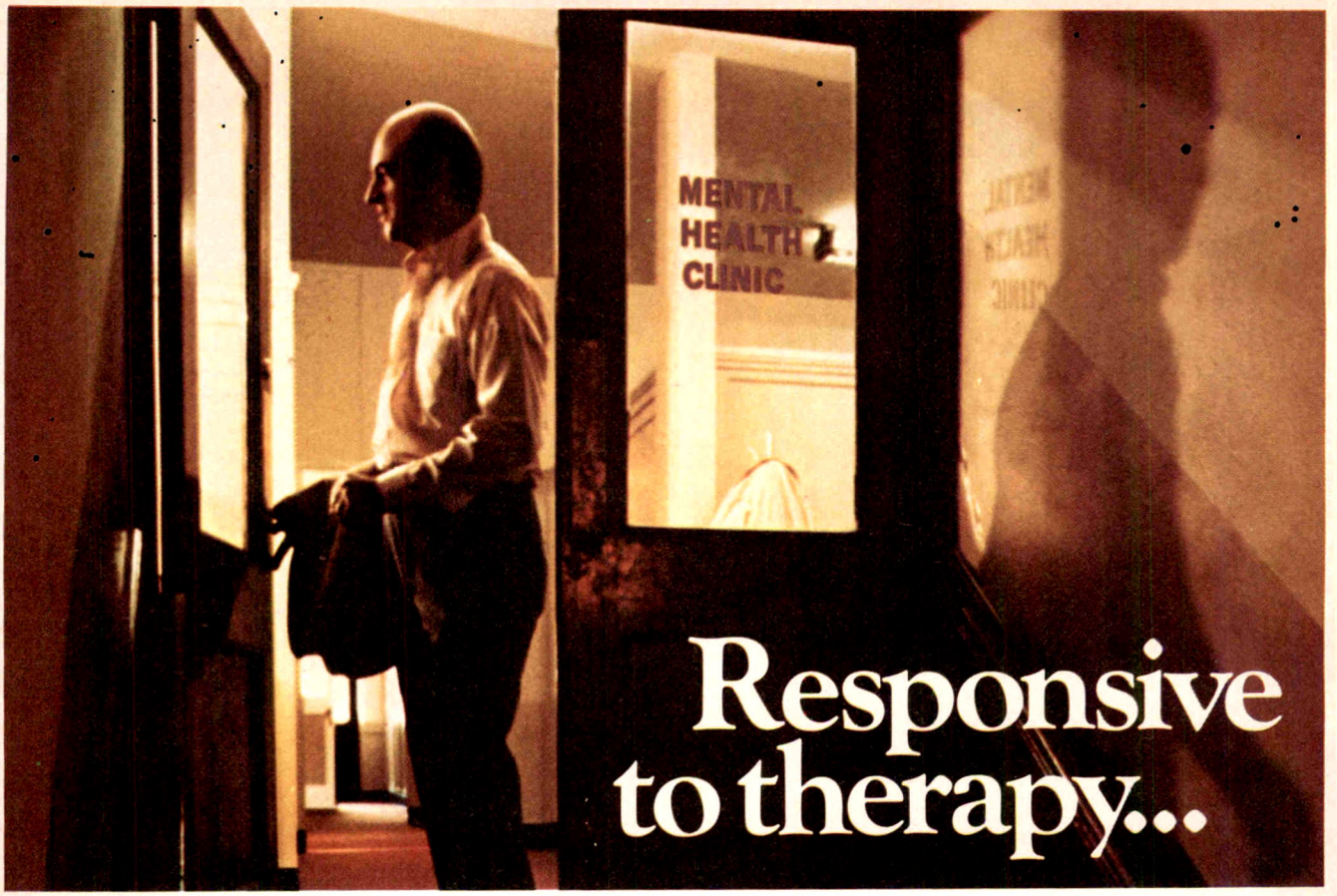
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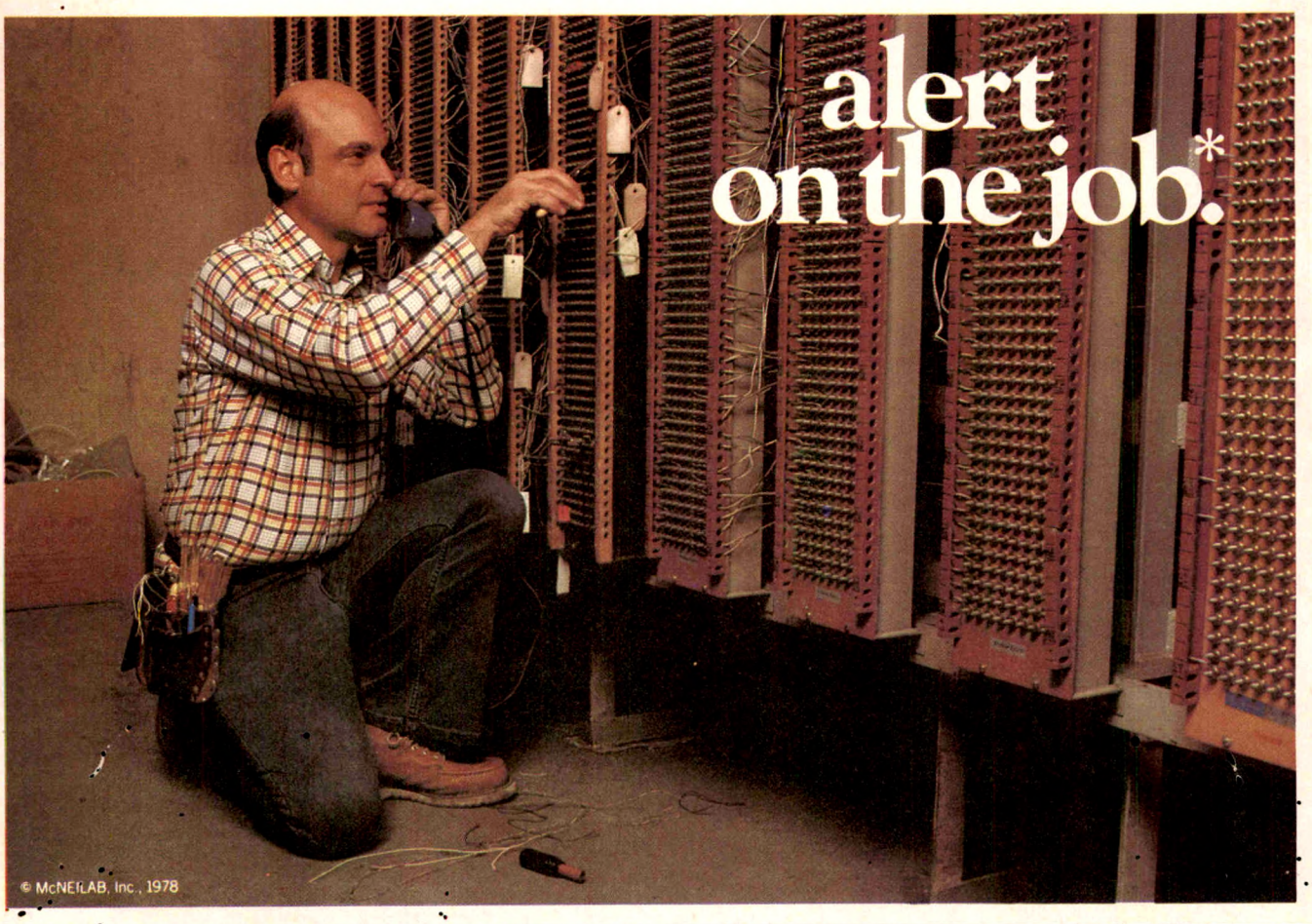
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Highly effective in a wide range of acute and chronic psychotic disorders[1-7]

Permits aggressive titration to effective dosage levels for optimal control[1,4]

Usually leaves patients alert and responsive...
easier to reach with supportive measures[1-3]

*Not an actual case history, this situation illustrates the action of HALDOL haloperidol as reported in various clinical studies (available on request).

Minimal risk of hypotension, oversedation, or troublesome anticholinergic effects[3-5,7-9]
Transient hypotension occurs rarely; severe orthostatic hypotension has not been reported. Although some instances of drowsiness have been reported, marked sedation is rare.

Common side effects easily controlled[1,3-7]
Although extrapyramidal symptoms (EPS) have been reported frequently, they are usually dose-related and readily controlled with dose adjustment or antiparkinson drugs. EPS often diminish spontaneously with continued use of HALDOL haloperidol.

References: 1. Man, P.L.: Dis. Nerv. Syst. *34*:113 (Feb.) 1973. **2.** Sugerman, A.A., et al.: Am. J. Psychiatry *129*:1190 (June) 1964. **3.** Ayd, F.J., Jr.: Med. Sci. *18*:55 (Oct.) 1967. **4.** Howard, J.S.: Dis. Nerv. Syst. *35*:458 (Oct.) 1974. **5.** Abuzzahab, F.S., Sr.: Psychosomatics *11*:188 (May-June) 1970. **6.** Darling, H.F.: Dis. Nerv. Syst. *34*:364 (Oct.-Nov.) 1973. **7.** Gerle, B.: Clin. Trials J. *3*:380 (Feb.) 1966. **8.** Snyder, S.H., et al: Science *184*:1243 (June 21) 1974. **9.** Stimmel, C.L.: Dis. Nerv. Syst. *34*:219 (Apr.) 1976.

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Summary of Prescribing Information

Contraindications: Severe depression, coma, CNS depression due to centrally-acting depressants, Parkinson's disease, hypersensitivity to the drug.

Warnings: ***Usage in Pregnancy:*** Safe use in pregnancy or in women likely to become pregnant has not been established; use only if benefit clearly justifies potential hazards. Infants should not be nursed during drug treatment.

Usage in Children: Safety and effectiveness not established; not recommended in pediatric age group.

Combined Use With Lithium: Patients receiving lithium plus haloperidol should be monitored closely for early evidence of neurological toxicity.

General: Bronchopneumonia, sometimes fatal, has followed use of major tranquilizers, including haloperidol. Prompt remedial therapy should be instituted if dehydration, hemoconcentration or reduced pulmonary ventilation occurs, especially in the elderly. Decreased serum cholesterol and/or cutaneous and ocular changes have been reported with chemically-related drugs, although not with haloperidol. Mental and/or physical abilities required for hazardous tasks or driving may be impaired. Alcohol should be avoided due to possible additive effects and hypotension.

Precautions: Administer cautiously to patients: (1) with severe cardiovascular disorders, due to the possibility of transient hypotension and/or precipitation of anginal pain (if a vasopressor is required, epinephrine should not be used since HALDOL haloperidol may block its vasopressor activity and paradoxical further lowering of blood pressure may occur); (2) receiving anticonvulsant medication since HALDOL haloperidol may lower the convulsive threshold; (3) with known allergies or a history of allergic reactions to drugs; (4) receiving anticoagulants. Concomitant antiparkinson medication, if required, may have to be continued after HALDOL haloperidol is discontinued because of different excretion rates; if both are discontinued simultaneously, extrapyramidal symptoms may occur. Intraocular pressure may increase when anticholinergic drugs, including antiparkinson drugs, are administered concomitantly with HALDOL haloperidol. When HALDOL haloperidol is used for mania in cyclic disorders, there may be a rapid mood swing to depression. Severe neurotoxicity may occur in patients with thyrotoxicosis receiving antipsychotic medication, including HALDOL haloperidol.

Adverse Reactions: ***CNS Effects:*** *Extrapyramidal Reactions:* Neuromuscular (extrapyramidal) reactions have been reported frequently, often during the first few days of treatment. Generally they involved Parkinson-like symptoms which were usually mild to moderately severe and usually reversible. Other types of neuromuscular reactions (motor restlessness, dystonia, akathisia, hyperreflexia, opisthotonos, oculogyric crises) have been reported far less frequently, but were often more severe. Severe extrapyramidal reactions have been reported at relatively low doses. Generally, extrapyramidal symptoms are dose-related since they occur at relatively high doses and disappear or become less severe when the dose is reduced. Antiparkinson drugs may be required. Persistent extrapyramidal reactions have been reported and the drug may have to be discontinued in such cases.

Withdrawal Emergent Neurological Signs: Abrupt discontinuation of short-term antipsychotic therapy is generally uneventful. However, some patients on maintenance treatment experience transient dyskinetic signs after abrupt withdrawal. In certain cases these are indistinguishable from "Persistent Tardive Dyskinesia" except for duration. It is unknown whether gradual withdrawal will reduce the occurrence of these signs, but until further evidence is available haloperidol should be gradually withdrawn.

Persistent Tardive Dyskinesia: Although rarely reported with HALDOL haloperidol, tardive dyskinesia may appear during or after long-term therapy. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and sometimes appear irreversible; there is no known effective treatment and all antipsychotic agents should be discontinued. The syndrome may be masked by reinstitution of drug, increasing dosage, or switching to a different antipsychotic agent.

Other CNS Effects: Insomnia, restlessness, anxiety, euphoria, agitation, drowsiness, depression, lethargy, headache, confusion, vertigo, grand mal seizures, and exacerbation of psychotic symptoms.

Cardiovascular Effects: Tachycardia and hypotension. ***Hematologic Effects:*** Reports of mild, usually transient leukopenia and leukocytosis, minimal decreases in red blood cell counts, anemia, or a tendency toward lymphomonocytosis; agranulocytosis rarely reported and only in association with other medication. ***Liver Effects:*** Impaired liver function and/or jaundice reported. ***Dermatologic Reactions:*** Maculopapular and acneiform reactions, isolated cases of photosensitivity, loss of hair. ***Endocrine Disorders:*** Lactation, breast engorgement, mastalgia, menstrual irregularities, gynecomastia, impotence, increased libido, hyperglycemia and hypoglycemia. ***Gastrointestinal Effects:*** Anorexia, constipation, diarrhea, hypersalivation, dyspepsia, nausea and vomiting. ***Autonomic Reactions:*** Dry mouth, blurred vision, urinary retention and diaphoresis. ***Respiratory Effects:*** Laryngospasm, bronchospasm and increased depth of respiration.

The injectable form is intended only for acutely agitated psychotic patients with moderately severe to very severe symptoms.

Caution: Federal law prohibits dispensing without prescription.

Full directions for use should be read before HALDOL haloperidol is administered or prescribed.

HALDOL tablets are manufactured by McNeil Laboratories Co., Dorado, Puerto Rico 00646.

* haloperidol present as the lactate

McNEIL McNeil Laboratories, McNEILAB, Inc.
Fort Washington, PA 19034

THE CHRONIC MENTAL PATIENT

Report of a Conference held in January 1978
Edited by
John A. Talbott, M.D.

with contributions by James T. Barter, M.D., Paul R. Friedman, J.D., Raymond Glasscote, M.A., Trevor D. Glenn, M.D., Norman V. Lourie, M.S.W., W. Walter Menninger, M.D., Arthur T. Meyerson, M.D., Kenneth Minkoff, M.D., Samuel Muszynski, M.S.W., Lucy D. Ozarin, M.D., Gordon L. Paul, Ph.D., Ronald Peterson, Steven S. Sharfstein, M.D., Judith Clark Turner, Jane Bloom Yohalem, J.D.

This 277-page Report of the Conference, sponsored by APA and President Carter's Commission on Mental Health, identifies the chronic mental patient population, spells out where they are and what their needs and rights are. It specifies what programs work and what programs do not work in meeting the needs of these patients. It elaborates on the obstacles to implementing effective programs and the economic issues involved. It delineates the pros and cons of case management and specifies responsibility for coordinating, implementing, and monitoring services to chronic mental patients.

Finally, it proposes a *Call to Action* which opens with this statement: "There is no more urgent concern than the needs of the chronic mentally ill who suffer from severe, persistent, or recurrent mental illnesses with residual social and vocational disabilities. As a result of the deinstitutionalization programs of the past decade and the continuing growth of high risk populations that generate chronically ill, the problems associated with the care of these patients constitute a national crisis."

Since the Conference, the APA Assembly and the Board of Trustees have both approved the "Call to Action" which calls upon the APA to take the lead in undertaking programs to elevate the prestige and value of work with chronic mentally ill patients.

It follows that all APA members should be thoroughly versed in the current problems of this chronic patient population. This is best accomplished by reading this Conference Report now available from APA Publications Services at $11.00 a copy.

Send coupon to: American Psychiatric Association
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Please send me _____ copy(ies) of *The Chronic Mental Patient*
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The APA will assume shipping expenses via regular channels on all prepaid orders, except to countries abroad. If invoicing is necessary, a postage and handling charge will be added. Please allow at least 15–20 days delivery.

HCP

When depressive symptoms come in a cluster of blues...

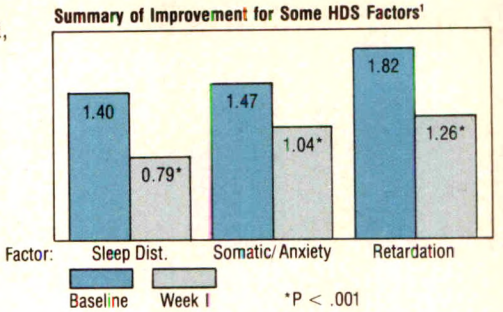


Adapin® (doxepin HCl) helps patients see life in all its colors

Improvements noted at one week

Depressed patients often present with fatigue, anorexia, anxiety, mood swings, loss of interest, *and most frequently, early awakening and insomnia*. In a summation of 9 clinical studies involving 198 patients, significant clinical improvement was noted after the first week of doxepin treatment.[1] However, optimal antidepressant response may not be evident for two to three weeks.

Summary of Improvement for Some HDS Factors[1]



Normalized sleep patterns

In a study of clinically depressed patients with symptoms of insomnia, treatment with doxepin significantly reduced difficulty falling asleep, difficulty staying asleep and frequency of early morning awakening.[2]

Low potential for cardiotoxicity

The incidence of tachycardia and hypotension, which have been observed during treatment with tricyclic antidepressants, has been reported infrequently with doxepin. In 452 patients given doxepin, the incidence of tachycardia was 2.88%; in 495 patients, the incidence of hypotension was 2.62%.[3]

Now available...New 75 mg capsules for h.s. dosage

Brief Summary of Prescribing Information ADAPIN® (doxepin HCl) Capsules
Indications–Relief of symptoms of anxiety and depression.
Contraindications–Glaucoma, tendency toward urinary retention or hypersensitivity to doxepin.
Warnings–Adapin has not been evaluated for safety in pregnancy. No evidence of harm to the animal fetus has been shown in reproductive studies. There are no data concerning secretion in human milk, nor on effect in nursing infants.

Usage in children under 12 years of age is not recommended. MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug, as serious side-effects and death have been reported with the concomitant use of certain drugs and MAO inhibitors.

In patients who may use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage.

Precautions–Drowsiness may occur and patients should be cautioned against driving a motor vehicle or operating hazardous machinery. Since suicide is an inherent risk in depressed patients they should be closely supervised while receiving treatment. Although Adapin has shown effective tranquilizing activity, the possibility of activating or unmasking latent psychotic symptoms should be kept in mind. This product contains FD&C Yellow No. 5 (tartrazine) which may cause allergic-type reactions (including bronchial asthma) in certain susceptible individuals. Although the overall incidence of FD&C Yellow No. 5 (tartrazine) sensitivity in the general population is low, it is frequently seen in patients who also have aspirin hypersensitivity.

Adverse Reactions–Dry mouth, blurred vision and constipation have been reported. Drowsiness has also been observed.

Adverse effects occurring infrequently include extrapyramidal symptoms, gastrointestinal reactions, secretory effects such as sweating, tachycardia and hypotension. Weakness, dizziness, fatigue, weight gain, edema, paresthesias, flushing, chills, tinnitus, photophobia, decreased libido, rash and pruritus may also occur.

Dosage and Administration–In mild to moderate anxiety and/or depression: 25 mg t.i.d. Increase or decrease the dosage according to individual response. Daily dosage, up to 150 mg may be taken at bedtime without loss of effectiveness. Usual optimum daily dosage is 75 mg to 150 mg per day not to exceed 300 mg per day.

Antianxiety effect usually precedes the antidepressant effect by two or three weeks.

How Supplied–Each capsule contains doxepin, as the hydrochloride. 10 mg, 25 mg, 50 mg, 75 mg and 100 mg capsules in bottles of 100 and 1000.

For complete prescribing information please see package insert or PDR.

References:

1. Barranco SF, Thrash ML, Hackett E, et al: Early onset of response to doxepin treatment. *J Clin Psychiatry* 40:265-269, 1979 (Sinequan®).
2. Karacan I, Blackburn AB, Thornby JI: The Effect of Doxepin HCl (Sinequan®) on Sleep Patterns and Clinical Symptomatology of Neurotic Depressed Patients with Sleep Disturbance, in Mendels J (ed): *Sinequan®: A monograph of recent clinical studies*, Princeton, NJ, Excerpta Medica, 1977, pp 4-22.
3. Pitts NE: The clinical evaluation of doxepin–A new psychotherapeutic agent. *Psychosomatics* 10:164-171, 1969.

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Adapin® (doxepin HCl) ... effective single-entity drug with antidepressant and antianxiety effects

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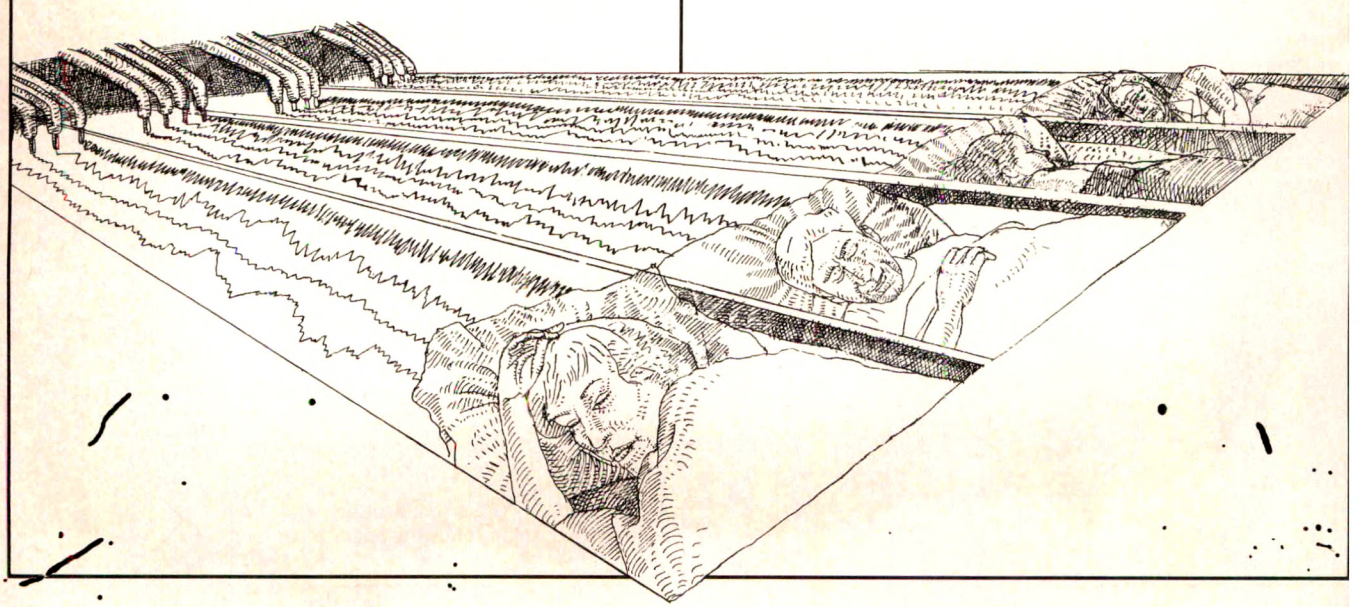
MORE POLYGRAPHIC EVIDENC

Sleep research laboratory testing of insomnia patients for 995 subject nights demonstrates efficacy of Dalmane (flurazepam HCl/Roche)[1]

In 14 separate studies, subjects given Dalmane were monitored with polygraphic recordings for a total of 995 nights, objectively demonstrating the efficacy of Dalmane.

Efficacy from the first night through 28 nights, without need to increase dosage

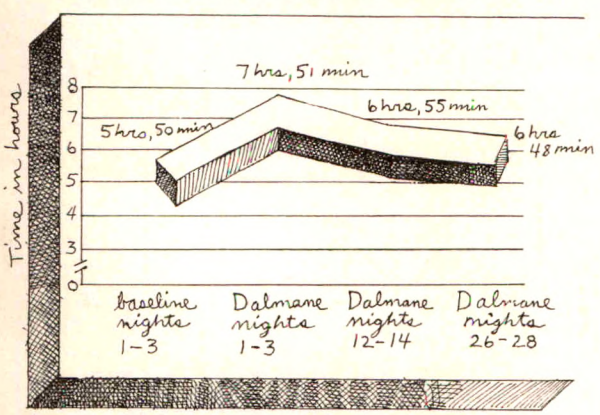
Dalmane (flurazepam HCl/Roche) is the only sleep medication objectively proven effective through 28 consecutive nights of administration in two separate studies in the sleep research laboratory.[2-4] The prolonged use of Dalmane is seldom necessary; should it be, appropriate patient evaluations such as periodic blood counts and liver and kidney function tests are recommended.



OFFERS

OF EFFICACY FOR INSOMNIA

Dalmane® (flurazepam HCl/Roche) improved sleep during short, intermediate and longer-term use



Improvement shown represents mean improvement, in hours, of total sleep time. n=5

Adapted from Dement WC, *et al: Behav Med* 5:25-31, Oct 1978

Since most insomnia is transitory in nature, initiate therapy at the lowest effective dose for the shortest time advisable to achieve a predetermined therapeutic goal.

References:
1. Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley NJ
2. Kales A, *et al: Clin Pharmacol Ther 19:* 576-583, May 1976
3. Dement WC, *et al: Behav Med 5:* 25-31, Oct 1978
4. Kales A, *et al: Clin Pharmacol Ther 18:* 356-363, Sep 1975

Clinical documentation of efficacy in 4950 patients with insomnia[1]

Dalmane (flurazepam HCl/Roche) efficacy was evaluated in 18 clinical studies. These included paired-night, double-blind crossover, double-blind comparison and patient preference studies. Results of these studies demonstrated and confirmed the effectiveness of Dalmane.

Periodic evaluation of patient's sleep habits may help prevent subsequent insomnia

Should insomnia recur, the patient may need guidance in setting up a regular sleep program. This may include establishment of a regular bedtime hour, taking a warm bath prior to retiring and avoidance of alcohol, stimulants or heavy meals late in the evening.

DALMANE® (IV)

flurazepam HCl/Roche
15-mg and 30-mg capsules

UNMATCHED PROOF, UNMATCHED EFFICACY

Please see summary of product information on following page.

ROCHE®

Protocol for insomnia management

☐ Treat physical and/or psychological factors that may underlie an inability to sleep.

☐ Thoroughly question and evaluate new patients who specifically request sleep medications.

☐ Consider other medications the patient may be taking (including alcoholic beverages) and be aware of possible drug interactions.

☐ Select a sleep medication with objectively *documented* proof of efficacy and a wide margin of safety.

☐ Initiate treatment at the lowest effective dose for the period of time deemed necessary *to achieve a reasonable therapeutic goal.*

☐ Monitor response to therapy and, based upon patient reassessment, determine need for continuing medication on an individual basis.

☐ Advise patients who ingest alcoholic beverages or other CNS depressants against doing so while on sleep medication. CNS stimulants such as caffeine should also be discontinued.

☐ Caution patients against engaging in hazardous occupations requiring complete mental alertness such as operating machinery or driving a motor vehicle shortly after ingesting the drug. In addition, as with all CNS depressants, patients may have reduced motor performance while on hypnotic therapy and should be advised accordingly.

☐ Avoid prescribing for suicidal or dependency-prone individuals whose histories indicate an inability to handle any psychoactive substances including alcohol.

Now available from Roche—important guidance for your patients with insomnia.

Roche Products Inc.
One Broad Avenue
Fairview, New Jersey 07022

Name _____

Address _____

City _____

State _____ Zip _____

DA-5T

DALMANE® (IV)
flurazepam HCl/Roche

One 15-mg capsule *h.s.*—recommended initial dosage for elderly or debilitated patients.
One 30-mg capsule *h.s.*—usual adult dosage
(15 mg may suffice in some patients).

Before prescribing, please consult complete product information, a summary of which follows:
Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Objective sleep laboratory data have shown effectiveness for at least 28 consecutive nights of administration. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended.
Contraindications: Known hypersensitivity to flurazepam HCl.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).
Usage in Pregnancy: **Several studies of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) suggest increased risk of congenital malformations during the first trimester of pregnancy. Dalmane, a benzodiazepine, has not been studied adequately to determine whether it may be associated with such an increased risk. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.
Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies, or with impaired renal or hepatic function. Periodic blood counts and liver and kidney function tests are advised during repeated therapy.
Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase.
Dosage: Individualize for maximum beneficial effect.
Adults: 30 mg usual dosage; 15 mg may suffice in some patients.
Elderly or debilitated patients: 15 mg recommended initially until response is determined.
Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

ROCHE PRODUCTS INC.
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ROCHE®

Alberta, Canada's fourth largest province, is experiencing unique economic growth based upon energy resource wealth, complemented by a commitment to the future and an enhancement of the quality of life for its citizens. In medicine and health care, this commitment is evident in support to universities, emphasis on medical research, and investment in facilities and services aimed at health care.

Alberta Hospital Edmonton is a progressive, active treatment psychiatric hospital comprised of acute, geriatric, rehabilitation, and forensic treatment services. The centre provides 120 beds for acute psychiatric admissions, and will be opening an expanded new forensic facility in late 1981. Fostering a multidisciplinary approach, Alberta Hospital Edmonton promotes progressive treatment methods and encourages active clinical and applied research.

FORENSIC AND CLINICAL PSYCHIATRISTS

prepared to accept the opportunity to practise in this vigorous, progressive environment, who hold F.R.C.P.(C), M.R.C. (PSYCH.) or equivalent are invited to forward Curriculum Vitae to:

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Medical Director
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Alberta, Canada
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Alabama Mental Health Department is currently recruiting General Primary Care Physicians and Psychiatrists. Positions are available in 11 locations, including Montgomery, Mobile and Tuscaloosa. We offer competitive salaries, 40 hour work week, a liberal benefits package, free health package and paid interviewing and relocation expenses. Alabama, with year round good weather, offers excellent recreation advantages, including theatre and the arts. Alabama is the place where good living and gracious manners are still the lifestyle. Our consultants can provide more information on interviewing qualified candidates. To find out more about these exciting opportunities, call our toll-free number listed below:

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PSYCHIATRIST

CERTIFIED

Professor (for position of Associate/or higher depending on qualifications.)

Must be native Arabic-speaker, with work experience in Arab countries. Preferably with strong background in research and/or clinical psychiatry.

Duties will include teaching research courses in newly established department and work in Public Health Clinic.

Benefits: separate housing allowance, furniture allowance, insurance coverage for self and family (up to four children under the age of eighteen), tickets to return home (place of recruitment) for self and family for annual vacation, two months bonus salary for each year of service, annual raise.

Salary, negotiable, depending on qualifications.

There are several positions available immediately. Send resume and certified copies of school and professional documents to: University of Garyounis in Benghazi, People's Bureau of The Socialist People's Libyan Arab Jamahiriya, Cultural Section, Post Office Box 19125, Washington, D.C. 20036.

DALHOUSIE UNIVERSITY
DEPARTMENT OF PSYCHIATRY

Chief of Service : Saint John Regional Hospital

The Saint John Regional Hospital, a teaching hospital of the Faculty of Medicine, Dalhousie University, is inviting applications from qualified medical practitioners for the position of Chief of the Department of Psychiatry.

A new 700-bed hospital with capacity to expand to 800 beds plus is completing construction to replace the present Regional Hospital in April 1982.

The Psychiatry facilities will include an expanded 58 bed in-patient unit and a new Day Ambulatory Care Unit.

The successful applicant will work closely with Dalhousie University and Centracare, a 500-bed Psychiatric facility in Saint John to plan and co-ordinate and organize a wide scope of Psychiatric services including Forensic, Pediatric and Adolescent Units.

The hospital is integrated with Dalhousie University for Resident and Interne Training. The Chief of Psychiatry will be appointed to an appropriate rank in the Faculty of Medicine.

The terms of employment are clearly competitive and can consider such areas as resettlement incentive and part-time private practice.

The residential environs of Saint John are most attractive, with a full range of educational (including University) and recreational facilities — golf, curling, skiing, sailing etc.

Submit applications with complete curriculum vitae to:

Chairman of Selection Advisory Committee
Department of Psychiatry — Dalhousie University
c/o The Dean's Office (New Brunswick)
P.O. Box 2100
Saint John, New Brunswick, Canada.
E2L 4L2.

CHILD AND ADOLESCENT PSYCHIATRIST

Child, Adolescent & Family Psychiatric Services
South Australia.

Willis House Adolescent Unit, Adelaide.

APPLICATIONS ARE INVITED FOR THE POSITION OF: — SENIOR PSYCHIATRIST-IN-CHARGE, MO8

$37,207.

The South Australian Health Commission is promoting the development of existing services in Child, Adolescent and Family Psychiatry in South Australia.

Opportunities exist for career psychiatrists in this field.

Willis House is the adolescent component of the Child, Adolescent and Family Psychiatric Services. The facility provides comprehensive diagnostic and therapeutic and educational services for in-patients, day patients and out patient adolescents and their families.

The position requires that candidates be well trained, adolescent and family psychiatrists with a firm clinical base. Skills in teaching, staff development, consultation and liaison work with a major emphasis on leadership are essential.

Qualifications and Experience:

1. Eligible for Membership of Royal Australian & New Zealand College of Psychiatrists.
2. Child Psychiatry (College accred.)
3. Minimum of 4 years experience since qualification, at least 2 of which should have been spent in C.A.F.P.S. is desirable.
4. Experience in clinical administration and staff management is a recommendation.

Enquiries:

Dr. C. Irwin,
Director,
Child, Adolescent and Family
Psychiatric Services.
Telephone (08) 223-2022.

Applications:

Applications are invited from persons regardless of sex, race, ethnic background or physical impairment. The names of 3 referees and curriculum vitae, should be forwarded and applications addressed to:

Manager,
Personnel Services,
South Australian Health
Commission,
52 Pirie Street,
ADELAIDE. 5000
South Australia.
Telephone (08) 228 0317.

Brief Summary of Prescribing Information

Indications and Usage: Symptomatic relief of anxiety, tension, agitation, irritability and insomnia associated with anxiety neuroses and transient situational disturbances; anxiety associated with depressive symptoms and as a treatment of symptoms of anxiety if such symptoms are a significant feature of functional or organic disorders, particularly gastrointestinal or cardiovascular.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients on lorazepam not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation.

Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions.

Observe usual precautions with impaired renal or hepatic function.

Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component.

Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg kg day. No effect dose was 1.25mg/kg/day (approximately 6 times the maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatric patients requires caution and frequent monitoring for symptoms of upper G.I. disease.

Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug.

In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Overdosage: In management of overdosage with any drug, bear in mind that multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring of vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

Ativan® IV for Anxiety
(lorazepam)

Dosage: **Individualize for maximum beneficial effects. Increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; insomnia due to anxiety or transient situational stress, 2-4mg h.s.**

How Supplied: **0.5, 1.0 and 2.0mg tablets.**

Wyeth Laboratories
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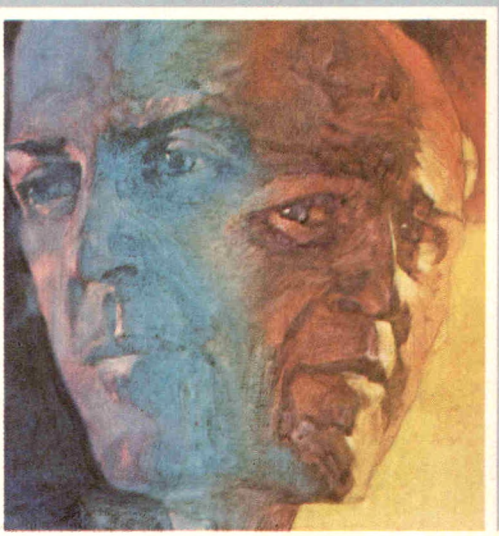
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A mixed syndrome. When drug intervention is indicated in patients whose anxiety is accompanied by depressive symptoms, the choice, essentially, is among three basic strategies: (1) an antidepressant; (2) a tranquilizer; (3) a combination of (1) and (2).

With respect to strategy (2), you should know that Ativan (lorazepam) has been shown to be statistically and clinically significantly effective in alleviating the anxiety component of this mixed syndrome, in common protocol controlled studies of 653 patients (337 on Ativan).

Since Ativan was also found to be compatible with tricyclics, if you opt for strategy (3), your patient has the benefit of rapid anxiolytic action, absence of long-acting metabolites and rapid clearance when you decide to discontinue Ativan. All benzodiazepines, however, including Ativan, produce added CNS depressant effects when administered with alcohol and other CNS depressants.

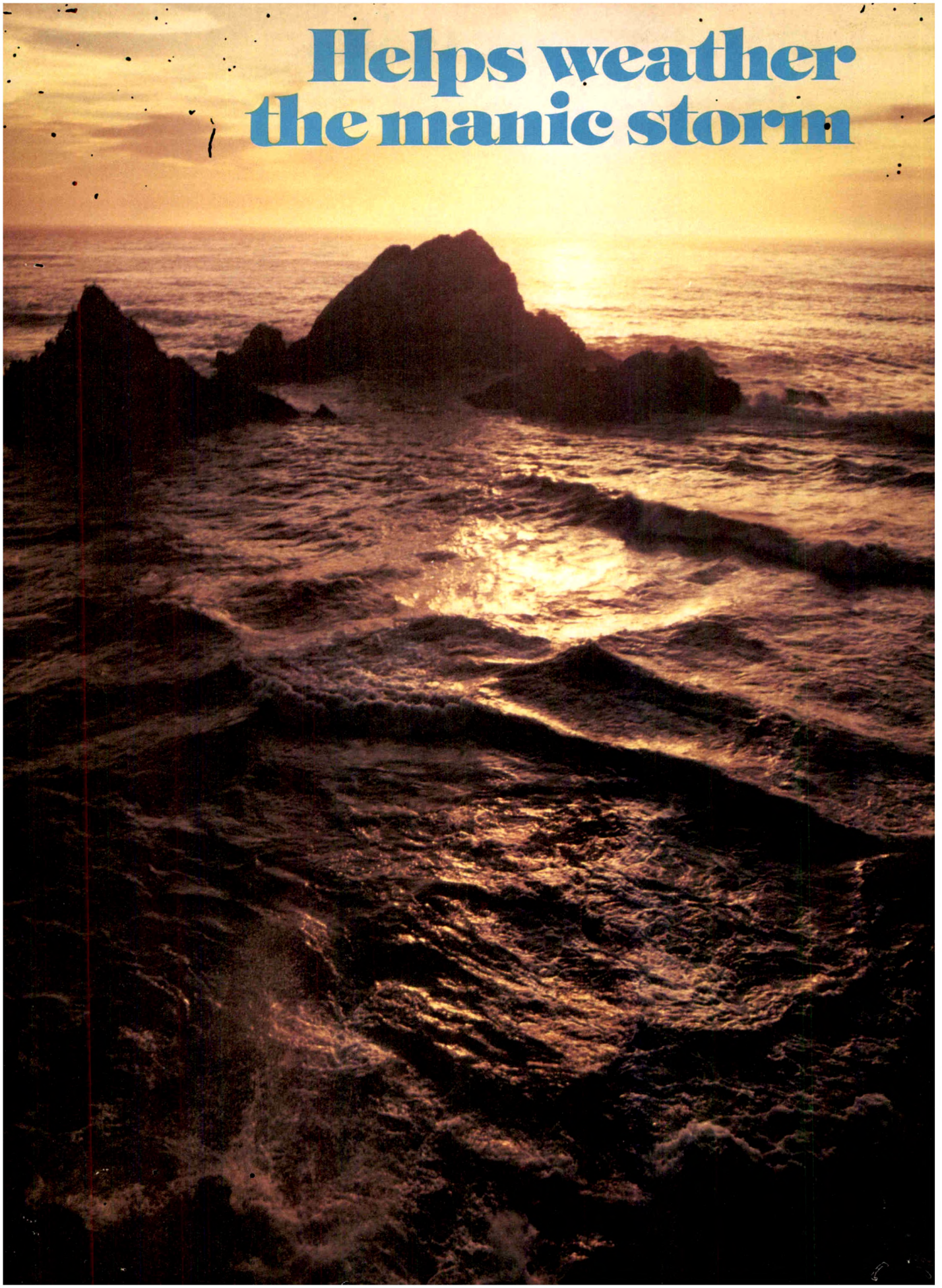
In either case, Ativan offers you the convenience of a b.i.d. schedule and a flexibility not possible with any fixed combination.



See important information on preceding page.

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associated with depressive symptoms


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In manic-depressive patients with a history of mania, 'Eskalith'

- prevents or diminishes the intensity of future episodes
- can increase the intervals between episodes
- helps patients function within normal limits

Before prescribing, see complete prescribing information in SK&F literature or PDR. The following is a brief summary.

> **WARNING**
> Lithium toxicity is closely related to serum lithium levels, and can occur at doses close to therapeutic levels. Facilities for prompt and accurate serum lithium determinations should be available before initiating therapy.

INDICATIONS: Treatment of manic episodes of manic-depressive illness. Maintenance therapy prevents or diminishes the intensity of subsequent episodes in manic-depressive patients with a history of mania.

WARNINGS: Lithium should generally not be given to patients with significant renal or cardiovascular disease, severe debilitation or dehydration, sodium depletion, or to patients receiving diuretics.

Lithium may prolong the effects of neuromuscular blocking agents. Such agents should be given with caution to patients receiving lithium. Lithium therapy has been reported in some cases to be associated with morphologic changes in the kidneys. The relationship between such changes and renal function has not been established.

Caution patient and family to watch for diarrhea, vomiting, tremor, mild ataxia, drowsiness, or muscular weakness as signs of lithium toxicity, and to discontinue therapy and contact a physician should they occur. Patients receiving combined therapy with lithium and an antipsychotic should be monitored closely for early evidence of neurologic toxicity and treatment discontinued promptly if such signs appear. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery).

Lithium should not be used in pregnancy, especially during the first trimester, unless potential benefits outweigh possible hazards. Except in rare and unusual circumstances, nursing should not be undertaken while a patient is on lithium therapy, since lithium is excreted in human milk.

Not recommended for children under 12.

PRECAUTIONS: Lithium tolerance is greater during the acute manic phase and decreases when manic symptoms subside.

Lithium therapy may lead to sodium depletion. Normal diet (including salt) and adequate fluid intake (2500-3000 ml.) must be maintained, at least during initial stabilization period. Protracted sweating or diarrhea can decrease tolerance; in such cases, administer supplemental fluid and salt.

Sweating, diarrhea, and concomitant infection with elevated temperatures may require temporary reduction or cessation of dosage.

Where hypothyroidism exists, thyroid function should be monitored during lithium stabilization and maintenance; where hypothyroidism occurs during stabilization and maintenance, supplemental thyroid treatment may be used.

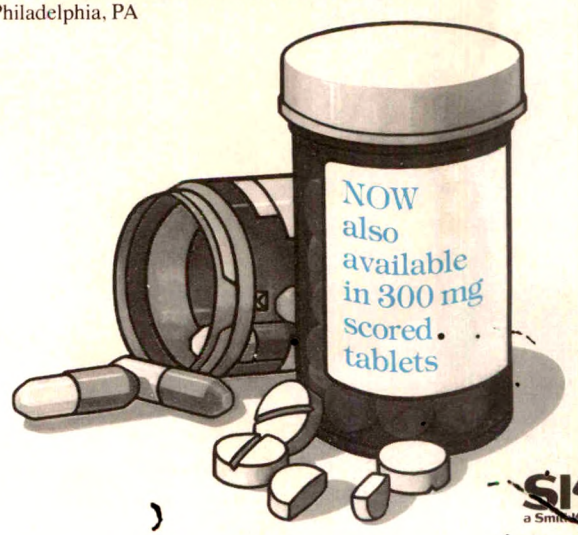
ADVERSE REACTIONS: Mild to moderate toxic reactions may occur at serum lithium levels from 1.5 to 2.5 mEq./1., and moderate to severe reactions at levels from 2.0 to 2.5 mEq./1. Fine hand tremor, polyuria, and mild thirst may occur during initial therapy and persist. Transient and mild nausea and general discomfort may also appear during initial therapy. These effects usually subside with continued treatment or temporary reduction or cessation of dosage. If persistent, discontinue dosage. Diarrhea, vomiting, drowsiness, muscular weakness, and lack of coordination may be early signs of toxicity and may occur at levels below 2.0 mEq./1. At higher levels, ataxia, giddiness, tinnitus, blurred vision, and a large output of dilute urine may be seen. Serum levels above 3.0 mEq./1. may produce a complex clinical picture, involving multiple organs and systems. Serum levels should not exceed 2.0 mEq./1. during acute phase.

The following reactions appear to be related to serum lithium levels, including levels within the therapeutic range: **Neuromuscular**—tremor, muscle hyperirritability (fasciculations, twitching, clonic movements of whole limbs), ataxia, choreo-athetotic movements, hyperactive deep tendon reflex; **Central Nervous System**—blackout spells, epileptiform seizures, slurred speech, dizziness, vertigo, incontinence of urine or feces, somnolence, psychomotor retardation, restlessness, confusion, stupor, coma; **Cardiovascular**—cardiac arrhythmia, hypotension, peripheral circulatory collapse; **Gastrointestinal**—anorexia, nausea, vomiting, diarrhea; **Genitourinary**—albuminuria, oliguria, polyuria, glycosuria; **Dermatologic**—drying and thinning of hair, alopecia, anesthesia of skin, chronic folliculitis, exacerbation of psoriasis, xerosis cutis; **Autonomic**—blurred vision, dry mouth; **Thyroid Abnormalities**—euthyroid goiter and/or hypothyroidism (including myxedema) with lower T_3 and T_4. I^{131} uptake may be elevated; rare cases of hyperthyroidism; **EEG Changes**—diffuse slowing, widening of the frequency spectrum, potentiation and disorganization of background rhythm; **EKG Changes**—reversible flattening, isoelectricity or inversion of T-waves; **Miscellaneous**—fatigue, lethargy, transient scotomata, dehydration, weight loss, tendency to sleep.

Reactions unrelated to dosage include: transient EEG and EKG changes, leukocytosis, headache, diffuse nontoxic goiter with or without hypothyroidism, transient hyperglycemia, generalized pruritus with or without rash, cutaneous ulcers, albuminuria, worsening of organic brain syndromes, excessive weight gain, edematous swelling of ankles or wrists, thirst or polyuria, sometimes resembling diabetes insipidus, and metallic taste. A single case of a syndrome resembling Raynaud's has been reported.

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Brief Summary

INDICATIONS — Symptomatic relief of anxiety and tension associated with anxiety disorders, other psychoneurotic disorders, transient situational disturbances, and functional or organic disorders. Symptomatic relief of acute alcohol withdrawal.

Effectiveness in long-term use (over 4 months) not assessed by systematic clinical studies. Physician should periodically reassess usefulness for each patient.

CONTRAINDICATIONS — Known hypersensitivity to the drug. Acute narrow angle glaucoma.

WARNINGS — Not for use in depressive neuroses or psychotic reactions. Caution patients against hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles. Advise against simultaneous use of other CNS depressants, and caution patients that effects of alcohol may be increased. Not recommended for patients under 18. Nervousness, insomnia, irritability, diarrhea, muscle aches, and memory impairment have followed abrupt withdrawal from long-term high dosage. Withdrawal symptoms were reported after abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months. Use caution in patients having psychological potential for drug dependence (dependence has been observed in dogs and rabbits).

Pregnancy and Lactation: Minor tranquilizers should almost always be avoided first trimester. Consider possibility of pregnancy before initiating therapy. Patient should consult physician about discontinuation if she becomes pregnant or plans pregnancy. Do not give to nursing mothers.

PRECAUTIONS — Observe usual precautions in depression accompanying anxiety, or in patients with suicidal tendency, or those with impaired renal or hepatic function. Do periodic blood counts and liver function tests during prolonged therapy. Use small doses and gradual increments in the elderly or debilitated.

ADVERSE REACTIONS — Drowsiness, dizziness, various g.i. complaints, nervousness, blurred vision, dry mouth, headache, mental confusion, insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression, slurred speech, abnormal liver and kidney function, decreased hematocrit, decreased systolic blood pressure.

DOSAGE — ANXIETY—Usual daily dose 30 mg or less (start the elderly or debilitated at 7.5-15 mg). Adjust gradually within 15-60 mg daily range. Capsules and scored tablets: divided doses; or once daily h.s. (start patient at 15 mg). Single Dose Tablets, 22.5 mg (for patients stabilized on 7.5 mg t.i.d.) or 11.25 mg: once daily at any hour. ALCOHOL WITHDRAWAL—In divided doses: 1st day 30 mg initially, then 30-60 mg; 2nd day 45-90 mg; 3rd day 22.5-45 mg; 4th day 15-30 mg. Then taper to 15-7.5 mg daily, and discontinue as soon as stable.

INTERACTIONS — Potentiation may occur with ethyl alcohol, hypnotics, barbiturates, narcotics, phenothiazines, MAO inhibitors, other antidepressants.

OVERDOSAGE — Take general measures as for any CNS depressant.

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Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

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Possibly effective: To control excessive anxiety, tension and agitation as seen in neuroses or associated with somatic conditions.

'Stelazine' has not been shown effective in the management of behavioral complications in patients with mental retardation.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose or greatly depressed states due to C.N.S. depressants; blood dyscrasias; bone marrow depression; liver damage.

Warnings: Generally avoid using in patients hypersensitive (e.g., blood dyscrasias, jaundice) to any phenothiazine. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery), especially during the first few days' therapy.

Additive effect is possible with other C.N.S. depressants, including alcohol.

Use in pregnancy only when necessary for patient's welfare.

Precautions: Use cautiously in angina. Avoid high doses and parenteral administration when cardiovascular system is impaired. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Prolonged administration of high doses may result in cumulative effects with severe C.N.S. or vasomotor symptoms. If retinal changes occur, discontinue drug. Agranulocytosis, thrombocytopenia, pancytopenia, anemia, cholestatic jaundice, liver damage have been reported.

Patients on long-term therapy, especially high doses, should be evaluated periodically for possible adjustment or discontinuance of drug therapy.

Adverse Reactions: Drowsiness, dizziness, skin reactions, rash, dry mouth, insomnia, amenorrhea, fatigue, muscular weakness, anorexia, lactation, blurred vision. Neuromuscular (extrapyramidal) reactions: motor restlessness, dystonias, pseudoparkinsonism, persistent tardive dyskinesia.

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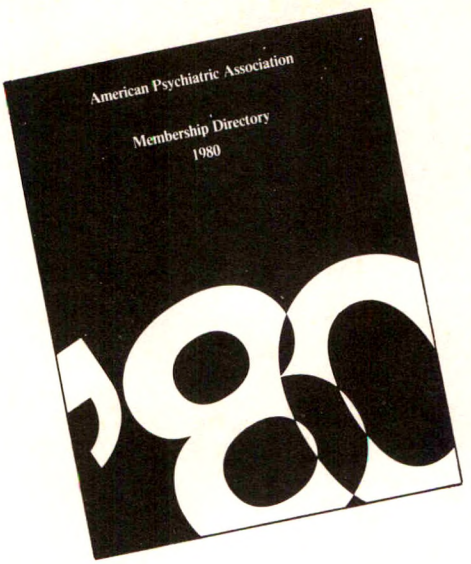
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Manuscripts that report the results of experimental investigation with human subjects must include a statement that informed consent was obtained after the procedure(s) had been fully explained.

Review Process

Papers are reviewed by at least two experts to determine the originality, validity, and significance of content and conclusions. Authors will usually be advised within 3–4 months of the decision on their paper, although delays are sometimes unavoidable. Reviewers' comments will be returned with rejected manuscripts if they are judged to be useful to the authors. All reviewers remain anonymous.

SUBMISSION OF MANUSCRIPTS

Manuscripts should be submitted in triplicate to John C. Nemiah, M.D., Editor, *American Journal of Psychiatry*, 1700 Eighteenth St., N.W., Washington, D.C. 20009. All correspondence will be sent to the first-named author unless otherwise specified. Papers that are not accompanied by a cover letter indicating that the paper is intended for publication will not be reviewed until such a statement is received. On a separate sheet include the names and mailing addresses of any authors who are not members of APA; they will be sent a complimentary copy of the issue in which their article appears.

Authors will be notified as soon as possible of the receipt of their paper and the number assigned to it. **This number must be included in all further correspondence.** It is imperative that the corresponding author of submitted papers notify the *Journal* of changes of address.

Annual Meeting Papers

The *Journal* has first refusal rights for numbered papers accepted for presentation at the Association's annual meetings, although not all papers can be published in the *Journal*. Authors of annual meeting papers should follow instructions they will receive in a letter from the Editor before the meeting. Annual meeting papers may be submitted for review before the meeting only if they are in **final form,** i.e., are ready for review. Authors must **not** submit their papers before the meeting if they expect any substantive revisions due to discussion at the meeting, further research, etc. These papers are subject to the same peer review criteria as other submissions. Authors who wish to submit their papers elsewhere must secure permission from the Editor. The *Journal* will assign its own number to each paper received for review; **this number must appear in any further communications about the paper.**

Length

The length of submitted material should not exceed the following specifications unless a special arrangement has been made with the Editor. Regular articles—3,800 words or the equivalent **(no more than 15 double-spaced pages, including references, tables, and figures).** Brief Communications—2,500 words **(no more than 10 double-spaced pages, including references, tables, and figures).** Clinical and Research Reports—1,000 words, 10 references, 1 table (no figures are used). Letters to the Editor—500 words, 5 references. The number of words, tables, and figures should be noted on the title page.

TYPES OF ARTICLES

Overviews

Overview articles attempt to bring together important relevant information on a topic of general interest to psychiatry. They are usually written at the invitation of the Editor; authors who have ideas for overview articles are advised to check with the Editor to ensure that a similar work is not in preparation. Overviews should not exceed 7,500 words **(no more than 25 double-spaced pages—including tables and figures—and 100 references).** All overviews, including those written by invitation, are given the same peer review received by other papers.

Regular Articles and Brief Communications

The primary difference between these two types of papers is length. The "brief communication" designation does not imply less sophisticated or complete work: it merely means that the content can be expressed within the upper limit of 2,500 words. There is no difference in the review procedure or scheduling of these articles. Authors who submit unnecessarily long articles with redundancies and loose writing style will be asked to shorten them, which can delay publication. Authors should choose their words carefully, avoiding lengthy introductions containing material that is common knowledge, summaries that merely repeat the results, unnecessary tables, figures, or references, etc. Single case reports should be submitted as Clinical and Research Reports.

Clinical and Research Reports

This section contains very brief articles (maximum of 1,000 words, 10 references, 1 table, and **no figures**) reporting new research findings, including preliminary data from pilot studies, and case reports that 1) describe new syndromes, 2) cast a new light on established ones, 3) indicate a new therapeutic procedure of potential value, or 4) describe adverse effects or previously unreported complications of drugs or therapeutic interventions. Because of the stringent criteria for this section, authors can expect more rapid publication than is possible in other sections. Submitted papers that substantially exceed the stated maximum length or contain figures will be returned to the author unreviewed.

Prompt Publication Policy

Articles submitted for prompt publication can be of either regular of Brief Communication length. (A "prompt publication policy" is automatically in effect for Clinical and Research Reports.) These papers are given priority in scheduling; however, authors should be aware that the minimum publication time is four months. Prompt publication papers must meet stringent criteria of originality and be of major immediate importance to the field. Authors must state their reasons for wanting rapid review in a cover letter to the Editor. It is important to think carefully about the nature of the paper before requesting prompt publication; a paper may be delayed if it is submitted inappropriately.

Other Sections

Letters to the Editor. Brief letters (maximum of 500 words and 5 references) will be considered if they include the notation "for publication" in the upper right corner. Letters critical of an article published in the *Journal* will automatically be sent to the authors for reply. Because of space limitations not all letters can be printed. The *Journal* is unable to notify authors of receipt of letters or to return those not published. All letters are subject to editing. Letters must be typed **double-spaced** throughout on letter-size bond paper; two copies are required. Letters that are not typed appropriately or do not conform to *Journal* reference style will be returned to the author for revision.

Book Reviews. Books for review may be sent directly to the Book Review Editor, Nancy C. Andreasen, M.D., University of Iowa College of Medicine, 500 Newton Rd., Iowa City, Iowa 52240. Book reviews are usually solicited by the Book Review Editor. Authors interested in reviewing a particular book are urged to contact the editorial office to see if the Book Review Editor wishes the book to be reviewed and to ascertain whether it has been previously assigned to another reviewer.

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All parts of the manuscript, including case reports, quotations, references, and tables, must be **double-spaced** throughout. All manuscripts must be typed in upper- and lowercase on one side only of 22 × 28 cm (8½ × 11 inches) **nonerasable** bond paper. All 4 margins must be 3¾ cm (1½ inches). The manuscript should be arranged in the following order, with each numbered item beginning a new page: 1) title page, 2) précis, 3) text, 4) references, 5) tables, 6) footnotes to text, and 7) figure captions. All pages must be numbered, with the title page as number 1. Please consult the following section on *Journal* style specifications for criteria for each part of the paper.

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Other requirements. The number of words, tables, and figures should appear in the upper right corner and a phone number for the corresponding author in the lower right.

Précis

The précis should be up to 100 words for regular articles and Brief Communications. (No précis is used in the Clinical and Research Reports section.) The précis should be a single paragraph using complete, connected sentences, active verbs, and the third person. In most cases, the précis replaces a summary.

Text

Headings and subheadings should be inserted at reasonable intervals in all types of papers. Footnotes to text material should be typed on a separate page at the end of the manuscript. Summaries are rarely desirable, although a final "Comment" or "Conclusions" section may be used. The body of the paper should be written in the active voice and first person where appropriate.

Statistics. Authors should be sure that statistics used are

both necessary and appropriate. Statistical tests should be described and a reference citation given if the tests are not generally known. It is not sufficient to say "differences between the groups were significant." In cases of lengthy complicated analyses, the authors may summarize results and indicate that details are available from them on request.

Abbreviations. All abbreviations (other than those for metric units) should be explained the first time they are used. Idiosyncratic abbreviations should be avoided: overuse of abbreviations may hinder rather than facilitate readability.

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References should be restricted to closely pertinent material: a complete review of the literature is rarely desirable. **Accuracy of citation is the author's responsibility.** References should conform exactly to the original spelling, accents, punctuation, etc. Authors should be sure that all references listed have been cited in text; no bibliographies can be used.

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Type references in the style shown below, **double-spaced throughout** (not just a line between references). List up to three authors; designate one or more authors past the third "et al." Abbreviations of journal names should conform to the style used in *Index Medicus*; journals not indexed there should not be abbreviated.

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2. Blackwell B, Marley E, Price J, et al: Hypertensive interactions between monoamine oxidase inhibitors and foodstuffs. Br J Psychiatry 113:349-365, 1967
3. Jones AB: Long-term administration of tricyclic antidepressants: three-year follow-up. Am J Psychiatry (in press)
4. Smythe JCW (ed): Psychiatry and Human Values. Springfield, Ill, Charles C Thomas, 1971
5. Brosin H: Communication systems of the consultation process, in The Psychiatric Consultation. Edited by Mendel W, Solomon P. New York, Grune & Stratton, 1968

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Figures should be used only for data that cannot be adequately presented in text or tabular form. Figures should be submitted as glossy prints with one photocopy attached to each manuscript copy. The author's name and the title of the paper should appear on a gummed label affixed to the back of each glossy. All figures should be able to withstand reduction to about 8 cm (3 1/4 inches). Authors are urged to consider carefully the necessity of figures; they may be asked to delete figures that repeat information availabile in text. It is often advisable to obtain professional assistance in the preparation of figures.

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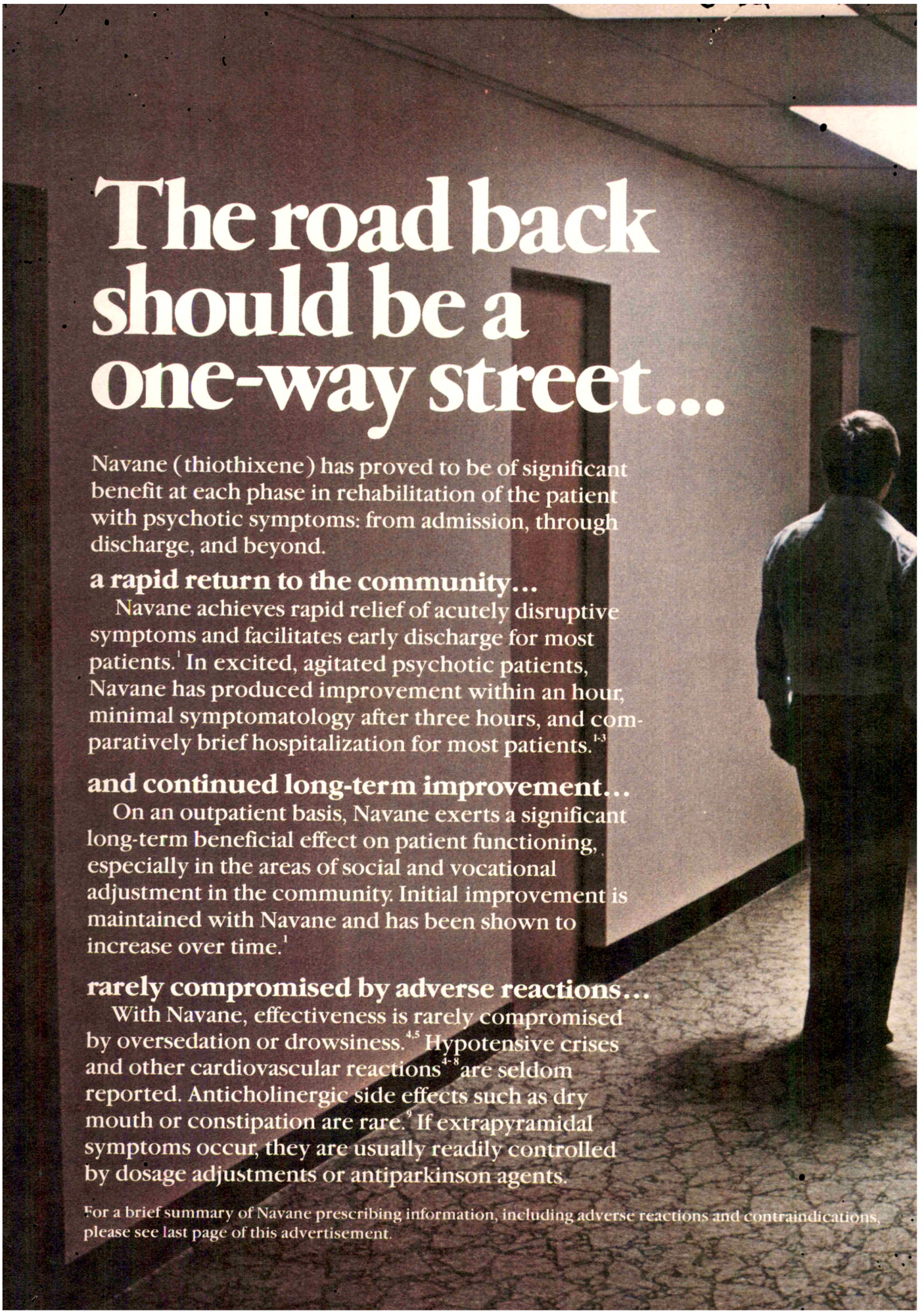
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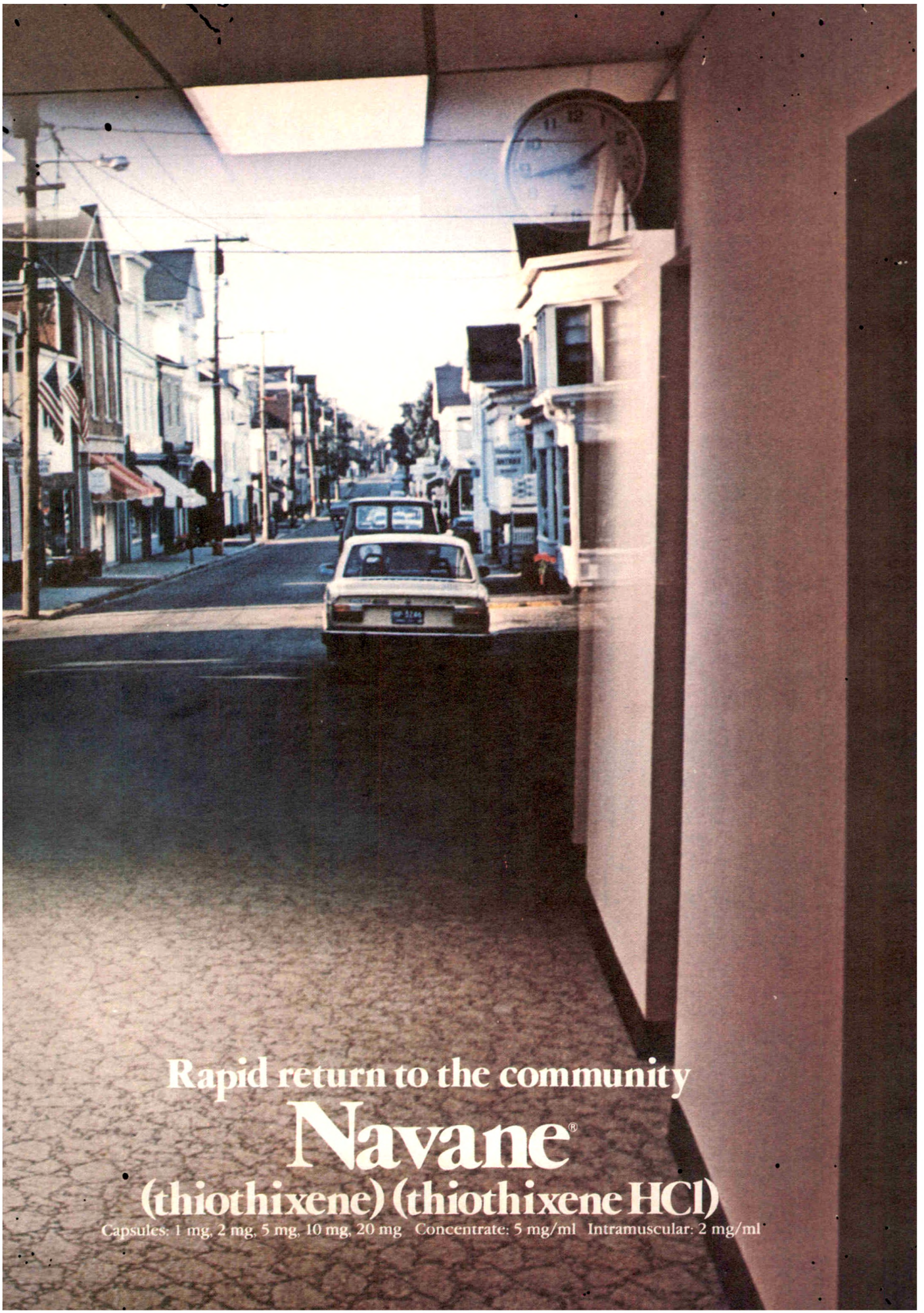
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With Navane, effectiveness is rarely compromised by oversedation or drowsiness.[4,5] Hypotensive crises and other cardiovascular reactions[4-8] are seldom reported. Anticholinergic side effects such as dry mouth or constipation are rare.[9] If extrapyramidal symptoms occur, they are usually readily controlled by dosage adjustments or antiparkinson agents.

For a brief summary of Navane prescribing information, including adverse reactions and contraindications, please see last page of this advertisement.


Rapid return to the community
Navane®
(thiothixene) (thiothixene HCl)
Capsules: 1 mg, 2 mg, 5 mg, 10 mg, 20 mg Concentrate: 5 mg/ml Intramuscular: 2 mg/ml

Rapid return to the community

Navane® (thiothixene) (thiothixene hydrochloride)

Capsules: 1 mg, 2 mg, 5 mg, 10 mg, 20 mg Concentrate: 5 mg/ml Intramuscular: 2 mg/ml

BRIEF SUMMARY OF PRESCRIBING INFORMATION
Navane® (thiothixene)
Capsules: 1 mg, 2 mg, 5 mg, 10 mg, 20 mg
(thiothixene hydrochloride) Concentrate: 5 mg/ml,
Intramuscular: 2 mg/ml

Contraindications. Navane (thiothixene) is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy*– Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration of Navane to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children– The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration– As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane (thiothixene). Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudo-parkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leukocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution–For Intramuscular Use Only. Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplan the injectable form as soon as possible. It may be necessary t adjust the dosage when changing from the intramuscular to ora dosage forms. Dosage recommendations for Navane (thiothixene Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate– In milder conditions an initial dose of 2 mg three times daily. If indicated, a subsequen increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily. The usual optimal dose is 20 to 30 mg daily. If indicated, ar increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficia response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Stimulants that may cause convulsions (e.g. picrotoxin or pentylenetetrazol) should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg of thiothixene in bottles of 100, 1,000, and unit-dose pack of 100 (10 x 10's). Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100, 500, and unit-dose pack of 100 (10 x 10's).

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg, and in 30 ml (1 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, and 5 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10 vials. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

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For additional information on Navane, please consult your Roerig representative or write to: Roerig Medical Department, 235 East 42nd Street, New York, NY 10017.

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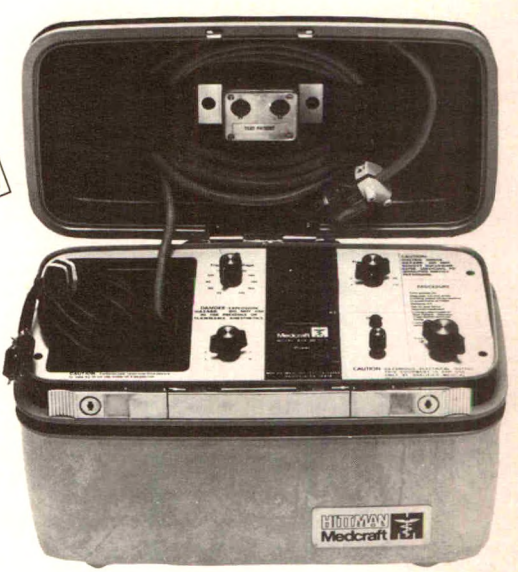
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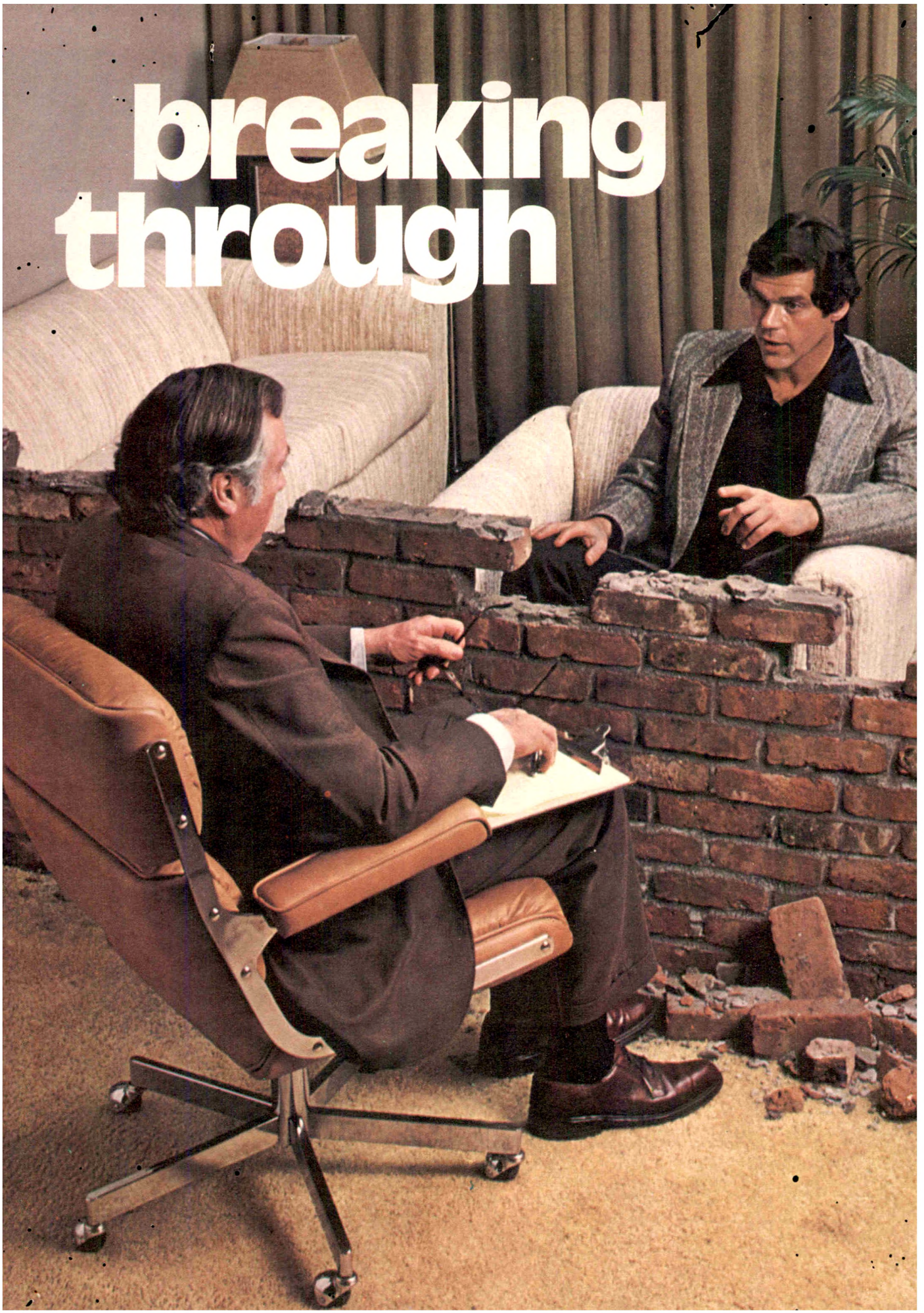
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Treatment with TRIAVIL—a balanced view:
TRIAVIL is contraindicated in CNS depression from drugs, in the presence of evidence of bone marrow depression, and in patients hypersensitive to phenothiazines or amitriptyline. It should not be used during the acute recovery phase following myocardial infarction or in patients who have received an MAOI within two weeks. Patients with cardiovascular disorders should be watched closely. Not recommended in children or during pregnancy. TRIAVIL may impair mental and/or physical abilities required for performance of hazardous tasks and may enhance the response to alcohol. Antiemetic effect may obscure toxicity due to overdosage of other drugs or mask other disorders. The possibility of suicide in depressed patients remains until significant remission occurs. Such patients should not have access to large quantities of the drug. Hospitalize as soon as possible any patient suspected of having taken an overdose.



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containing perphenazine and amitriptyline HCl

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TRIAVIL® 4-25: Each tablet contains
4 mg perphenazine and 25 mg amitriptyline HCl.
TRIAVIL® 4-10: Each tablet contains
4 mg perphenazine and 10 mg amitriptyline HCl.

CONTRAINDICATIONS: Central nervous system depression from drugs (barbiturates, alcohol, narcotics, analgesics, antihistamines); evidence of bone marrow depression; known hypersensitivity to phenothiazines or amitriptyline. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred from such combinations. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with TRIAVIL. Therapy should then be initiated cautiously with gradual increase in dosage until optimum response is achieved. Not recommended for use during acute recovery phase following myocardial infarction.

WARNINGS: TRIAVIL should not be given concomitantly with guanethidine or similarly acting compounds since TRIAVIL may block the antihypertensive action of such compounds. Use cautiously in patients with history of urinary retention, angle-closure glaucoma, increased intraocular pressure, or convulsive disorders. Dosage of anticonvulsive agents may have to be increased. In patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely. Tricyclic antidepressants, including amitriptyline HCl, have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time, particularly in high doses. Myocardial infarction and stroke have been reported with tricyclic antidepressant drugs. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Not recommended in children or during pregnancy.

PRECAUTIONS: Suicide is a possibility in depressed patients and may remain until significant remission occurs. Such patients should not have access to large quantities of this drug.

Perphenazine: Should not be used indiscriminately. Use with caution in patients who have previously exhibited severe adverse reactions to other phenothiazines. Likelihood of some untoward actions is greater with high doses. Closely supervise with any dosage. The antiemetic effect of perphenazine may obscure signs of toxicity due to overdosage of other drugs or make more difficult the diagnosis of disorders such as brain tumor or intestinal obstruction. A significant, not otherwise explained, rise in body temperature may suggest individual intolerance to perphenazine, in which case discontinue.

If hypotension develops, epinephrine should not be employed, as its action is blocked and partially reversed by perphenazine. Phenothiazines may potentiate the action of central nervous system depressants (opiates, analgesics, antihistamines, barbiturates, alcohol) and atropine. In concurrent therapy with any of these, TRIAVIL should be given in reduced dosage. May also potentiate the action of heat and phosphorous insecticides. There is sufficient experimental evidence to conclude that chronic administration of antipsychotic drugs which increase prolactin secretion has the potential to induce mammary neoplasms in rodents under the appropriate conditions. There are recognized differences in the physiological role of prolactin between rodents and humans. Since there are, at present, no adequate epidemiological studies, the relevance to human mammary cancer risk from prolonged exposure to perphenazine and other antipsychotic drugs is not known.

Amitriptyline: In manic-depressive psychosis, depressed patients may experience a shift toward the manic phase if they are treated with an antidepressant. Patients with paranoid symptomatology may have an exaggeration of such symptoms. The tranquilizing effect of TRIAVIL seems to reduce the likelihood of this effect. When amitriptyline HCl is given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. Paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs.

Caution is advised if patients receive large doses of ethchlorvynol concurrently. Transient delirium has been reported in patients who were treated with 1 g of ethchlorvynol and 75-150 mg of amitriptyline HCl.

Amitriptyline HCl may enhance the response to alcohol and the effects of barbiturates and other CNS depressants.

Concurrent administration of amitriptyline HCl and electroshock therapy may increase the hazards associated with such therapy. Such treatment should be limited to patients for whom it is essential. Discontinue several days before elective surgery if possible. Elevation and lowering of blood sugar levels have both been reported. Use with caution in patients with impaired liver function.

ADVERSE REACTIONS: Similar to those reported with either constituent alone.
Perphenazine: Extrapyramidal symptoms (opisthotonus, oculogyric crisis, hyperreflexia, dystonia, akathisia, acute dyskinesia, ataxia, parkinsonism) have been reported and can usually be controlled by the concomitant use of effective antiparkinsonian drugs and/or by reduction in dosage, but sometimes persist after discontinuation of the phenothiazine.

Tardive dyskinesia may appear in some patients on long-term therapy or may occur after drug therapy with phenothiazines and related agents has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw. Involuntary movements of the extremities sometimes occur. There is no known treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms. It is advised that all antipsychotic agents be discontinued if the above symptoms appear. If treatment is reinstituted, or dosage of the particular drug increased, or another drug substituted, the syndrome may be masked. Fine vermicular movements of the tongue may be an early sign of the syndrome. The full-blown syndrome may not develop if medication is stopped when lingual vermiculation appears.

Other side effects are skin disorders (photosensitivity, itching, erythema, urticaria, eczema, up to exfoliative dermatitis); other allergic reactions (asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions); peripheral edema; reversed epinephrine effect; hyperglycemia; endocrine disturbances (lactation, galactorrhea, gynecomastia, disturbances of menstrual cycle); altered cerebrospinal fluid proteins; paradoxical excitement; hypertension, hypotension, tachycardia, and ECG abnormalities (quinidine-like effect); reactivation of psychotic processes; catatonic-like states; autonomic reactions, such as dry mouth or salivation, headache, anorexia, nausea, vomiting, constipation, obstipation, urinary frequency or incontinence, blurred vision, nasal congestion, and a change in pulse rate; other adverse reactions reported with various phenothiazine compounds, but not with perphenazine, include grand mal convulsions, cerebral edema, polyphagia, pigmentary retinopathy, photophobia, skin pigmentation, and failure of ejaculation.

The phenothiazine compounds have produced blood dyscrasias (pancytopenia, thrombocytopenic purpura, leukopenia, agranulocytosis, eosinophilia); and liver damage (jaundice, biliary stasis).

Pigmentation of the cornea and lens has been reported to occur after long-term administration of some phenothiazines. Although it has not been reported in patients receiving TRIAVIL, the possibility that it might occur should be considered.

Hypnotic effects, lassitude, muscle weakness, and mild insomnia have also been reported.

Amitriptyline: Note: Listing includes a few reactions not reported for this drug, but which have occurred with other pharmacologically similar tricyclic antidepressant drugs and must be considered when amitriptyline is administered. *Cardiovascular:* Hypotension; hypertension; tachycardia; palpitation; myocardial infarction; arrhythmias; heart block; stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth; blurred vision; disturbance of accommodation; increased intraocular pressure; constipation; paralytic ileus; urinary retention; dilatation of urinary tract. *Allergic:* Skin rash; urticaria; photosensitization; edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis; leukopenia; eosinophilia; purpura; thrombocytopenia. *Gastrointestinal:* Nausea; epigastric distress; vomiting; anorexia; stomatitis; peculiar taste; diarrhea; parotid swelling; black tongue. Rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido; elevated or lowered blood sugar levels. *Other:* Dizziness, weakness; fatigue; headache; weight gain or loss; increased perspiration; urinary frequency; mydriasis; drowsiness; alopecia. *Withdrawal Symptoms:* Abrupt cessation after prolonged administration may produce nausea, headache, and malaise. These are not indicative of addiction.

OVERDOSAGE: All patients suspected of having taken an overdosage should be admitted to a hospital as soon as possible. Treatment is symptomatic and supportive. However, the intravenous administration of 1–3 mg of physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage of physostigmine should be repeated as required particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine. On this basis, in severe overdosage with perphenazine-amitriptyline combinations, symptomatic treatment of central anticholinergic effects with physostigmine salicylate should be considered.

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Responsive
to therapy...
alert
on the job.*
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Haldol®
(haloperidol)
tablets/concentrate/injection

Promptly controls psychotic symptoms... usually leaving the patient alert and productive

Highly effective in a wide range of both acute and chronic psychotic disorders,[1-7] such as hallucinations, delusions, suspiciousness, hostility, mania.

Permits aggressive titration to effective dosage levels for optimal response.[1,4]

Facilitates prompt initiation of other therapeutic efforts... and often helps avoid hospitalization.[1-3]

Minimal risk of hypotension, oversedation, or troublesome anticholinergic effects.[2,4-6,8,9] Transient hypotension occurs rarely; severe orthostatic hypotension has not been reported. Although some instances of drowsiness have been reported, marked sedation is rare.

Common side effects easily controlled.[1,2,4-7] Although extrapyramidal symptoms (EPS) have been reported frequently, they are usually dose-related and readily controlled with dose adjustment or antiparkinson drugs. EPS often diminish spontaneously with continued use of HALDOL haloperidol.

References: 1. Man, P.L.: Dis. Nerv. Syst. *34*:113 (Feb.) 1973. **2.** Ayd, F.J., Jr.: Med. Sci. *18*:55 (Oct.) 1967. **3.** Rapp, M.S.: Can. Psychiatr. Assoc. J. *15*:73 (Feb.) 1970. **4.** Howard, J.S.: Dis. Nerv. Syst. *35*:458 (Oct.) 1974. **5.** Gerle, B.: Clin. Trials J. *3*:380 (Feb.) 1966. **6.** Abuzzahab, F.S., Sr.: Psychosomatics *11*:188 (May-June) 1970. **7.** Darling, H.F.: Dis. Nerv. Syst. *34*:364 (Oct.-Nov.) 1973. **8.** Snyder, S.H., et al: Science *184*:1243 (June 21) 1974. **9.** Stimmel, C.L.: Dis. Nerv. Syst. *34*:219 (Apr.) 1976.

*Not an actual case history, this situation illustrates the action of HALDOL haloperidol as reported in various clinical studies (available on request).

Please turn page for summary of prescribing information.

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concentrate

A tasteless, odorless, colorless liquid concentrate for better patient compliance: 2 mg per ml.

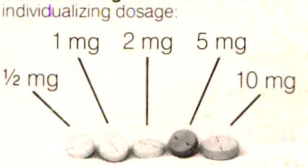
injection

A rapid-acting injection for psychiatric emergencies: 5 mg per ml,* with 1.8 mg methylparaben and 0.2 mg propylparaben per ml, and lactic acid for pH adjustment to 3.4 ± 0.2.



tablets

5 tablet strengths for convenience in individualizing dosage:



HALDOL® (haloperidol)

tablets / concentrate / injection

A dosage form for every therapeutic need

Summary of Prescribing Information

Contraindications: Severe depression, coma, CNS depression due to centrally-acting depressants, Parkinson's disease, hypersensitivity to the drug.

Warnings: ***Usage in Pregnancy:*** Safe use in pregnancy or in women likely to become pregnant has not been established; use only if benefit clearly justifies potential hazards. Infants should not be nursed during drug treatment.

Usage in Children: Safety and effectiveness not established; not recommended in pediatric age group.

Combined Use With Lithium: Patients receiving lithium plus haloperidol should be monitored closely for early evidence of neurological toxicity.

General: Bronchopneumonia, sometimes fatal, has followed use of major tranquilizers, including haloperidol. Prompt remedial therapy should be instituted if dehydration, hemoconcentration or reduced pulmonary ventilation occurs, especially in the elderly. Decreased serum cholesterol and/or cutaneous and ocular changes have been reported with chemically-related drugs, although not with haloperidol. Mental and/or physical abilities required for hazardous tasks or driving may be impaired. Alcohol should be avoided due to possible additive effects and hypotension.

Precautions: Administer cautiously to patients: (1) with severe cardiovascular disorders, due to the possibility of transient hypotension and/or precipitation of anginal pain (if a vasopressor is required, epinephrine should not be used since HALDOL haloperidol may block its vasopressor activity and paradoxical further lowering of blood pressure may occur); (2) receiving anticonvulsant medication since HALDOL haloperidol may lower the convulsive threshold; (3) with known allergies or a history of allergic reactions to drugs; (4) receiving anticoagulants. Concomitant antiparkinson medication, if required, may have to be continued after HALDOL haloperidol is discontinued because of different excretion rates; if both are discontinued simultaneously, extrapyramidal symptoms may occur. Intraocular pressure may increase when anticholinergic drugs, including antiparkinson drugs, are administered concomitantly with HALDOL haloperidol. When HALDOL haloperidol is used for mania in cyclic disorders, there may be a rapid mood swing to depression. Severe neurotoxicity may occur in patients with thyrotoxicosis receiving antipsychotic medication, including HALDOL haloperidol.

Adverse Reactions: ***CNS Effects:*** *Extrapyramidal Reactions:* Neuromuscular (extrapyramidal) reactions have been reported frequently, often during the first few days of treatment. Generally they involved Parkinson-like symptoms which were usually mild to moderately severe and usually reversible. Other types of neuromuscular reactions (motor restlessness, dystonia, akathisia, hyperreflexia, opisthotonos, oculogyric crises) have been reported far less frequently, but were often more severe. Severe extrapyramidal reactions have been reported at relatively low doses. Generally, extrapyramidal symptoms are dose-related since they occur at relatively high doses and disappear or become less severe when the dose is reduced. Antiparkinson drugs may be required. Persistent extrapyramidal reactions have been reported and the drug may have to be discontinued in such cases.

Withdrawal Emergent Neurological Signs: Abrupt discontinuation of short-term antipsychotic therapy is generally uneventful. However, some patients on maintenance treatment experience transient dyskinetic signs after abrupt withdrawal. In certain cases these are indistinguishable from "Persistent Tardive Dyskinesia" except for duration. It is unknown whether gradual withdrawal will reduce the occurrence of these signs, but until further evidence is available haloperidol should be gradually withdrawn.

Persistent Tardive Dyskinesia: Although rarely reported with HALDOL haloperidol, tardive dyskinesia may appear during or after long-term therapy. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and sometimes appear irreversible; there is no known effective treatment and all antipsychotic agents should be discontinued. The syndrome may be masked by reinstitution of drug, increasing dosage, or switching to a different antipsychotic agent.

Other CNS Effects: Insomnia, restlessness, anxiety, euphoria, agitation, drowsiness, depression, lethargy, headache, confusion, vertigo, grand mal seizures, and exacerbation of psychotic symptoms.

Cardiovascular Effects: Tachycardia and hypotension. ***Hematologic Effects:*** Reports of mild, usually transient leukopenia and leukocytosis, minimal decreases in red blood cell counts, anemia, or a tendency toward lymphomonocytosis; agranulocytosis rarely reported and only in association with other medication. ***Liver Effects:*** Impaired liver function and/or jaundice reported. ***Dermatologic Reactions:*** Maculopapular and acneiform reactions, isolated cases of photosensitivity, loss of hair. ***Endocrine Disorders:*** Lactation, breast engorgement, mastalgia, menstrual irregularities, gynecomastia, impotence, increased libido, hyperglycemia and hypoglycemia. ***Gastrointestinal Effects:*** Anorexia, constipation, diarrhea, hypersalivation, dyspepsia, nausea and vomiting. ***Autonomic Reactions:*** Dry mouth, blurred vision, urinary retention and diaphoresis. ***Respiratory Effects:*** Laryngospasm, bronchospasm and increased depth of respiration.

The injectable form is intended only for acutely agitated psychotic patients with moderately severe to very severe symptoms.

Caution: Federal law prohibits dispensing without prescription.

Full directions for use should be read before HALDOL haloperidol is administered or prescribed.

HALDOL tablets are manufactured by McNeil Laboratories Co., Dorado, Puerto Rico 00646.

* haloperidol present as the lactate

McNEIL

McNeil Laboratories, McNEILAB, Inc.
Fort Washington, PA 19034

PREVENTING MENTAL ILLNESS

Efforts and Attitudes

A new publication of The Joint Information Service of the American Psychiatric Association and the Mental Health Association
Raymond M. Glasscote; *Eleanor Kohn, Allan Beigel, Merrill F. Raber, Nancy Roeske, Bonnie A. Cox, James R. Raybin, Bernard L. Bloom*

There has long been interest in preventing mental illness, but no great emphasis on financing necessary research and pilot programs. Dramatic discoveries of nutritional, toxic, and infective causes of certain psychoses were made long ago and led to their prevention, but prevention of schizophrenia, mania, sociopathy, and biological depression, among other major disorders, requires further knowledge.

In recent years faith has grown that programs supporting those experiencing heavy stress and privation will provide benefits increasing the likelihood that they will remain functional, productive, and well. This volume provides a balanced, fairminded, and realistic explication of present possibilities and reasonable expectations. It explores a variety of viewpoints and describes in significant detail a variety of prevention-motivated programs for a variety of vulnerable people and provides such outcome information as is presently available.

1980. 138 pp., Casebound.

Mail to: Publication Sales Department, American Psychiatric Association,
1700 18th Street, N.W., Washington, D.C. 20009.

Please send me _____ copies of **Preventing Mental Illness** for which remittance of $ _____ is enclosed. (Note: Price per copy is $10.00; for orders of 4 or more copies, $8.50 each.) **Order #158.**

All domestic orders amounting to $35.00 or less must be accompanied by payment; all foreign orders, regardless of dollar amount, must be accompanied by payment in U.S. funds. Orders over $35.00 will be invoiced with shipping and handling charges included.

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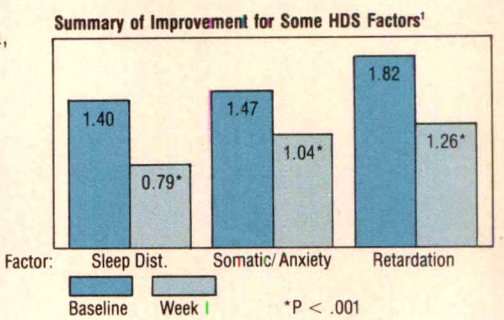
When depressive symptoms come in a cluster of blues...



Adapin® (doxepin HCl) helps patients see life in all its colors

Improvements noted at one week

Depressed patients often present with fatigue, anorexia, anxiety, mood swings, loss of interest, *and most frequently, early awakening and insomnia*. In a summation of 9 clinical studies involving 198 patients, significant clinical improvement was noted after the first week of doxepin treatment.[1] However, optimal antidepressant response may not be evident for two to three weeks.



Normalized sleep patterns

In a study of clinically depressed patients with symptoms of insomnia, treatment with doxepin significantly reduced difficulty falling asleep, difficulty staying asleep and frequency of early morning awakening.[2]

Low potential for cardiotoxicity

The incidence of tachycardia and hypotension, which have been observed during treatment with tricyclic antidepressants, has been reported infrequently with doxepin. In 452 patients given doxepin, the incidence of tachycardia was 2.88%; in 495 patients, the incidence of hypotension was 2.62%.[3]

Now available...New 75 mg capsules for h.s. dosage

Brief Summary of Prescribing Information ADAPIN® (doxepin HCl) Capsules

Indications–Relief of symptoms of anxiety and depression.

Contraindications–Glaucoma, tendency toward urinary retention or hypersensitivity to doxepin.

Warnings–Adapin has not been evaluated for safety in pregnancy. No evidence of harm to the animal fetus has been shown in reproductive studies. There are no data concerning secretion in human milk, nor on effect in nursing infants.

Usage in children under 12 years of age is not recommended. MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug, as serious side-effects and death have been reported with the concomitant use of certain drugs and MAO inhibitors.

In patients who may use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage.

Precautions–Drowsiness may occur and patients should be cautioned against driving a motor vehicle or operating hazardous machinery.

Since suicide is an inherent risk in depressed patients they should be closely supervised while receiving treatment.

Although Adapin has shown effective tranquilizing activity, the possibility of activating or unmasking latent psychotic symptoms should be kept in mind.

This product contains FD&C Yellow No. 5 (tartrazine) which may cause allergic-type reactions (including bronchial asthma) in certain susceptible individuals. Although the overall incidence of FD&C Yellow No. 5 (tartrazine) sensitivity in the general population is low, it is frequently seen in patients who also have aspirin hypersensitivity.

Adverse Reactions–Dry mouth, blurred vision and constipation have been reported. Drowsiness has also been observed.

Adverse effects occurring infrequently include extrapyramidal symptoms, gastrointestinal reactions, secretory effects such as sweating, tachycardia and hypotension. Weakness, dizziness, fatigue, weight gain, edema, paresthesias, flushing, chills, tinnitus, photophobia, decreased libido, rash and pruritus may also occur.

Dosage and Administration–In mild to moderate anxiety and/or depression: 25 mg t.i.d. Increase or decrease the dosage according to individual response. Daily dosage, up to 150 mg may be taken at bedtime without loss of effectiveness. Usual optimum daily dosage is 75 mg to 150 mg per day not to exceed 300 mg per day.

Antianxiety effect usually precedes the antidepressant effect by two or three weeks.

How Supplied–Each capsule contains doxepin, as the hydrochloride. 10 mg, 25 mg, 50 mg, 75 mg and 100 mg capsules in bottles of 100 and 1000.

For complete prescribing information please see package insert or PDR.

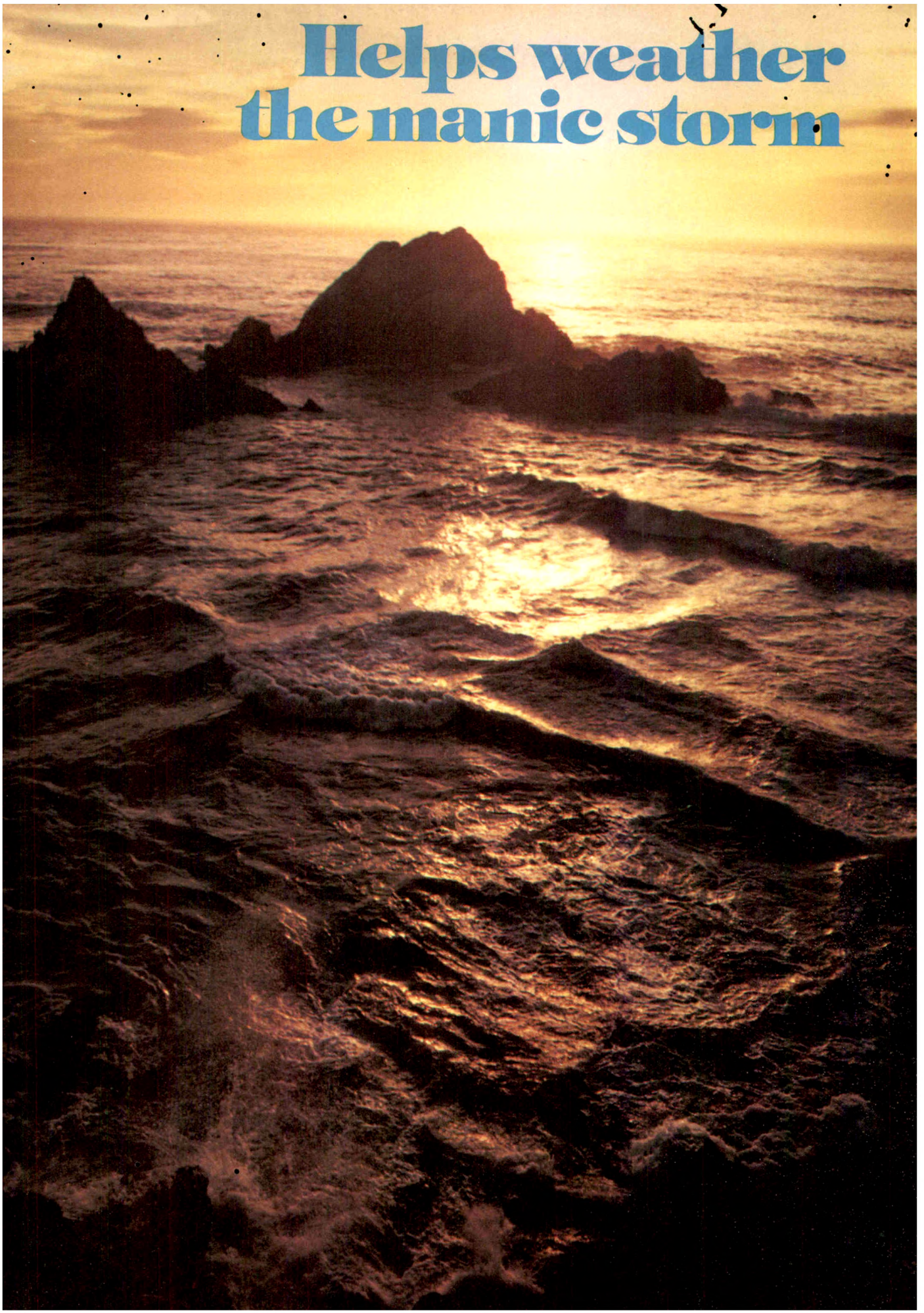
References:

1. Barranco SF, Thrash ML, Hackett E, et al: Early onset of response to doxepin treatment. *J Clin Psychiatry* 40:265-269, 1979 (Sinequan®)
2. Karacan I, Blackburn AB, Thornby JI: The Effect of Doxepin HCl (Sinequan®) on Sleep Patterns and Clinical Symptomatology of Neurotic Depressed Patients with Sleep Disturbance, in Mendels J (ed): *Sinequan®: A monograph of recent clinical studies*, Princeton, NJ, Excerpta Medica, 1977, pp 4-22.
3. Pitts NE: The clinical evaluation of doxepin–A new psychotherapeutic agent. *Psychosomatics* 10:164-171, 1969.

Rx DIVISION
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ROCHESTER, NEW YORK 14623

Adapin® (doxepin HCl) ... effective single-entity drug with antidepressant and antianxiety effects

A-252


Helps weather
the manic storm

maintenance therapy with Eskalith®

300 mg capsules and
300 mg scored tablets

brand of

lithium carbonate

In manic-depressive patients with a history of mania, 'Eskalith'

- prevents or diminishes the intensity of future episodes
- can increase the intervals between episodes
- helps patients function within normal limits

Before prescribing, see complete prescribing information in SK&F literature or PDR. The following is a brief summary.

> **WARNING**
> Lithium toxicity is closely related to serum lithium levels, and can occur at doses close to therapeutic levels. Facilities for prompt and accurate serum lithium determinations should be available before initiating therapy.

INDICATIONS: Treatment of manic episodes of manic-depressive illness. Maintenance therapy prevents or diminishes the intensity of subsequent episodes in manic-depressive patients with a history of mania.

WARNINGS: Lithium should generally not be given to patients with significant renal or cardiovascular disease, severe debilitation or dehydration, sodium depletion, or to patients receiving diuretics.

Lithium may prolong the effects of neuromuscular blocking agents. Such agents should be given with caution to patients receiving lithium. Lithium therapy has been reported in some cases to be associated with morphologic changes in the kidneys. The relationship between such changes and renal function has not been established.

Caution patient and family to watch for diarrhea, vomiting, tremor, mild ataxia, drowsiness, or muscular weakness as signs of lithium toxicity, and to discontinue therapy and contact a physician should they occur. Patients receiving combined therapy with lithium and an antipsychotic should be monitored closely for early evidence of neurologic toxicity and treatment discontinued promptly if such signs appear. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery).

Lithium should not be used in pregnancy, especially during the first trimester, unless potential benefits outweigh possible hazards. Except in rare and unusual circumstances, nursing should not be undertaken while a patient is on lithium therapy, since lithium is excreted in human milk.

Not recommended for children under 12.

PRECAUTIONS: Lithium tolerance is greater during the acute manic phase and decreases when manic symptoms subside.

Lithium therapy may lead to sodium depletion. Normal diet (including salt) and adequate fluid intake (2500-3000 ml.) must be maintained, at least during initial stabilization period. Protracted sweating or diarrhea can decrease tolerance; in such cases, administer supplemental fluid and salt.

Sweating, diarrhea, and concomitant infection with elevated temperatures may require temporary reduction or cessation of dosage.

Where hypothyroidism exists, thyroid function should be monitored during lithium stabilization and maintenance; where hypothyroidism occurs during stabilization and maintenance, supplemental thyroid treatment may be used.

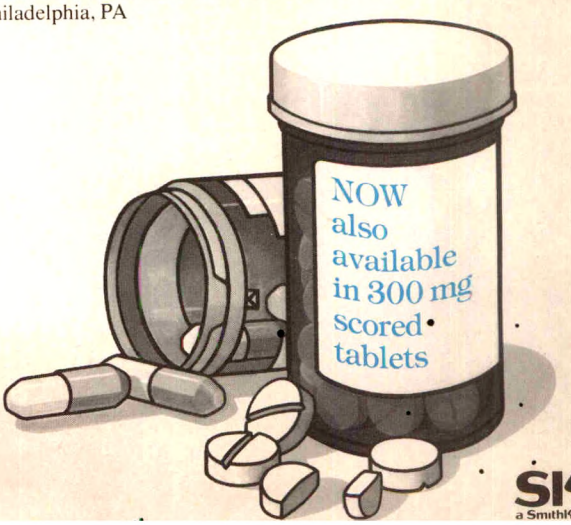
ADVERSE REACTIONS: Mild to moderate toxic reactions may occur at serum lithium levels from 1.5 to 2.5 mEq./1., and moderate to severe reactions at levels from 2.0 to 2.5 mEq./1. Fine hand tremor, polyuria, and mild thirst may occur during initial therapy and persist. Transient and mild nausea and general discomfort may also appear during initial therapy. These effects usually subside with continued treatment or temporary reduction or cessation of dosage. If persistent, discontinue dosage. Diarrhea, vomiting, drowsiness, muscular weakness, and lack of coordination may be early signs of toxicity and may occur at levels below 2.0 mEq./1. At higher levels, ataxia, giddiness, tinnitus, blurred vision, and a large output of dilute urine may be seen. Serum levels above 3.0 mEq./1. may produce a complex clinical picture, involving multiple organs and systems. Serum levels should not exceed 2.0 mEq./1. during acute phase.

The following reactions appear to be related to serum lithium levels, including levels within the therapeutic range: **Neuromuscular**—tremor, muscle hyperirritability (fasciculations, twitching, clonic movements of whole limbs), ataxia, choreo-athetotic movements, hyperactive deep tendon reflex; **Central Nervous System**—blackout spells, epileptiform seizures, slurred speech, dizziness, vertigo, incontinence of urine or feces, somnolence, psychomotor retardation, restlessness, confusion, stupor, coma; **Cardiovascular**—cardiac arrhythmia, hypotension, peripheral circulatory collapse; **Gastrointestinal**—anorexia, nausea, vomiting, diarrhea; **Genitourinary**—albuminuria, oliguria, polyuria, glycosuria; **Dermatologic**—drying and thinning of hair, alopecia, anesthesia of skin, chronic folliculitis, exacerbation of psoriasis, xerosis cutis; **Autonomic**—blurred vision, dry mouth; **Thyroid Abnormalities**—euthyroid goiter and/or hypothyroidism (including myxedema) with lower T_3 and T_4, I^{131} uptake may be elevated; rare cases of hyperthyroidism; **EEG Changes**—diffuse slowing, widening of the frequency spectrum, potentiation and disorganization of background rhythm; **EKG Changes**—reversible flattening, isoelectricity or inversion of T-waves; **Miscellaneous**—fatigue, lethargy, transient scotomata, dehydration, weight loss, tendency to sleep.

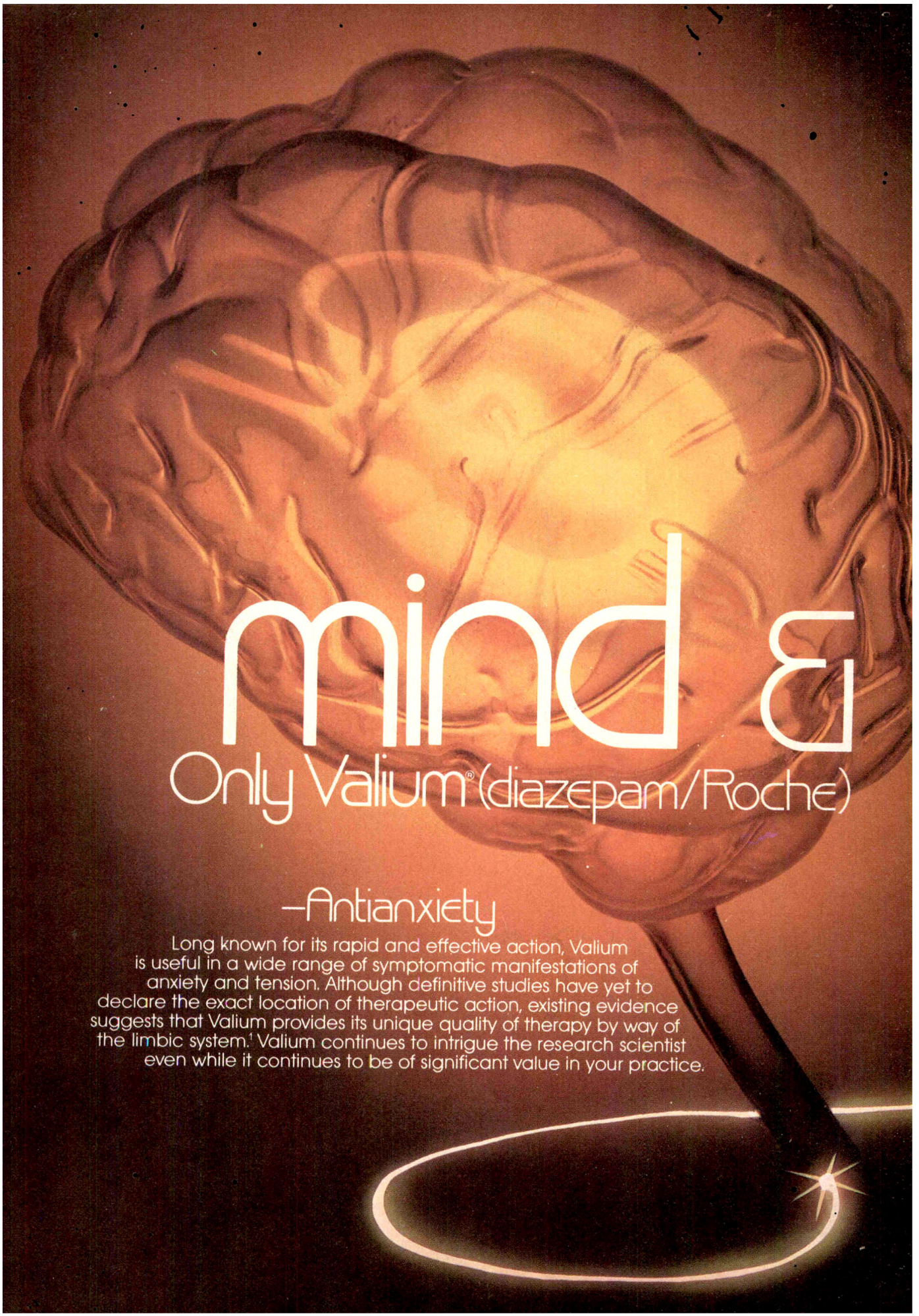
Reactions unrelated to dosage include: transient EEG and EKG changes, leukocytosis, headache, diffuse nontoxic goiter with or without hypothyroidism, transient hyperglycemia, generalized pruritus with or without rash, cutaneous ulcers, albuminuria, worsening of organic brain syndromes, excessive weight gain, edematous swelling of ankles or wrists, thirst or polyuria, sometimes resembling diabetes insipidus, and metallic taste. A single case of a syndrome resembling Raynaud's has been reported.

HOW SUPPLIED: 300 mg. capsules in bottles of 100.
300 mg. scored tablets in bottles of 100.

Smith Kline &French Laboratories
Philadelphia, PA



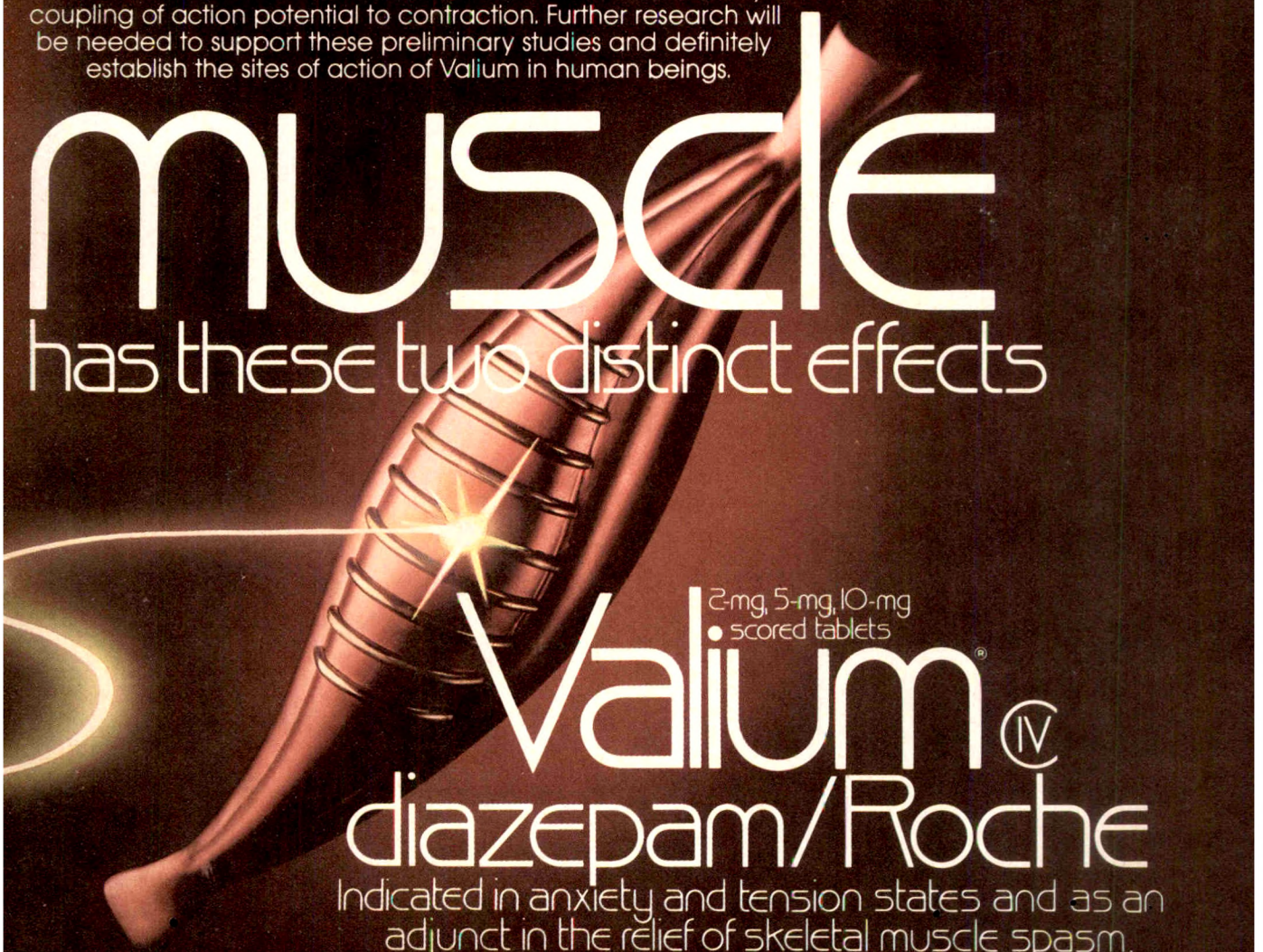
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Only Valium® (diazepam/Roche)
–Antianxiety
Long known for its rapid and effective action, Valium is useful in a wide range of symptomatic manifestations of anxiety and tension. Although definitive studies have yet to declare the exact location of therapeutic action, existing evidence suggests that Valium provides its unique quality of therapy by way of the limbic system.[1] Valium continues to intrigue the research scientist even while it continues to be of significant value in your practice.

–Skeletal muscle relaxant

In addition to producing muscle relaxation by action at the cerebral and spinal levels,[2] Valium may exert direct action on the muscle itself. Experiments have demonstrated that, at a concentration of 10^{-6}M to 10^{-5}M, diazepam directly counteracts contraction in the fast skeletal muscle of the frog.[3] The resulting decrease in muscle contraction is attributed to a net loss of muscle calcium, which is necessary for efficient coupling of action potential to contraction. Further research will be needed to support these preliminary studies and definitely establish the sites of action of Valium in human beings.

muscle has these two distinct effects



2-mg, 5-mg, 10-mg scored tablets

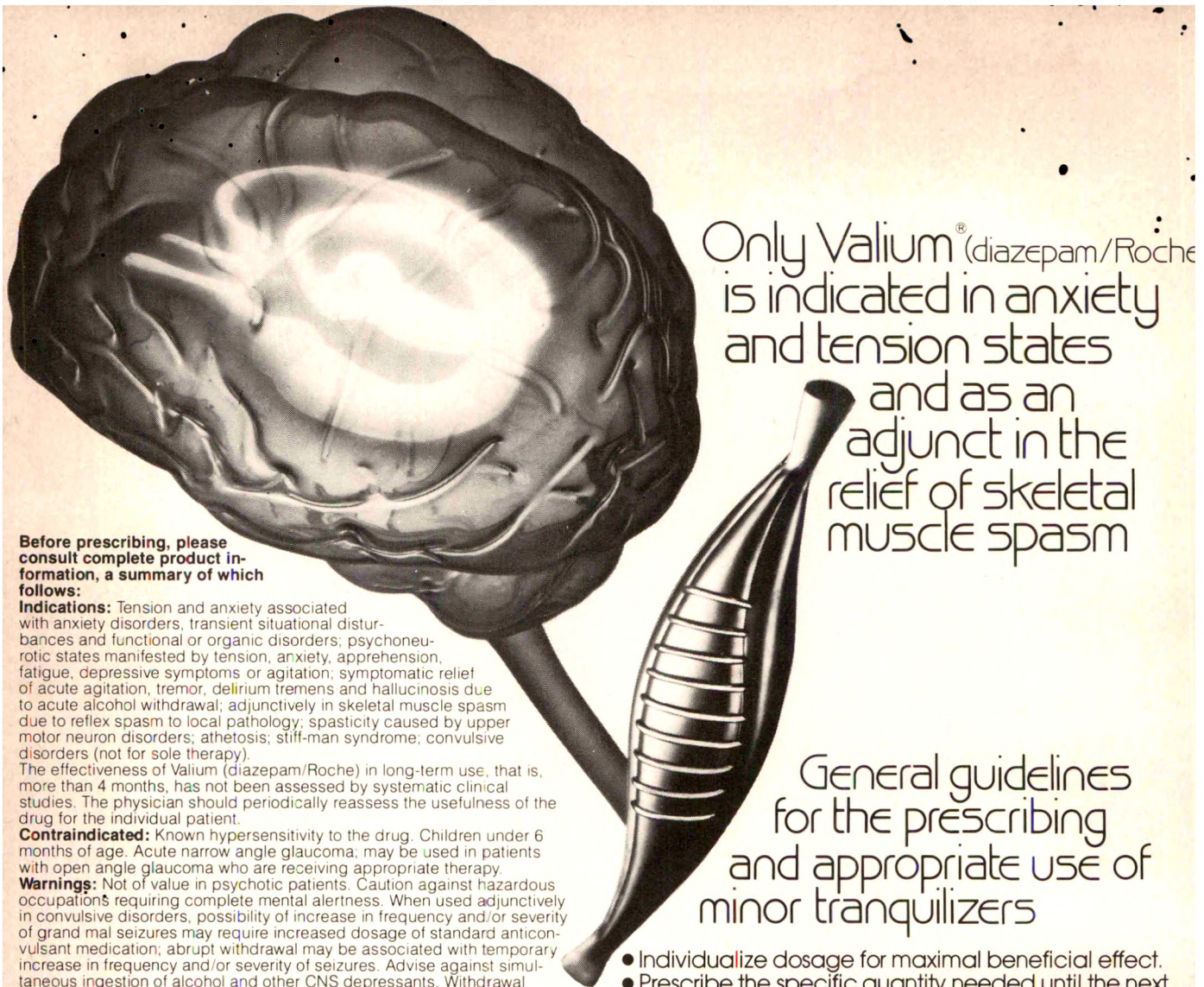
Valium® (IV)

diazepam/Roche

Indicated in anxiety and tension states and as an adjunct in the relief of skeletal muscle spasm.

Please see summary of product information on following page.

ROCHE®



Only Valium® (diazepam/Roche) is indicated in anxiety and tension states and as an adjunct in the relief of skeletal muscle spasm

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety associated with anxiety disorders, transient situational disturbances and functional or organic disorders; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).
The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

Supplied: Valium® Tablets, 2 mg, 5 mg and 10 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available in trays of 10.

General guidelines for the prescribing and appropriate use of minor tranquilizers

- Individualize dosage for maximal beneficial effect.
- Prescribe the specific quantity needed until the next checkup period, schedule frequent, periodic reexaminations to monitor results of therapy.
- Establish treatment goals and gradually discontinue medication when these have been met.
- Avoid prescribing for individuals who appear dependency-prone or whose histories indicate the potential for misuse of psychoactive substances, including alcohol.
- Caution patients against engaging in hazardous occupations requiring complete mental alertness such as operating machinery or driving.
- Advise patients against the ingestion of alcoholic beverages while undergoing therapy with minor tranquilizers.
- Counsel patients to follow label directions, keep medication out of children's reach, and dispose of unused or old medication.
- Caution patients against giving medication to others.
- Avoid abrupt cessation of extended therapy by tapering dosage before discontinuing medication.

1. Brazier MAB, Crandall PH, Walsh GO: *Exp Neurol 51*:241-258, Apr 1976
2. Naftchi NE, Lowman EW: The effect of diazepam on presynaptic inhibition. Scientific exhibit at the meeting of the American Academy of Family Physicians, Las Vegas, Nev, Oct 10-13, 1977
3. deGroof RC, Bianchi CP: *Fed Proc 37*:787, 1978

ROCHE®
Roche Laboratories
Division of Hoffmann-La Roche Inc.
Nutley, New Jersey 07110

Before prescribing or administering, see Sandoz literature for full product information. The following is a brief summary.

Contraindications: Severe central nervous system depression, comatose states from any cause, hypertensive or hypotensive heart disease of extreme degree.

Warnings: Administer cautiously to patients who have previously exhibited a hypersensitivity reaction (e.g., blood dyscrasias, jaundice) to phenothiazines. Phenothiazines are capable of potentiating central nervous system depressants (e.g., anesthetics, opiates, alcohol, etc.) as well as atropine and phosphorus insecticides; carefully consider benefit versus risk in less severe disorders. During pregnancy, administer only when the potential benefits exceed the possible risks to mother and fetus.

Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy, observed primarily in patients receiving larger than recommended doses, is characterized by diminution of visual acuity, brownish coloring of vision, and impairment of night vision; the possibility of its occurrence may be reduced by remaining within recommended dosage limits. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving), and increase dosage gradually. Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension since phenothiazines may induce a reversed epinephrine effect on occasion. Daily doses in excess of 300 mg should be used only in severe neuropsychiatric conditions.

Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; rarely, nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—ECG changes (see *Cardiovascular Effects* below). *Other*—Rare cases described as parotid swelling.

It should be noted that efficacy, indications and untoward effects have varied with the different phenothiazines. It has been reported that old age lowers the tolerance for phenothiazines; the most common neurologic side effects are parkinsonism and akathisia, and the risk of agranulocytosis and leukopenia increases. The following reactions have occurred with phenothiazines and should be considered whenever one of these drugs is used. *Autonomic Reactions*—Miosis, obstipation, anorexia, paralytic ileus. *Cutaneous Reactions*—Erythema, exfoliative dermatitis, contact dermatitis. *Blood Dyscrasias*—Agranulocytosis, leukopenia, eosinophilia, thrombocytopenia, anemia, aplastic anemia, pancytopenia. *Allergic Reactions*—Fever, laryngeal edema, angioneurotic edema, asthma. *Hepatotoxicity*—Jaundice, biliary stasis. *Cardiovascular Effects*—Changes in terminal portion of electrocardiogram, including prolongation of Q-T interval, lowering and inversion of T-wave, and appearance of a wave tentatively identified as a bifid T or a U wave have been observed with phenothiazines, including Mellaril (thioridazine); these appear to be reversible and due to altered repolarization, not myocardial damage. While there is no evidence of a causal relationship between these changes and significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients showing characteristic electrocardiographic changes while taking the drug. While proposed, periodic electrocardiograms are not regarded as predictive. Hypotension, rarely resulting in cardiac arrest. *Extrapyramidal Symptoms*—Akathisia, agitation, motor restlessness, dystonic reactions, trismus, torticollis, opisthotonus, oculogyric crises, tremor, muscular rigidity, and akinesia. *Persistent Tardive Dyskinesia*—Persistent and sometimes irreversible tardive dyskinesia, characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and sometimes of extremities may occur on long-term therapy or after discontinuation of therapy, the risk being greater in elderly patients on high-dose therapy, especially females; if symptoms appear, discontinue all antipsychotic agents. Syndrome may be masked if treatment is reinstituted, dosage is increased, or antipsychotic agent is switched. Fine vermicular movements of tongue may be an early sign, and syndrome may not develop if medication is stopped at that time. *Endocrine Disturbances*—Menstrual irregularities, altered libido, gynecomastia, lactation, weight gain, edema, false positive pregnancy tests. *Urinary Disturbances*—Retention, incontinence. *Others*—Hyperpyrexia; behavioral effects suggestive of a paradoxical reaction, including excitement, bizarre dreams, aggravation of psychoses, and toxic confusional states; following long-term treatment, a peculiar skin-eye syndrome marked by progressive pigmentation of skin or conjunctiva and/or accompanied by discoloration of exposed sclera and cornea; stellate or irregular opacities of anterior lens and cornea; systemic lupus erythematosus-like syndrome.

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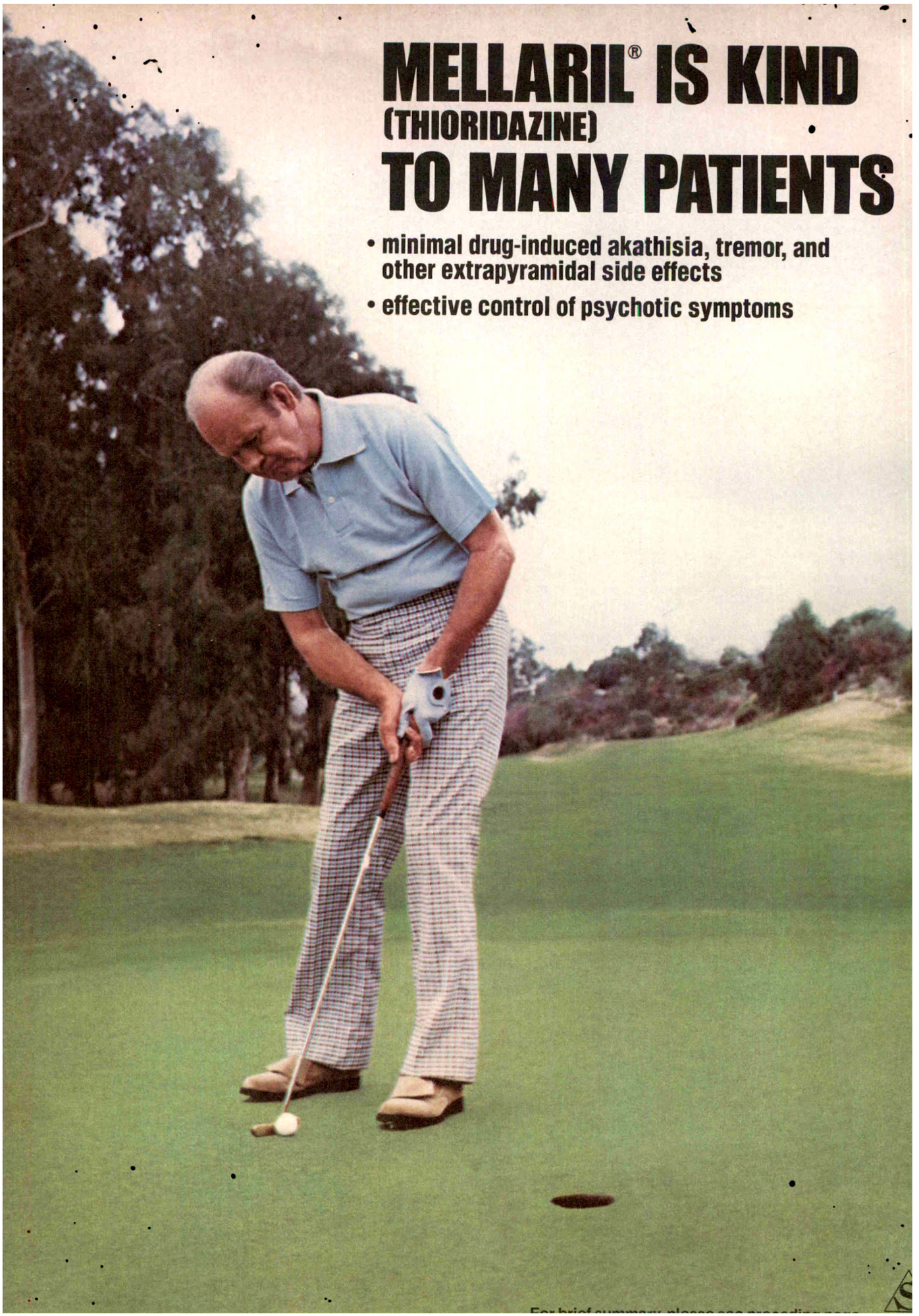
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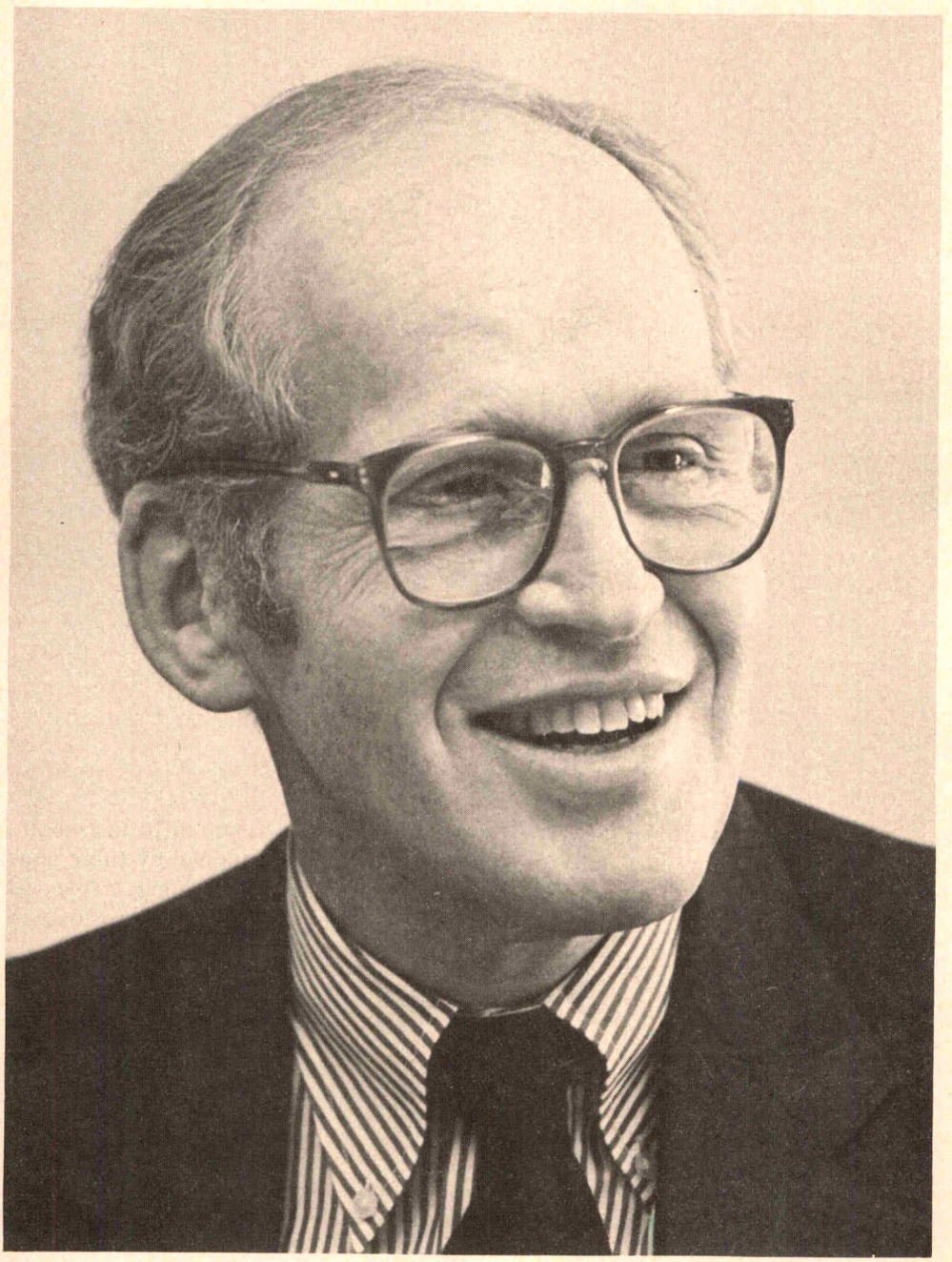

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ALAN A. STONE, M.D.

THE AMERICAN JOURNAL OF PSYCHIATRY.

Presidential Address: Conceptual Ambiguity and Morality in Modern Psychiatry

BY ALAN A. STONE, M.D.

Last year in my response to the Presidential Address I rather presumptuously associated myself with the wisdom of the owl of Minerva—the owl that Hegel said only spreads its wings at dusk. From that lofty position I described a new era in psychiatry that I called pragmatic eclecticism, an era in which the four competing models in psychiatry had learned peaceful coexistence. These models—the biological, the psychodynamic, the behavioral, and the social—I suggested were held together by the medical model, which provided clinical coherence if not conceptual clarity. As I present the Presidential Address I must confess that the wings of the owl of Minerva were not fully extended last year. I recant nothing, but I now believe I glided too easily over that qualifying phrase about the absence of conceptual clarity. I intend today therefore to speak about the ambiguity of our eclectic medical model and my concern that having overcome the tyranny of narrow orthodoxy we are now in danger of retreating behind new walls. I shall touch briefly on subjects familiar to you all: racism, homosexuality, and the situation of women. I shall not attempt to deal in a deep way with the substance of these matters; rather, my intention is to examine how American psychiatry has grappled with them. These are all issues which have confronted us in our practice, challenged the moral assumptions that lie concealed in our theories, and confounded us with disputes and acrimony in our Association. It is as we attempt to deal with this kind of issue that the new walls are being built, and it is no accident that each invites psychiatry to take a stand on human values. Human values after all are a crucial link in the chain that binds the self to society. To take a stand on them reveals something about our own selves, our own relation to society, and our own vision of what it means to love and to work.

Many psychiatrists believe that our Association should limit itself to issues that are clearly psychiatric, and they look back at some of the strong positions we have taken on what they consider social issues with regret. Many other psychiatrists believe these social issues are clearly psychiatric; they are proud of what we have done and urge us to do more. I shall claim that what separates these two groups can be understood only as part of the deeper theoretical dilemma in which American psychiatry finds itself, its lack of conceptual clarity. I know I shall be addressing a subject in public that most of us prefer to talk about in private, but my temerity is sustained by three considerations. First, our professional training obliges us to understand conflict rather than repress or deny it, and I believe that in view of recent events the time has come to confront this conflict openly. Second, I have become convinced that the basic limitation of pragmatic eclecticism is that it creates and sustains a mood of caution and expediency in all things, including matters that require moral ambition. Thus, perhaps without thinking it through, we are losing the courage of conscience. Finally, I do not intend to suggest an ultimate solution. I mean only to describe the problem as I encounter it in my own heart and mind.

THE PARABLE OF THE BLACK SERGEANT

I turn to what I have called the parable of the black sergeant. It is a parable about racism, about guilt and forgiveness, and about psychiatric theory and practice. The identifying information in this parable-case

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history has been disguised for reasons of confidentiality, but the value conflicts it presents are real enough. The patient, a black man, was a supply sergeant. He was caught by the military police stealing a deodorant stick from the post exchange. The Army, which had reason to suspect the sergeant of other thefts and was undeterred by Constitutional restraints on search and seizure, went to his home and reclaimed every piece of Army property that the sergeant could not account for. The pile of supplies—uniforms, blankets, picks and shovels, cartons of canned goods, mess kits, etc.—could have filled a trailer truck. It had all been photographed on the sergeant's front lawn, and that picture became part of his Army medical file.

The Army was determined to court-martial the sergeant, but he had been examined by a civilian psychiatrist who decided that much of what was stolen was of no use to the sergeant and, based on his understanding of the sergeant's psychodynamics, had diagnosed him as a kleptomaniac. This civilian psychiatrist was prepared to testify at court-martial that the stealing was due to unconscious and irresistible impulses. Unhappy with the civilian psychiatrist's report, the Army sent the sergeant to be evaluated at an Army hospital. There he was told repeatedly that anything he said could be used against him at the court-martial. The sergeant took the warning rather impassively, and his Army psychiatrist set to work to construct a very detailed anamnesis over the course of three weeks.

The sergeant, who was a very intelligent man, got caught up in telling the story of his life. He had grown up in a Southern city during the days of racial segregation. A good and serious student from a deeply religious family, he had done well in school and had gone to a small college where he studied literature. After graduation, despite his hopes and dreams, he had found no appropriate work and eventually was drafted into the Korean War. After the war, seeing no alternatives, he remained in the Army to serve his 20 years. As the years passed he became increasingly bitter. He was convinced that life had cheated him because he was black and that the Army, in the work and position it gave him, continued to discriminate against him. Out of this sense of being cheated there grew a sense of entitlement, and he came to feel that he was justified in taking whatever he could whenever he could. He had no sense of being impulsively driven to steal Army property; instead, he stole with a sense of entitlement and reparation in protest of the racist world that had deprived him of his hopes.

It is not clear why this black supply sergeant, despite being warned, told all this to the Army psychiatrist. At any rate he did, and the Army psychiatrist, after puzzling over the diagnostic possibilities (which included paranoid personality and depression), concluded that the sergeant did not have kleptomania or any other mental disorder that should excuse him from responsibility. Subsequently the Army psychiatrist testified at the court-martial. He told the story I have told you while trying to avoid the sergeant's eyes. The sergeant sat there in his dress uniform with his medals, his wife, and their small children. He was sentenced to five years at hard labor. As you may have guessed, I was the Army psychiatrist, and I felt something terrible happened that day. Each time my mind takes me back to that occasion I have a sense of dismay that will not be dissipated.

The Influence of Morality and History

It is tempting when one reflects on this parable of the black sergeant to conclude that it demonstrates no more than the abyss between the moral concerns of the law and the therapeutic concerns of psychiatry. But none of us, I trust, would claim that if a man like the sergeant came voluntarily seeking treatment his psychiatrist would at no point consider moral issues. And how could one treat such a man with no moral perspective and revealing no opinion about racism? Surely a psychiatrist treating the sergeant in 1980 would approach his problems differently than our predecessors would have done in 1940. Psychiatry does not stand outside history or morality, but how do we decide which history and which morality to accept? Is it all a matter of individual choice? Certainly one cannot find the rules controlling what history or morality to accept clearly delineated in the major theories of our biological, psychodynamic, and behavioral paradigms. Psychiatrists are taught to avoid value judgments in their dealings with patients, but I do not believe I make a radical claim when I assert that history and morality are a presence in the therapist's office. The only question is how do they get there.

Let me turn to a narrower question: What role does our practice and our theory play in the courtroom drama of guilt and forgiveness? The legal system, even the Army's legal system, was hesitant to assess guilt in the face of a diagnosis of kleptomania. That diagnosis and the account of human behavior that goes with it invoke the unconscious and the symbolic meaning of the theft. Those who sit in judgment are not asked to consider the sergeant's race and social identity. His behavior is presented in isolation from its historical and cultural context. His kleptomania is understood in the confines of a personality development that repeats a timeless sequence outside culture and history. The psychiatrist applying this kind of theory comprehends the sergeant's motives as nonutilitarian and not intended to offend the system. Such a man can be forgiven; his quarrel is not with us but with his introjects.

On the other hand, the account I gave takes the man as he experiences his life within a particular culture and history. His stealing is understood as part of his response to his predicament. It is his revenge against an unjust system. Such a man cannot be forgiven by our law, but if his judges are willing to condemn the

racism of their society they can lighten his sentence. And if the psychiatrist is willing to lend his authority to the condemnation of a racist society he might even help the judges to reach that lighter sentence. But the moral and historical perspective that might have led me in that direction was not a presence in my office when I examined the sergeant. If in some sense I betrayed the sergeant, and I believe I did, it was because of the historical and moral perspective I brought to the subject of racism.

Psychodynamic Versus Social Considerations

I have carefully distinguished the psychodynamic and the social account of the sergeant's behavior, not only because that is how the two psychiatrists presented them but also to emphasize the radically different moral implications and consequences of the two accounts. There is an irony here that should be underscored. The theory that excludes history and morality has the power to exculpate without disturbing the status quo. Thus the psychiatrist's choice of theory becomes crucial. I shall return to this question of choice. Many of you must be impatient with what seems an artificial distinction because as eclectics we are all accustomed to invoking both accounts. Indeed, the word "psychosocial" seems to have been invented to make this point. Most of us are prepared to believe that there was probably some truth in both accounts of the sergeant's behavior and to insist that anyone who hopes to understand the sergeant must give weight to both, not to mention biological and behavioral factors that are missing from this 20-year-old parable.

Here we touch on the greatest strength of eclecticism and its greatest weakness. Its strength lies in its openness to consider the complexity of the forces that touch our lives. The eclectic psychiatrist sees biological man, moved by dynamic passions and ideals, shaped by culture and shaping culture. The eclectic has the most comprehensive view of what Sartre called the "critical mirror which alone offers man his image." But still that image is only a reflection; even less than a reflection, it is a composite sketch pieced together from the accounts of different witnesses whose vision depends on different theoretical perspectives. It is not just the problem of getting a biological, a psychodynamic, a behavioral, and a social psychiatrist to agree among themselves. How does the individual eclectic psychiatrist who conscientiously considers all of these factors reach a judgment about their relative significance?

This is our greatest weakness. We do not know what weight to give each perspective's account. We can diligently catalogue the different accounts. We can artfully construct Venn diagrams to suggest the possible relationships of these accounts. We can in rare instances assert with confidence the primacy of a particular account. But most often we are condemned to the ultimate ambiguity that is the inevitable consequence of our eclecticism. This is not a problem to be resolved by mere numbers; we do not have a formula or a way of deciding how much something should count. We psychiatrists often say that behavior is overdetermined, but might it be that this concept of overdetermination conceals our very deepest confusion, our inability to order the different considerations? As clinicians each of us treats the whole person, and each day we try to piece together the composite sketch. But we also have a pressing responsibility to ease human suffering. Compassion draws us inevitably to expedient remedies. As pragmatic eclectics uncertain that we have put the pieces of the picture together correctly, we can never be confident that we can distinguish between the sick patient and the sick society.

Lacking that ability and given that it is easier to apply expedient remedies to sick patients than to sick societies, will we not inevitably be led by our clinical responsibilities to distort the composite sketch of the human condition? At the very least we should understand that we might seem to the outside world to be doing just that.

Sensibility at the Cost of Ambiguity[1]

Here is a second irony to be underscored: it was only when our generation of psychiatrists had the courage to overcome the walls of narrow orthodoxy that we confronted our greatest weakness. Only within the narrow paradigms could we construct if not a causal account at least something close to it. When we ventured outside we purchased sensibility at the cost of ambiguity. This theoretical ambiguity is at the very core of the conflicts that confound us. When we testify in court, when we formulate our diagnostic nosology, when we issue position papers, indeed, whenever we attempt to act, we confront this ambiguity. It is no wonder, then, that many psychiatrists are now eager to go back to their narrow paradigms, where they hope to capture the rigor of conceptual clarity that comforts the scientist and to escape from the complexity of human values that accompany a greater sensibility.

Biologists, psychoanalysts, and behaviorists can at least imagine this alternative, but social psychiatrists have no place to hide, no way to narrow their sensibilities. They have always stood outside the walls, urging their colleagues to broaden their horizons. It might even be possible to describe social psychiatry as the fractious adolescent member of the psychiatric family, disrupting the serenity of the household by arguing about values, morality, and social justice, the way all adolescents do. Shall we now expel them? Even if we could, I think it is too late. The broad sensibility they brought to psychiatry has opened eyes that will not be closed.

What I have described thus far is an honest picture

[1]I wish to acknowledge my colleague Roberto Mangabeira Unger, whose ideas were helpful in the formulation of this section.

of my own perplexity, if not of yours. First, I know that I do not abandon history, morality, and human values when I enter my office, but I do not know how I decide what to take in with me. I believe that most of you are in the same position, and for reasons I shall come to shortly I am deeply suspicious of anyone who makes the contrary claim that history, morality, and human values are all irrelevant to psychiatry. Second, I know that I at least am aware of no rules of ordering the different paradigms and their interactions. When I sat in judgment of the black sergeant, I believed that the hard truth of the matter was to be decided by choosing the particular paradigm which best fit the facts of the case. Having chosen the correct paradigm, I applied the explanatory theory of that paradigm and thus never experienced a lack of conceptual clarity. In a sense the four paradigms provided me a framework of differential diagnosis within which I proceeded rigorously, with the skill and objectivity of a scientist. My recognition that the goal of enlightened eclectics like Adolf Meyer was not to choose but to reconcile came when I began to examine the scientific problem of prediction in psychiatry and the moral consequences of my professional activity. Is it possible that our inability to reconcile our paradigms stands in the way of both our scientific and our moral progress?

Thus far I have drawn a picture in which most of psychiatry might seem to stand to one side on the crucial questions of morality and human values, with only social psychiatry on the other side demanding that we get involved in the real world. But during this century all of science has learned painfully and tragically that science is not remote. Every thinking person knows that the physicist's equations play a crucial role in the destiny of humanity. Psychiatry's role is also crucial, although its influence is much more subtle. One of the most remarkable developments in this century is the speed with which the psychiatrist's theories of human nature move from the arcane jargon of our journals through the mass media and into the consciousness of our culture. Psychiatry has played no small part in the transformation of the mind of modern man. This transformation takes place outside our control; theories are bowdlerized and sensationalized. Our drugs are prescribed and self-prescribed everywhere. Other treatment methods have also been coopted and arrayed in the struggles of interpersonal politics. Young people make love after they make therapy. There is in all the affairs of daily living, public and private, the pervasive if subtle influence of our profession.

The most powerful aspect of psychiatry is its contribution to what it means to be a person. This is not under our control, nor can it be in a free society. But we do bear a certain responsibility, and one of the themes in that responsibility is the hidden values in the theories and therapies that originated with us and contribute to the shaping of contemporary consciousness.

The thrust of this analysis is to claim that even without social psychiatry we are not on the other side. We have been engaged in an enterprise that involves concealed positions on human values, moral postures, and even politics. This claim comes not just from unfriendly critics, it comes from responsible colleagues. This is the indictment that confronts those psychiatrists who assert that their psychiatry has nothing to do with these things; although the indictment can be overdrawn and viciously expressed, the fundamental truth in it cannot be gainsaid. Therefore, given the power of our enterprise, whether we like it or not we are in some measure responsible for the influence of these hidden values. It is also important to remember that many of us have wanted to use psychiatry to influence the public to confront and even to treat the sick society through the media. It is not just a matter of aloof scientists being victimized by the vulgarity of the mass media.

HOMOSEXUALITY

One of the first great battlefields in the attack on psychiatry's hidden values was homosexuality. Psychiatrists had long assumed that as part of their humanistic tradition they had brought their scientific perspective to things that were once considered evil. Homosexuality became sickness rather than sin, and this perspective in this century was accepted not only by the secular masses but even by most religious authorities. However, gay liberation brought a different perspective. Their argument was that our judgments about homosexuality as sickness contained hidden values, a limited vision of human sensuality and intimacy, the old morality under a new guise, and perhaps even our own phobic limitations. A campaign was undertaken to remove the diagnosis of homosexuality from the nomenclature. Our Association, after considerable deliberation and not a little acrimony, accepted that perspective. Our Association went even further—it called for an end to legal discrimination against homosexuality. Here I come to the heart of the matter—the relationship of our lack of conceptual clarity to the actions we take. Recall the analysis I have developed here. Each of the four paradigms in psychiatry inevitably had its own theories about homosexuality.

Biological explanations can be sought in the endocrine system, psychodynamic explanations in childhood experiences and the Oedipus complex. Behaviorists can find answers in the sexual gratification reinforcement schedule. The social psychiatrist has the only paradigm that directly addresses the basic issues raised by gay liberation. Imagine if you will the eclectic psychiatrist who accepts all four paradigms but does not know how to reconcile them. Imagine such a psychiatrist who uses the four paradigms as a kind of differential diagnosis. Might that psychiatrist not conclude that there were four different kinds of homosex-

uality and that only the kind described by social psychiatry had been removed from our diagnostic manual?

This analysis may seem far-fetched and bizarre to you, but let me emphasize the structure of my argument. If we have no overarching theory that organizes the theories of the four basic paradigms, those paradigms cannot be changed from the top down, at least not by the logic of a controlling theory. Thus it is possible to argue that for many psychiatrists no fundamental change in their basic approach had occurred. They could go on as before. Therefore, the struggle for those concerned about gay liberation had to continue at a second level, which has two directions.

First, would psychiatry invest more in the effort to influence the public perception of homosexuality? Second, would each of the paradigms undertake the necessary theoretical reformulations essential to a fundamental change? My analysis in no way is meant to demean the decisions our Association reached. Nor do I minimize the importance of what we did. It was not an empty gesture. But I believe the real significance of our actions once again was moral. We changed the moral element in our composite sketch of homosexuality. I understand it as an act of moral compassion producing a small change in public perception. That small change in the long run can have a powerful effect on the most insular theory.

WOMEN'S ISSUES

Perhaps the most penetrating and convincing attack on the hidden and destructive values in psychiatry has related to the subject of women. Indeed, when one has the occasion to read the standard works in psychiatry written before 1970 it no longer seems appropriate to talk about hidden values. It is no wonder, then, that psychiatrists who are concerned about the quality of the composite sketch of women promulgated by their profession and assimilated into our culture should turn to this Association to rectify it. There was obviously a great deal to be rectified, but they confronted an enormous obstacle. There was no diagnosis of female that could be expunged from the diagnostic manual; thus there could be no obvious battlefield. The struggle had to begin at the second level. How far would our Association go in its efforts to rectify the public's perception of women, and how thoroughgoing would be the necessary changes in the theoretical structure of each of the paradigms?

Here I come to the last piece in the structure of my analysis of how American psychiatry deals with these issues, that is, the effect of our own psychology and our own historical situation. As far as I can see, the case against psychiatry as regards women is far more damaging, requires far more than a minor adjustment of our composite sketch, indeed compels each of us to reexamine not only our theories and therapy but also our own lives and relationships. One might even say that psychiatry has only recently discovered that the maxim to love and to work applies to women.

Is it possible that the issues raised concerning women cut deeper in American psychiatry than even racism and homosexuality? That the questions raised in connection with women touch our personal as well as our professional identity? There can be no new psychology of women that does not require a new psychology of men. That makes necessary a new conception of all our human values and all the paradigms of psychiatry. This challenge comes at a time when our profession is struggling—when we have trimmed our sails and yearn for the safety of calmer waters. It comes at a time when many would like to retreat to their narrow paradigm. It comes at a time when the spirit of pragmatic expedience dominates our profession, narrows our horizons, and saps our moral courage.

Most psychiatrists are not by temperament polemical or adversarial. We do not seek out moral controversy. Most of us spend our lives listening compassionately to people who are suffering, whose lives are disrupted by the uncontrolled demands of passion and by the pains of helplessness. We try to be nonjudgmental while we help them weave together the delicate fabric of personal strength that sustains love and intimacy. And we sustain ourselves by small triumphs and acts of private altruism. Our successes often go unnoticed and our failures loom large. Ours is a vulnerable profession, easy to attack and to belittle, but it is a noble profession because it is both a moral and a scientific enterprise.

When we move from the safety of our office to take action in the real world we usually are motivated by the same moral enterprise that guides us in our office: a mixture of compassion, understanding, art, and science. The world outside our office may seem increasingly treacherous. But that treacherous world is already inside our office, if only in microcosm, and our work can never be carried on in a moral and historical vacuum. If like Hercules we were to succeed in lifting our giant of a science from its earthly connections, if we were to cut away all the links that hold self to society, then psychiatry would become a corpse. We will make mistakes if we go forward, but doing nothing can be the worst mistake. What is required of us is moral ambition. Until our composite sketch becomes a true portrait of humanity we must live with our uncertainty; we will grope, we will struggle, and our compassion may be our only guide and comfort.

Am J Psychiatry 137:8, August 1980

Response to the Presidential Address

BY DONALD G. LANGSLEY, M.D.

President Stone has set the theme of human values for this meeting, citing the criteria for maturity suggested by Freud in "Civilization and Its Discontents" (1). In these times, when all of the health professions are changing rapidly, we would do well to review the values of psychiatrists and to place them in the context of changing values in society and government. Let us start with Aristotle, who repeatedly used medicine as a model for the study of values in his Nichomachean and Eudemian Ethics (2). He did so because medicine deals with both life and education. Aristotle divided virtues (called *arete*) into those of the intellect (called *sophia*) and those of the soul (the psyche). How have we psychiatrists fared since Aristotle?

Our medical values are often traced to the Hippocratic tradition, which focused on the responsibility of each physician for his or her patient. Today we expand those concerns to a broad variety of issues affecting health. We have seen a shift from the Flexner tradition, in which science formed the most important base of medicine, to a variety of concerns about ethical, moral, and social issues. As a profession, we are increasingly concerned with social justice. In our roles both as psychiatrists and as citizens, we have come to realize that social action and health are of one piece. We must make that point convincingly when we consider the effect of the social role of women on their mental health.

The values of psychiatry are partially expressed in its ethical statements, in which our usual codes are rules rather than laws. Some cannot be enforced. The law guards against the grosser violations of human rights, but ethics set an ideal higher than law can guarantee. What is legal is not always ethical. Professional ethics must transcend the law for the larger human good.

Robert Moore (3) has reminded us of the sources of medical codes and values. They derive from prayers and oaths written long before the birth of Christ. The Hippocratic Oath from the fourth century B.C. expressed both a convenant among physicians and a set of principles about patient care. With the Middle Ages came the Prayer of Maimonides and a set of Admonita and Epistulae; the Renaissance contributed the Medicus Politicus; and during the eighteenth century a variety of treatises on the duties and character of physicians further amplified the values of the physician in his professional and personal life. Similar codes were suggested by our Chinese colleagues as far back as the sixth century, when Sun Ssu-miao, the father of Chinese medicine, expressed such values in his book *The Thousand Golden Remedies* and again, in the seventeenth century, when Chen Shih Kung proposed 5 commandments and 10 requirements for physicians. The healing professions in all cultures have developed codes of values as they became invested with privileges by their particular society.

Before we ascribe too much to these external codes, we must remind ourselves of the deeper sources of values and the history of their development in the individual. Freud's concepts of unconscious determinism and of a superego and ego shook the world's assumptions about rationality and free will. Our values are rooted at unconscious as well as conscious levels. They are influenced by the experiences of our earliest years of life and are not readily changed by external codes.

It is not only professional codes that influence values. Government has played an increasing role. Health has become a responsibility of government. Governments make life-or-death decisions—even to the giving of life through health care or the withholding of it. Through a series of decisions more often based on economics than on social values, government is in the business of choosing among lives. Because health has become such an enormously costly affair, we see the increasing conflict of values among patients and physicians and those who pay—the government and insurance companies. This complicates the life and work of the physician, who used to say, "I am only concerned with preserving life." Just as we have been cautioned about the power and influence of the military-industrial complex, so should we be mindful of the power and influence of the health care industry, now the second largest in our total economy.

The mass media have had a significant influence on values related to health. Sad to say, the public knows Trapper John and Hawkeye far better than it knows Sir William Osler. The media have fostered the

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notion that there is a medicine for every problem, and they have enhanced the demand for high-technology tertiary care medicine. Television doctors cause dissatisfaction in viewers who do not receive the same degree of personal satisfaction from their own physicians. Yet little of this display is for the benefit of the public—media programs are generated to sell pills and other treatments for profit.

Our ethical values also move in the direction of libertarian ideals, a right to health care and the equally important right to refuse. We are concerned that patients have the right to set their own goals and not simply to accept those of the psychiatrist. Many people do not wish to change from behavior that the profession might choose to define as pathological. Do we, then, have the right to define illness with the purpose of insisting that everyone achieve something we call normality or submit to psychotherapy, drugs, or surgery? We are traditionally concerned with privacy and confidentiality. Patients need these in order to develop the trust that makes psychotherapy possible. But at the same time, we are reminded that the public must be protected. In some jurisdictions we are required to warn potential victims or suffer serious consequences.

RESPECT FOR HUMAN LIFE

As physicians we remind ourselves that respect for human life is the basic moral obligation binding our professional decisions. In those circumstances and in this scientific era when we seem on the verge of creating biologic structure, we are entitled to ask what life is and when it begins and ends. We are concerned about the beginnings of what can be an independent life when we consider abortion, just as we are concerned about the termination of life when we consider brain death. We discuss issues of prolonging life through new technologies and treatments, including transplants, chronic dialysis, and new medications, but we need to ask whether this prolongs life in a happy and productive fashion or simply prolongs the existence of certain cells and tissues. We ourselves ask for demonstrations of the efficacy and safety of our treatments, but it is just as often that government and others who pay demand those assurances.

In a sense, the psychiatrist is more sophisticated about motivation and individual values than his or her fellow physicians are. Although we are primarily concerned about advocacy for the patient, his needs as well as his rights, a respect for his autonomy, and the need to obtain his consent, we recognize that the competence to give consent is not a simple or rational process. We among all professions are aware of the irrational bases for making decisions. We know about unconscious reasons for decisions, and of the influences of transference on feeling, thought, and behavior. Is consent given without persuasion or influence and because of rational knowledge, as the courts require? That proposition assumes a different human being than the psychiatrist knows. The physician himself is a major influence. Indeed, as Michael Balint would remind us, the most frequently used drug is the doctor himself. But our special knowledge of behavior does not make us immune from the interests of patients, courts, and legislators—especially those who are lawyers. In his response to the President a year ago (4), Alan Stone reminded us that we have to learn to live with lawyers—as he himself learned to do. But that obligation does not relieve us of the responsibility to educate those same lawyers concerning what we know about human behavior. Our values as psychiatrists are to avoid restriction for each human being—not only the restriction of hospital walls, but also the restrictions and inhibitions of the illness itself and the stigma that society still attaches to mental disorder and, indeed, to a degree, to all of illness.

Tomorrow's psychiatrists—the theme of our meeting next year—are the promise of the future of our profession and its service to humanity. How shall we help them develop their own values? Exhortation alone will not ensure their virtue. Tomorrow's ideals and values derive from the manner in which we select and teach today's students. The major influences on the behavior of tomorrow's psychiatrists are not the facts we present—those change all too frequently. More significant are their identifications during formative years.

Today's students are different from those of yesterday. They approach our profession with a strong commitment to social goals such as peace, civil rights, the reduction of poverty, and the protection of the environment. They prefer egalitarian attitudes and nonauthoritarian relationships with patients. They declare an interest in holistic medicine, the community, and primary care medicine. They are more concerned about responsibility to their own family and themselves and in participation in civic, cultural, and recreational activities. They are healthy, adaptive, and dedicated women and men—more so than my generation.

All professions must constantly reconsider their values. Some of them change, but the past remains a useful guideline for basic values. There are few as meaningful as those outlined by Sir William Osler (5):

> I have three personal ideals. One to do the day's work well. . . . The second . . . to act the Golden Rule . . . towards my professional brethren and towards the patients committed to my care. And the third has been to cultivate such a measure of equanimity as would enable me to bear success with humility, the affection of my friends without pride and to be ready when the day of sorrow and grief came to meet it with . . . courage (pp. 450–451). . . . And, if a fight is for principle and justice, even when failure seems certain . . . cling to your ideal. (p. 8)

I return to Hippocrates, Maimonides, and the other ancients of our profession. Their ghosts would remind

us that our first and most lasting value as psychiatrists should be a concern for our patients. Many people carry the label of patient—those with mild problems and those with problems that interfere with achieving their potential. But our major concerns must be with those who suffer most intensely. I refer to those with acute psychosis or a major affective disorder and those with chronic mental illness, who were often thrust prematurely into communities unprepared to receive them. Many nonpsychiatrists in the mental health field claim to and do possess expertise in psychotherapy and can be of help to some patients with milder forms of mental illness. However, the seriously ill and the chronic patient must not and cannot be neglected by psychiatrists.

The first responsibility of the psychiatrist is to treat the sick. That is the keystone of our value system.

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Alan A. Stone, M.D.
One Hundred and Eighth President, 1979–1980

BY SILVIO J. ONESTI, M.D.

It speaks for the wisdom of the members of the American Psychiatric Association that they nominated and elected Alan A. Stone as their 108th President. Alan brought vigor, knowledge, and judgment to the presidency at a time of forceful challenge to accepted ideals. Patients challenge us, legislatures challenge us, insurers challenge us, colleagues in allied professions challenge us, opinions within psychiatry challenge us, expanding scientific knowledge challenges us—all in ways that threaten our ability to provide professional and humane care to those whom we serve. Alan has recognized the challenge, and with it the opportunity to lead psychiatry in the direction of increasing its availability and effectiveness and its commitment to its patients. As psychiatrist, educator, and advocate, and as President of the American Psychiatric Association, he has been nationally and internationally the right spokesman for psychiatry at the right time.

BACKGROUND AND EARLY YEARS

Alan Abraham Stone was born in Boston, Mass., on August 15, 1929. All four of Alan's grandparents had come to the United States from Lithuania to escape oppression and to find opportunity. Alan traces antecedents of his intellectual and scholarly interests to Rabbi Elijah, the famous Vilna Gaon. Alan's grandfathers—both of them hardworking—established small businesses and provided for their families. Both died when Alan's parents were adolescents. Alan's father, one of seven children, worked while continuing his education through Boston University Law School, which in those days did not require a university degree for enrollment.

Alan's father graduated from law school at age 20 and became a law clerk for a judge in the East Boston Court. Soon married, he and Alan's mother took his mother's two sisters in to live with them. Alan's parents had three sons in rapid succession. Alan, the middle son, was born into a supportive family, Jewish in custom and belief but not in adherence to religious dogma.

Alan's father wanted to become the judge of the East Boston Court. Because of that he remained in East Boston after the Jewish population left. Alan grew up surrounded by Italian and Irish people. In that sense he felt himself an outsider. The few remaining elderly Jews who attended the synagogue on holidays did not provide a model of identification for Alan, who looked elsewhere.

Alan found a resource in books. The judge had given Alan's father his library. Alan read Scott, Hugo, de Maupassant, Balzac. One book that influenced him greatly was *This Believing World* (1) by a rabbi who had become an atheist. The idea that all religions were based on fear of death or fear of nature had a great impact on Alan's thinking. He became a secret atheist or at least an agnostic. A sense that the way of the world could be understood through knowledge of human motivation replaced Alan's earlier system of belief.

Social awareness accompanied Alan's psychological awareness. Alan's father, who was practicing law in Boston and East Boston, shared his offices with a black lawyer. He believed in the strength of people coming together; he began a camp for poor children of all races and religions. He believed that if whites and blacks, Catholics, Protestants, and Jews were exposed to each other as children they could overcome their racism. The camp played a large part in Alan's growing years.

As Alan's father and mother had given a home to his mother's younger sisters, so, during the 1930s, Alan's father and mother provided a home for a stream of Jewish refugees, all speaking foreign languages. Alan's parents gave generously, with his mother feeding and sheltering them and his father helping them to find work and homes. Alan's father became a passionate Zionist.

This background of compassion, generosity, responsibility, and devotion molded Alan's youth. Alan was a good son. He read, studied, and was interested in everything around him. It was easy for his father to see promise in Alan and invest in him hopes for the future. Alan's mother found in Alan a thoughtful confidant who returned affection and nurture with companion-

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ship. Alan believes that his mother's love and joy in living have been the most vital ingredients in his own life.

One day, when Alan was eight years old, his father brought home a football. Alan began playing, and within a year became part of a green-jerseyed neighborhood team called the Shamrocks, consisting of an Irish boy who was captain, Alan, and the rest all Italian. Football became a large part of Alan's life and was to remain so well into his college career. From football Alan acquired the nickname "Rocky," related more to his indomitable spirit as a linesman than to his surname.

The Boston Latin School for Boys, a special public school for able students, expanded Alan's world. He was outstanding among the brightest and most industrious students in Boston.

FORMATIVE YEARS

Alan entered Harvard University in 1946 among a large group of returning World War II veterans. He began as an economics major, intending to go to law school, until as a sophomore he took a course titled History of Religions. It was *This Believing World* revisited. It explored the phenomenon of conversion as well as the ideas of Judaism and Christianity and added the perspective of anthropology. He switched his major to psychology. Alan played freshman and varsity football. He broke his jaw in a practice during his senior year; his football career ended.

Alan's interest in psychology led him to considerations about aggression. At the time, the frustration-aggression hypothesis was in its ascendancy; it held to the notion that frustrations caused an increase in the instigation of aggression. Alan, guided by Prof. Henry Murray, who had developed the Thematic Apperception Test, administered that test to football players before and after scrimmages and after the season. He found that playing football provided no catharsis at all at the fantasy level; if anything, it seemed to provoke fantasies of retaliatory aggression. Alan elaborated his findings in his senior thesis (2) and received a thesis grade of summa cum laude.

The faculty asked Alan to stay at Harvard in psychology and social relations. Alan consulted Henry Murray, who advised him to go to medical school. Professor Murray said that medicine had a tradition with a past and a future; physicians belonged to a continuing historical and social institution. Alan decided to try to find his roots in medicine and psychiatry. Alan had read a novel about a man who, given a chance to live his life again, repeated the crucial unfortunate choice (3). Alan adopted the idea that one had to take a chance and do what was not expected in order to escape the inevitability of a patterned way. The suggestion of medical school jolted him, yet it sustained his intellectual interest in human nature. He spent a year completing the premedical requirements and applied to medical school.

Alan decided to go to Yale, partly because everyone expected him to go to Harvard and partly because he was impressed by Lawrence Kubie, whose lectures he attended when visiting for his interviews. He did not regret his decision. At Yale he came under the influence and guidance of Ernst Kris.

Alan started a weekly psychiatric study group of medical students that continued for four years. Ernst Kris met with them and encouraged Alan to participate in the longitudinal study of child development at the Yale Child Study Center. He asked Alan to undertake the bibliographic research—to read and catalogue all that had been done before in the way of longitudinal studies. Alan and a fellow student completed this as their medical school thesis; it was later published as a reference book (4).

Alan pursued his interest in child development by taking an internship in pediatrics. He wanted to observe the personality development he had read about in his bibliographic research. Alan thought that after Freud the most interesting contributions to psychiatry and psychoanalysis had come from people such as Ernst Kris, Anna Freud, Melanie Klein, and Erik Erikson, who had worked with children.

He remained at Yale. The Department of Pediatrics there was unique. Milton Senn, as chairman of the Department of Pediatrics and of the Yale Child Study Center, had united excellence in child development, child psychiatry, and child psychoanalysis with excellence in pediatric medicine. At Yale the pediatric intern did everything in the care of the total patient: bacteriology, electrolyte determinations, fluoroscopy, routine blood and urine analyses, care of newborns, care of nursing mothers, developmental assessments, and longitudinal individual and family care. All of the fragmentation of modern medicine was overcome in that department. It has remained for Alan the highest point of his medical experience; he was saving children's lives in a model setting dedicated to furthering the total health and well-being of children and families.

Alan went on into psychiatry. Ernst Kris recommended him to Grete Bibring for psychoanalysis and psychoanalytic training. Alan chose McLean Hospital for his psychiatric residency. He had developed an interest in psychotic patients while an undergraduate student at Harvard and a medical student at Yale. At McLean he could work with psychotic patients in intensive long-term psychotherapy under the close supervision of psychiatrists such as Alfred Stanton and Paul Howard. He took his final year of psychiatric residency training as a fellow in child psychiatry at the James Jackson Putnam Children's Center with Beata Rank. There, he increased his understanding of human development through treating its deviations in severely disturbed, psychotic preschool children.

Alan spent the subsequent two years in the U.S. Army Medical Corps, stationed in Denver and practicing psychiatry. When he returned to his home in Cambridge, he was invited by Mrs. Rank to join her research project regarding children with atypical development. He received a special NIMH grant to do so. He also studied blind retarded children. He observed and reported stereotypic behavior similar to that found in psychotic children (5). He continued to study psychotic adults (6, 7) and adult patients showing constellations of serious suicidal behavior (8–10). His interest in the psychodynamic understanding of severe psychopathologic symptoms united these endeavors.

The following year Alfred Stanton asked Alan to become Director of Residency Training at McLean Hospital, a position Alan held from 1962 to 1968. He is remembered by his residents as a gifted supervisor and educator who observed their therapeutic work directly or through videotapes, consulted on their evaluations, assisted them with psychodynamic formulations, taught them through classic and current literature in seminars at his home, and advised them in their careers. Alan furthered his development by completing his psychoanalytic training at the Boston Psychoanalytic Institute during this period.

PSYCHIATRY AND LAW

Alan's career in law began by chance. Prof. Alan Dershowitz had come to Harvard Law School from Yale. He wanted to introduce at Harvard a course that had originated at Yale called Psychoanalytic Theory and Legal Assumptions, but he needed a psychoanalyst to teach with him. Through Grete Bibring and Erik Erikson he found Alan Stone. Alan, the son of a lawyer and a person who at one time had considered law as a career, found himself in law.

The course the two Alans taught together was a remarkable success. Alan Dershowitz then asked Alan to join him in teaching the more standard course, Psychiatry and Law. Again the course was a success. A crucial ingredient was the dialogue between these two teachers, each committed to his own discipline but each willing to respect the other's view.

Alan decided he had learned all he could about law as an outsider. He received an NIMH grant to spend a year at Harvard Law School. He gave up his position as Director of Residency Training at McLean Hospital and took courses in criminal law, torts, contracts, constitutional law, and family law. Prof. Frank Sander asked him to help teach two courses, Family Law and Human Relations Problems in Legal Practice. The following year Professors Dershowitz and Sander left their courses to Alan. Two years later, at age 40, Alan became Professor of Law and Psychiatry in the Faculty of Law and the Faculty of Medicine at Harvard.

Alan's professorship has allowed him to pursue the life of a scholar; an important element in that life is detailed, rigorous interchange with colleagues in what he calls his "study groups." This tradition, which he began as a medical student, has continued to this day.

Alan entered the field of law at a time of challenge to the assumption that psychiatry and psychoanalysis were value-free sciences. He viewed the legal attacks on the psychiatric establishment not as a narrow problem of forensic psychiatry but as part of a deeper challenge to the hidden value assumptions in psychiatry that become visible in various legal arrangements. Alan has spent the subsequent 10 years of his career examining the moral implications of psychiatry.

Alan became aware of the need for a reasonable spokesman for psychiatry who understood the law and what was taught in law schools, and who knew what was expected of psychiatry and what a constitutional legal system required. Alan views his book *Mental Health and Law: A System in Transition* (11), published in 1975, as a contribution in this regard and his most important book. He formulated positions on a number of issues, such as the standards and procedures for civil commitment, the juvenile justice system, the determination of competency to stand trial, the right to treatment, and the right to refuse treatment. His positions have become those of the psychiatric establishment in briefs presented to state and federal supreme courts. Alan received the Manfred S. Guttmacher Award of the American Psychiatric Association for this work.

As chairman of the Committee on Law of the Group for the Advancement of Psychiatry, Alan was principal author of two reports: *The Right to Abortion: A Psychiatric View* (12) and *The Misuse of Psychiatry in the Administration of Criminal Justice: Competency to Stand Trial* (13). The report on abortion was important personally to Alan because initially he was opposed, both for moral reasons and because he accepted the view that a woman who has had an abortion unconsciously feels that she has committed murder. In the course of meeting with colleagues, Alan reconsidered his previous views, taken from authority rather than from experience and evidence. Although he did not change his moral position, he became convinced that psychiatry had no valid justification for opposing a woman's choice. The report provided him an opportunity to examine how law and psychiatry related to a major social problem. The report was to have practical importance; the New York State legislature used it in passing a law to include nonmedical as well as medical criteria for abortion. This was the prelude to two Supreme Court decisions.

The report on competency to stand trial addressed the controversial issue of the value of a psychiatric opinion in that assessment. The report took Alan's view that the psychiatrist can describe mental processes, but, when the concepts that go into the definitions

of competency and sanity are social, legal, political, and ultimately moral, psychiatrists have nothing in their science that allows them to decide the ultimate legal questions.

Alan has continued to add his ideas regarding the working relationship between law and psychiatry (14, 15). He is widely recognized as a leading spokesman in American psychiatry on this increasingly important subject.

AMERICAN PSYCHIATRIC ASSOCIATION

Alan's career in the American Psychiatric Association began with a request from the Committee of Concerned Psychiatrists that he be a petition candidate for Trustee. Alan agreed with their idea that the members should have a choice of candidates with new views. His election confirmed this. As Trustee he pressed to have the American Psychiatric Association respond to issues confronting it from government and the courts. At the end of his three-year term he sought the Vice-Presidency, again by petition. Alan served as Vice-President for two years and accomplished a great deal. His influence led to the American Psychiatric Association's organizing a Commission on Judicial Action (16) that could prepare amicus curiae briefs for courts throughout the country. He served as chairman. Alan felt it was important to obtain an excellent lawyer as general counsel for the American Psychiatric Association. He found one[1] who not only was a gifted lawyer but who knew the issues as seen by psychiatrists. Alan regards that lawyer, Joel Klein, as a mainstay of our Association. Alan's protective influence allowed the Commission and lawyer to articulate sensible positions that enabled the American Psychiatric Association to present a broad, adaptive, responsible stance.

Alan was nominated for the Presidency and won the election, stating the need for the leadership of the American Psychiatric Association to address social, political, and governmental issues. While President-Elect he held a Guggenheim Fellowship, which supported travel and time to study these issues. Alan also began writing a column for *Psychiatric News* in order to share his ideas with the members.

A major chapter in Alan's life was his participation in a group representing the American Psychiatric Association that was invited by the government of South Africa to investigate alleged abuses of blacks in their psychiatric facilities.[2] The study (17) was a landmark. It meant that the American Psychiatric Association, even with knowledge of the imperfections of psychiatric care in its own country, was willing to provide a model of objective criticism and to recommend specific improvements in the care of 10,000 blacks in mental hospitals in a foreign country. The study showed that psychiatric care in South Africa had been pervasively influenced by the system of apartheid.

While President-Elect, Alan visited Poland by invitation to discuss a new civil commitment law and a new mental health law for that country. Alan was requested to examine General Pyotr Grigorenko, a Russian dissident who had gained entry into the United States. Alan visited Russia, where he spoke to the leading Russian psychiatrist, Prof. Andrei Snezhnevsky, who, at the 1977 meeting of the World Psychiatric Association in Honolulu, had tried unsuccessfully to defend Russian psychiatry against the resolution that political dissidents in Russia were being misdiagnosed as psychiatrically ill and were being detained in mental hospitals. Professor Snezhnevsky stated that the Russians welcomed an examination of General Grigorenko and were certain that a diagnostic team would find him insane. Alan viewed this as a significant opportunity for American psychiatry to make an objective evaluation of a prominent dissident. The diagnostic team found no evidence of insanity and no need for mental hospitalization. Alan sent the report, as he had the report on South Africa, to the responsible authorities to give them an opportunity to comment prior to its publication.

As President of the American Psychiatric Association, Alan in his inaugural address called upon American psychiatry to hold true to its concern for the patient as a whole human being (18). During his presidency, Alan established the Council on Aging to apply this principle in regard to the psychiatric problems of the most rapidly enlarging segment of our population. He added the Council on Government Policy and Law to enable psychiatrists to develop model regulations and submit them to state and federal legislatures.

Alan inherited administrative problems within the American Psychiatric Association that required attention and action: the effects of inflation, the need for new headquarters, the need to raise funds and raise dues, and the need to travel to district branches to obtain informed support. He has taken appropriate steps with all of them.

As President of the American Psychiatric Association, Alan has been a strong spokesman for psychiatry. He testified in Congress, conferred with representatives and senators, appeared on television shows, talked endlessly to reporters who quoted him frequently in newspapers and journals. He addressed issues in a manner that repeatedly helped our patients and strengthened our profession. In his own words, "When our patients' needs are recognized, our profession's future will be secure."

[1]Joel I. Klein, who has written to confirm Alan's commitment to excellence, his humanitarian concerns, and his contribution to the law as a gifted teacher and thinker (personal communication, March 1980).

[2]The group was composed of Charles Pinderhughes, former Trustee; Jeanne Spurlock, Deputy Medical Director; Jack Weinberg, Past President; and Alan Stone, President-Elect.

FAMILY

Alan is a devoted husband and father; his greatest happiness has come from the love and companionship of his wife and children and from experiences shared with them. When the children were young, the family enjoyed summers at Cape Cod and Martha's Vineyard—boating, swimming, playing tennis, collecting mussels and oysters, and reading. Later, vacations were spent in Europe. More recently, Alan and his wife have traveled on holiday to distant parts of the world such as Brazil, Peru, India, and Iran.

Alan met his wife, then Sue Smart, while he was a senior at Harvard and she a sophomore at Radcliffe majoring in poetry. They married as soon as Sue completed college. Sue continued her interest in poetry for several years and a number of her works have been published. When Alan and Sue returned to Cambridge from the Army they collaborated on a book that combined their abilities and interests: *The Abnormal Personality Through Literature* (19). It was an immediate success. Many departments of English and psychology have used it as a textbook, as have a number of medical schools. Published in 1966, the book has been reprinted every year.

Later, when their children were grown, Sue went to law school. Since graduation in 1975 she has worked on projects of public interest. She has served the mentally retarded, has monitored implementation of regulations regarding consent for treatment of the mentally ill, and has examined conflicts of interest of state employees. She is current collaborating with Alan on a book about psychiatric malpractice.

Their daughter, Karen, is 27, a graduate of the University of California at Berkeley and a graduate student in psychology at Harvard; she will do her clinical work next year at McLean Hospital. Their older son, Douglas, is 25, a graduate of Harvard, a Rhodes Scholar, and at present a Karl Taylor Compton Fellow in Physics at the Massachusetts Institute of Technology. Their younger son, David, 22, is a senior at Harvard majoring in psycholinguistics and plans to go to law school. Alan holds his close relationships with all three of his children as very precious. He feels fortunate that he has them all nearby in Cambridge.

Alan and I have been friends for 25 years. We met as pediatric house officers at Yale. We are now psychiatrists and psychoanalysts at Harvard. I respect and esteem him greatly and admire his dedication to everything he values. He has been psychiatrist and psychoanalyst, educator and advocate, for all that is best in our field. He has been loyal to ideas and ideals, articulate in their expression, effective in their pursuit. He has brought his interest in the relationship of psychiatry and values to the attention of his colleagues through his writing and through his selection of the theme of his presidential meeting of the American Psychiatric Association. The theme "To Love and to Work: Human Values in Psychiatry" applies equally to Alan, a man whose love and work and whose human values have benefited those who suffer psychiatric illness and those who attempt to alleviate that suffering.

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Tardive Dyskinesia: Review and Update

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Tardive dyskinesia is an extrapyramidal syndrome associated with the chronic administration of major neuroleptic agents. The pathogenesis of the disorder appears to relate to chronic striatal dopaminergic receptor site blockade; the pathophysiology of tardive dyskinesia appears to relate to the resultant denervation hypersensitivity. Agents that deplete the brain of dopamine are the mainstay of therapy for tardive dyskinesia. Cholinergic agents that potentially modulate the balance between dopamine and acetycholine in the striatum offer possible additional therapeutic options. Clearly, resumption of neuroleptic therapy is treatment with the presumed pathogenic agent and is to be avoided whenever possible.

Lingual-facial-buccal dyskinesias are abnormal involuntary, persistent movements of the tongue, lips, and facial muscles (1), initially described as an integral part of the hyperkinesias of Huntington's chorea and other choreatic states. Following the introduction of neuroleptics into the practice of psychiatry in France in 1952, reports began to appear in the late 1950s of an extrapyramidal disorder characterized by persistent dyskinesias, often of a lingual-facial-buccal nature, which were associated with the long-term administration of neuroleptics. The late appearance led Uhrbrand and Faurbye in 1960 to apply the term "tardive dyskinesia" (2). By definition this term implies an iatrogenic disease caused by chronic neuroleptic therapy.

The early description of this syndrome emphasized abnormal motor movements affecting the face, the so-called bucco-lingual masticating syndrome. This consisted of involuntary mouthing, chewing, sucking, and licking movements of the tongue. Later, the syndrome description was broadened to include a variety of abnormal muscular manifestations, including choreoathetoid-type movements of the fingers, hands, arms, and feet and flinging or ballistic-type movements, particularly of the arms, as well as axial hyperkinesias and diaphragmatic movement resulting in grunting and difficult breathing. As the condition was studied more, the mouthing movements were further refined to include puckering, panting, smacking, and tongue movements inside the mouth. The clinical picture closely resembles L-dopa-induced dyskinesias and other choreatic disorders, both in the character of the movement abnormality and in the diversity of the individual symptoms.

EPIDEMIOLOGY

A number of population studies have been conducted to establish the frequency of tardive dyskinesia and the significance of such factors as sex, age, and type of underlying psychiatric disease. The prevalence of tardive dyskinesia has been estimated to range from .5% to 56% of patients treated with prolonged neuroleptic agents (3–6). This wide variance may be partly explained by the patient selection process and the diagnostic methods used to define the disorder. Studies of patients who were chronically institutionalized and older tended to report the highest incidence of tardive dyskinesia, but studies that focused on middle-aged and younger patients reported lower incidences (7–9). Smith and associates (10) used the Abnormal Involuntary Movement Scores (AIMS) to assess the prevalence of tardive dyskinesia and demonstrated that abnormal movement scores tended to be more severe with increasing age in both men and women up to age 70. In patients over 70, the mean AIMS decreased in the male population sampled and continued to increase in women. Hence, the often quoted 2:1 female to male prevalence ratio of the disorder was true only for patients over 70; otherwise no sex difference was found.

Other factors besides age and sex that have been studied in relation to the epidemiology of tardive dyskinesia include length of hospitalization, history of neuroleptic-induced extrapyramidal syndromes, and type of schizophrenia. No established patterns have

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emerged to clarify these epidemiological issues, and it appears that these are not significant factors in the development of tardive dyskinesia (11). Jus and associates (11) reported a higher prevalence of tardive dyskinesia in those schizophrenic patients who had an insidious rather than an acute onset of their psychiatric disease. They felt, however, that this may be an epiphenomenon because the insidious clinical presentation is more characteristic for the older patient population as a whole. Furthermore, they concluded that the prevalence of tardive dyskinesia was significantly higher if the mean age was higher at the beginning of medication therapy, again a possible reflection of age alone.

The fact that neuroleptic drugs are associated with the development of tardive dyskinesia might suggest that the greater the total amount of neuroleptic a patient has received, the higher the risk of severity of tardive dyskinesia. Two studies (12, 13) suggested an association between total amount of neuroleptic received and tardive dyskinesia, but a third study using higher neuroleptic doses (14) indicated no relation. Smith and associates (10) reviewed drug history and tardive dyskinesia in more than 100 patients and concluded that the total amount of neuroleptic received over many years is not the main variable related to the development of tardive dyskinesia; instead, a positive correlation was found between the maximal amount of neuroleptic administered in any one year and the tardive dyskinesia score. These results suggest that the temporal schedule and daily amount of neuroleptic administered may be epidemiologically more important than the total amount of medicine a patient receives. No increased prevalence of tardive dyskinesia in patients chronically treated with antiparkinsonian drugs has been definitely established (15–17). In fact, one study (5) reported a lower prevalence of tardive dyskinesia in patients receiving concurrent neuroleptic and antiparkinsonian drugs than in patients receiving neuroleptics alone.

NEUROPATHOLOGY

The observation that tardive dyskinesia frequently remains permanent even after discontinuation of antipsychotic drugs (2, 18–21) has led investigators to search for a permanent structural alteration in the brains of patients with tardive dyskinesia. Neuropathological studies following acute or chronic administration of antipsychotic drugs in laboratory animals have not demonstrated specific or localized neuropathologic changes in the brain beyond those secondarily produced by the diverse systemic effects of these drugs (22, 23). There is a single report of reduced neuronal cell count in the basal ganglia of rats chronically receiving phenothiazines (24), but this is of uncertain importance because gliosis or degenerative changes were not observed. Postmortem neuropathologic changes following chronic phenothiazine treatment in patients without extrapyramidal syndromes have usually consisted of scattered areas of neuronal degeneration and gliosis without convincing localization (22). Individual patients with drug-induced parkinsonism or tardive dyskinesias have been reported to have postmortem changes in the globus pallidus and putamen (25), in the caudate nucleus and substantia nigrae (26), and in the inferior olive, but other investigators (27) have reported no significant neuropathologic abnormality in patients with tardive dyskinesias.

Christensen and associates (28) have reported the presence of neuronal degeneration and gliosis of the substantia nigrae in 27 of 28 brains from patients with chronic oral dyskinesias, 21 cases of which were attributed to antipsychotic drugs. Only 7 of 28 matched control brains showed similar changes. Although the reported nigral degeneration and gliosis may represent a toxic effect of the drug, their occurrence in some elderly individuals who did not have tardive dyskinesia raises the possibility that they may be accompanying events of aging and not directly related to the appearance of tardive dyskinesia. The fact that no obvious anatomical alterations are associated with the development of tardive dyskinesia has led investigators away from microscopic studies and toward a more thorough evaluation of receptor site dynamics, neurochemistry, and pharmacology of the disorder.

PATHOGENESIS AND PATHOPHYSIOLOGY

Although the pathologic sequence of changes underlying the development of tardive dyskinesia remains unknown, the similarity of the abnormal movements to other forms of chorea suggests a common biochemical basis. A number of clinical and pharmacologic observations have helped to clarify the pathophysiology of the movements seen in tardive dyskinesia; these observations have etiologic as well as therapeutic implications. In summary, there is substantial evidence that the pathogenesis of tardive dyskinesia relates to chronic dopamine receptor site blockade; the pathophysiology of the disorder relates to the resultant receptor site hypersensitivity.

The first clues concerning the pathophysiology of tardive dyskinesias came from the observation that the neuroleptics—agents associated with the development of tardive dyskinesias—are the same agents known to cause drug-induced parkinsonism. Parkinsonism is felt to result from deficient dopaminergic activity in the striatum (29). The fact that the neuroleptics can induce a parkinsonian syndrome suggests that these agents may alter central dopaminergic balance and thereby chemically simulate the structural abnormality seen in Parkinson's disease. Biochemical studies have corroborated these hypotheses and have shown that the

neuroleptics act by blocking dopamine receptor sites in the limbic system and the striatum (30). This blockade of dopamine receptors in the striatum is felt to underlie the neuroleptic-induced parkinsonism so often seen within the first weeks or months after neuroleptic induction. These studies show that neuroleptics affect the dopaminergic system and that, at least initially, the effect is one of functional underactivity of dopamine at dopamine receptor sites of the striatum.

This blockade may be thought of as a form of chemical denervation or functional inactivation of dopamine receptors in the striatum. If the blockade is complete at any receptor site, the receptor is prevented from receiving its normal neurotransmitter influence. This does not imply that the blockade is complete for all dopaminergic receptors throughout the striatum. The concept is important because it implies that not all receptors are blocked to the same extent by a single agent. If the latter were true, chemical denervation could occur only following complete blockage of all dopamine receptors, which would necessarily be accompanied by a severe form of drug-induced parkinsonism.

A second clue concerning the pathophysiology of tardive dyskinesias comes from the observation that these dyskinesias are remarkably similar to and often indistinguishable from the abnormal movements seen in Huntington's disease and L-dopa-induced dyskinesias. These latter two movement disorders are felt to relate to a heightened response of the dopamine receptor sites in the striatum. The similarity of tardive dyskinesias to movement disorders whose pathophysiology is better understood suggests that tardive dyskinesias, too, may relate to altered sensitivity of the striatal dopaminergic system.

Whereas parkinsonism results from underactivity of the dopaminergic system, tardive dyskinesias appear to result from overactivity. The neuroleptics would then appear to be associated with both dopaminergic syndromes, initially parkinsonism, implicating dopamine underactivity, and eventually tardive dyskinesias, implicating dopamine overactivity. The explanation behind this apparent dichotomy relates to the fact that after chronic blockade, receptor function is altered. A large body of evidence demonstrates that after prolonged denervation, a receptor site becomes hypersensitive to ambient amounts of neurotransmitter exposure. This results in an enhanced response of the postsynaptic cell to neurotransmitter concentrations that previously led only to low rates of baseline firing. This phenomenon of denervation hypersensitivity has been extensively studied in the peripheral receptor site system and has been shown to apply to the dopaminergic striatal system as well. Ohye and associates (31) showed that after prolonged denervation the postsynaptic striatal cells fire with enhanced frequency in response to ambient dopamine that previously caused only low, baseline firing rates. A similar behavioral hypersensitivity can be seen in animals after chronic neuroleptic administration (32). The ultrastructural basis for this chronic hypersensitivity has been studied in detail, and it appears that hypersensitivity relates pathophysiologically to alterations in dopaminergic receptor site populations. After chronic neuroleptic administration to animals, there is an increase in the number of striatal dopamine receptors that clearly parallels the behavioral changes (32, 33). Analogous alterations may underlie the pathophysiology of tardive dyskinesia in man.

Dopamine turnover studies are consistent with the gradual development of denervation hypersensitivity. Cerebrospinal fluid levels of homovanillic acid, the major metabolite of dopamine, tend to increase acutely following the administration of neuroleptics (34). Within the first few weeks of treatment, this elevation tends to decrease so that after chronic treatment there is generally decreased turnover of dopamine (35–37).

The question of which dopamine receptor sites are sensitized is of central importance to an understanding of the pathogenesis and pathophysiology of tardive dyskinesias. If tardive dyskinesias result from prolonged blockade of the same dopaminergic receptors whose less chronic blockade leads to parkinsonism, some degree of parkinsonism would be a prerequisite for the subsequent occurrence of drug-induced tardive dyskinesias. Furthermore, these dyskinesias should occur in the same muscles, supplied by the same receptors that were involved in the parkinsonian manifestations. This is clearly not the case: tardive dyskinesias often occur in patients who have had no history or evidence on physical examination of drug-induced parkinsonism (29). Additionally, although patients with tardive dyskinesia tend to have fewer hypokinetic parkinsonian symptoms than neuroleptic-treated patients without tardive dyskinesia, case reports demonstrate that patients may suffer simultaneously from both tardive dyskinesia and drug-induced parkinsonism (38–40).

These observations suggest the possibility of more than one dopamine receptor site in the striatum. In fact, there is evidence from cellular recordings that at least two different dopamine receptor populations exist in the striatum. Dopamine receptors of the striatum have been demonstrated to respond to dopamine with facilitation or inhibition. Since the response of a receptor to a transmitter is a property of the receptor site and not of the transmitter, two different responses suggest two different receptors. When dopamine activates one type of receptor, the neuron is hyperpolarized or inhibited. These receptors could be called dopamine-inhibited receptors. The second type of receptor responds to dopamine stimulation with depolarization or facilitation and could be called the dopamine-facilitated receptors. Parkinsonism is usually related to decreased activity of dopamine at the dopamine-inhibited receptors.

It is of interest that although the neuroleptics block

both types of dopamine receptors, the degree of blockade is different for the two populations. The dopamine-facilitated neurons are blocked by levels of haloperidol or chlorpromazine that do not block the action of dopamine at dopamine-inhibited receptor sites. Only with larger doses of the neuroleptics are the dopamine-inhibited receptors also blocked (41). This observation, together with the observation that tardive dyskinesias can occur independently of drug-induced parkinsonism, suggests that the two disorders both relate to dopamine but to different receptor populations. One could visualize tardive dyskinesias as an abnormal hyperactive response of dopamine-facilitated receptors to dopamine, while parkinsonism relates to a lack of response of the dopamine-inhibited receptors to dopamine.

No large studies measuring receptor site populations in people with tardive dyskinesia have been performed, although receptor site measurements from brains of schizophrenic patients have been investigated. Owen and associates (42) demonstrated increased dopamine receptor sensitivity in both receptor numbers and affinity as measured by spiroperidol binding in the postmortem brains of schizophrenic patients. These biochemical changes were seen in patients who were receiving neuroleptic agents at the time of death, as well as in patients who were not receiving medication. However, those patients who were currently receiving medication demonstrated a greater degree of receptor site sensitivity. No information was reported on the incidence of tardive dyskinesia in these patients, so these data cannot be applied directly to the pathophysiology of tardive dyskinesia. However, dopamine receptor site sensitivity was increased in the caudate nucleus and putamen as well as in the nucleus accumbens of the limbic system. The former two basal ganglia are believed to be related to movements and motor disorders, while the latter limbic dopaminergic nucleus has been suggested to relate to the pathophysiology of psychosis.

A study by Lee and associates (43), which was similar to that of Owen and associates, reported increased haloperidol binding in the caudate nucleus, putamen, and nucleus accumbens of brains from patients with schizophrenia. Again, the prevalence of tardive dyskinesia in these patients was not discussed. Since ^{3}H-apomorphine binding was not altered in these brains, Nagy and associates (44) suggested that neuroleptic-induced alterations in receptor site binding in all three dopamine-rich areas were predominantly related to postsynaptic receptors.

A number of investigators have attempted to demonstrate dopaminergic hypersensitivity in patients with tardive dyskinesia by the use of various neuroendocrine tests. Neuroendocrine interest in relation to tardive dyskinesia and dopamine stems from the observation that there is a readily accessible and well-defined hypothalamine dopaminergic pathway that involves prolactin and human growth hormone (HGH) control (45–49). If tardive dyskinesia represents or is associated with a diffuse hypersensitive response of dopaminergic systems, neuroendocrine hypersensitivity might be expected. If a selective striatal alteration underlies the pathophysiology of tardive dyskinesia, however, necessary neuroendocrine changes in dopaminergic function would not be anticipated. Cohen and associates (50) reported normal prolactin response after exposure to thyrotropin-releasing factor in 4 schizophrenic patients with tardive dyskinesia and could demonstrate no hypothalamine dopaminergic supersensitivity. Similarly, the prolactin measurement in patients with tardive dyskinesia challenged with apomorphine showed no enhanced or hypersensitive response when compared with normal subjects (51). Growth hormone measurements after apomorphine challenge have suggested the same lack of hypothalamic hypersensitivity. One study (51) failed to show evidence of any difference between normal control subjects and patients with tardive dyskinesia, but a second study (52, 53), using the same apomorphine dose, reported that chronic schizophrenic patients with and without tardive dyskinesia showed slightly but not significantly lower growth hormone levels than control subjects. Although growth hormone responses in acute schizophrenia appeared higher than responses in control subjects, these responses cannot be viewed as relative to tardive dyskinesia because it is seen by definition after prolonged neuroleptic therapy for chronic disease. Neuroendocrine studies have therefore not revealed evidence of hypothalamic hypersensitivity in patients with tardive dyskinesia. These data would suggest that if the disorder relates to dopaminergic hypersensitivity, it is a selective hypersensitivity of certain receptor populations. Because of the clinical similarity of tardive dyskinesia to Huntington's chorea, the likely level of selective hypersensitivity would be the striatum. Studies of striatal receptor site hypersensitivity in nonschizophrenic patients with tardive dyskinesia have not yet been reported.

PHARMACOLOGIC THERAPY

With this pharmacologic background, one can consider therapeutic options in terms of the pathogenesis and pathophysiology of tardive dyskinesia. If the pathogenesis of the disorder relates to chronic dopaminergic receptor site blockade and the pathophysiology relates to the resultant denervation hypersensitivity, agents that interrupt this sequence would be of potential benefit. Reserpine acts by blocking the reuptake of dopamine, norepinephrine, and serotonin into the intraneuronal storage vesicles. In this way, reserpine depletes the brain of dopamine. Since it acts presynaptically, this drug would allow less endogenous

dopamine to be released onto the presumed hypersensitive dopamine receptor, thereby effecting a more normal dopaminergic homeostasis. Reserpine given in doses of 1–5 mg/day has been shown to reduce tardive dyskinesia in a number of clinical trials (54, 55). The potential side effects of hypotension and psychological depression must be monitored carefully. Reserpine-induced depression is infrequent in schizophrenic patients who do not have a history of affective disorder.

Jus and associates (56) recently reported treating tardive dyskinesia on the basis of a concept of slow desensitization of dopamine receptor sites. They hypothesized that because a long period of time is required to build up striatal hypersensitivity, a prolonged and slow decrease in neuroleptic blockade might resolve the problem. They treated 62 chronic schizophrenic patients who had tardive dyskinesia by a very slow (average period of 3.9 years), progressive, stepwise diminution of neuroleptic and antiparkinsonian medication. At the same time, they administered slowly increasing doses of reserpine or, when it was clinically necessary to control psychosis, a more sedating neuroleptic rather than the long-acting phenothiazines. Their long-term results were disappearance of tardive dyskinesia in 37% of the patients, improvement in 42%, and no effect in 21%. No patients demonstrated progression of abnormal movements. This long-term inpatient study has not yet been expanded to include outpatients.

Tetrabenazine, a synthetic benzoquinolizine that depletes monoamines by a mechanism similar to that of reserpine, is more rapid in onset, more selective for CNS activity, and has less hypotensive effects than reserpine. Given in doses of 100–300 mg/day, tetrabenazine has reduced or abolished tardive dyskinesia in several clinical trials, most of which, however, were short-term or lasted less than 6 months (57–60). Agents that deplete central dopaminergic stores serve as the mainstay of current therapy for tardive dyskinesia.

Formerly, tardive dyskinesias were often treated by increasing the dose of neuroleptics. This shortsighted treatment generally abates the abnormal movements but clearly is contraindicated. This method of therapy treats the pathophysiology of tardive dyskinesia but can aggravate the pathogenesis by further denervation and subsequent hypersensitivity. Epidemiological evidence shows a direct relationship between the incidence of tardive dyskinesia and the duration of neuroleptic therapy. In the individual patient, abnormal movements show a gradual progression over time, usually beginning with mild, localized lingual-facial-buccal dyskinesias and later becoming more severe and generalized. As neuroleptic therapy is prolonged, there is an increase in both the severity of abnormal movements and the number of body areas involved. As discussed in the section titled Epidemiology, the issue of age relative to prevalence of tardive dyskinesia can be difficult to separate from chronicity of medication. Nevertheless, it appears that the longer the pathogenesis persists, the more frequently and severely the physiology will be disrupted (21, 29, 54). For these reasons neuroleptics have no role in the routine treatment of tardive dyskinesias.

An additional therapeutic option is the manipulation of other neurotransmitters. There is a well-known and accepted balance between dopamine and acetylcholine in the striatum. Although Parkinson's disease relates primarily to dopamine alteration, the cholinergic system is known to modify the movements of the disease (29). Presumably, since dopamine is already decreased in the illness, decreasing the cholinergic influence as well would reestablish the relative neurotransmitter balance and reinstitute the former homeostasis at a different absolute level. Under the same premise, if tardive dyskinesias relate to an increase in dopaminergic effect, elevating the cholinergic effect would reestablish the former homeostasis, again at a new neurotransmitter level. In fact this appears to be the case: anticholinergic agents appear to aggravate the abnormal movements of tardive dyskinesias, and cholinergic agents may ameliorate them. Klawans and Rubovits (61) administered intravenous physostigmine to patients with tardive dyskinesias and demonstrated clear abatement of the abnormal movements. Although this study demonstrated the importance of neurotransmitter balance to the pathophysiology of tardive dyskinesias, this therapy has not been of practical benefit to patients because physostigmine cannot be given orally. Orally available precursors of acetylcholine include choline chloride and phosphatidylcholine (lecithin). In short-term studies (62), both agents have been reported to be useful in the control of tardive dyskinesias. Long-term controlled studies have not yet been conducted; these drugs remain experimental. Dimethylaminoethanol (deanol) was originally reported to be efficacious in the treatment of tardive dyskinesias, but more extensive studies have not corroborated this finding (63).

Using the same concept of dopaminergic-cholinergic antagonism relative to tardive dyskinesia, but with a focus on early or presymptomatic detection, Chouinard and associates (64) advocated the very short-term use of anticholinergics. They exposed patients who were receiving chronic neuroleptic therapy to small doses of anticholinergic drugs for a short period of time to see whether these doses uncovered or precipitated choreatic movements. If subclinical dyskinesias could be transiently unmasked by this alteration of dopaminergic-cholinergic balance, this precipitation would theoretically warn the clinician that striatal hypersensitivity was developing. Appropriate reduction of the neuroleptic or introduction of dopaminergic depleting agents could then be instituted as an early intervention. The efficacy of such treatment is unproven, and the distinction between this short-term provocation course of anticholinergic therapy and the

chronic exposure to anticholinergic drugs is essential. The latter course has been suggested, although not proven, to be associated with an enhanced chance of developing tardive dyskinesia.

Other agents have also been tried but remain unproven in the treatment of tardive dyskinesia:

1. Lithium. In two short-term studies mild improvement has been reported in tardive dyskinesia with lithium therapy (65, 66). Other studies reported no improvement or exacerbation (67, 68). Additionally, reemergence of tardive dyskinesia that had been quiescent for several years was seen in a patient when lithium therapy was introduced (69). The interaction of lithium with the dopaminergic system is not understood, but it is known that lithium accelerates synaptic reuptake of norepinephrine (*70*).

2. Baclofen (in patients who have to be maintained on neuroleptics). It has been suggested that baclofen, as a presumed γ-aminobutyric acid (GABA) agonist, affects the dopaminergic system at the level of the striatum or substantia nigra (71).

3. Levodopa. There are reports of amelioration following initial worsening of tardive dyskinesia with L-dopa (72).

4. Alpha-methyldopa. One report suggested amelioration of symptoms (73), but another indicated no change in tardive dyskinesia after methyldopa therapy (74).

5. Clonazepam. A benzodiazepine derivative, this drug is used in the treatment of seizure disorders. It has been reported to improve dyskinesia in 42 patients given 1–3 mg daily (75).

6. Manganese. The successful treatment of tardive dyskinesia has been reported in a small series of subjects given 60 mg/day of manganese chelate (76–78).

7. ECT. It has been suggested that ECT could increase the likelihood of the development of tardive dyskinesia in patients receiving neuroleptic drugs (2, 21, 79). These reports, however, lacked important historical and drug treatment information, so direct association could only be conjectured. In fact, a 1978 report (80) suggested that tardive dyskinesia may be ameliorated after ECT. The patient reportedly had had a stable degree of abnormal movement before ECT and showed a sudden dramatic and sustained improvement after the first three ECT treatments.

All of these agents were studied for a short period of time in small groups of patients. None of these modalities can be definitely recommended at this time.

RECOMMENDATIONS

The following principles should help to decrease the incidence of tardive dyskinesia:

1. Decrease the number of subjects at risk. Neuroleptics should be used only when clearly indicated and continued only when efficacy is clear.

2. Keep the daily dosage as low as possible.

3. Avoid the chronic use of anticholinergics if possible. Since anticholinergic agents clearly exacerbate already present tardive dyskinesia, it is possible that with chronic administration these drugs may alter the striatal neurochemical balance and potentiate the development of abnormal movements de novo (29).

4. Keep the duration of therapy as short as possible. Most schizophrenic patients are maintained on neuroleptics to control chronic symptoms or to prevent exacerbation. If used for the latter purpose it is quite possible that neuroleptics can be withdrawn for at least some period of time without a severe exacerbation. It is possible that such drug holidays might decrease the overall incidence of tardive dyskinesias. Data collected from animal studies suggest that drug-free periods should be at least one month long to be effective (32). The decision to withdraw neuroleptics must be individualized for each patient, and the relative morbidity of the abnormal movements must be weighed against the danger of a psychotic exacerbation.

5. Detect tardive dyskinesia early and quickly withdraw medication when possible.

6. In the event that neuroleptics are absolutely required, we tend to switch the medication to thioridazine. This practice is based on several considerations. First, there appears to be some relationship between drug-induced parkinsonism and tardive dyskinesia, and thioridazine has the lowest incidence of drug-induced parkinsonism of available neuroleptics. Evidence from animal experiments also suggests that some agents may be less likely to induce receptor hypersensitivity than others. In animals, thioridazine is a much weaker blocker of striatal dopamine receptors than other neuroleptics. Furthermore, in our laboratory we have been unable to produce receptor-site hypersensitivity measured either biochemically or behaviorally with chronic thioridazine but can easily produce this state with other phenothiazines and haloperidol in equivalent doses (81). There are no human data to establish a lower incidence of tardive dyskinesia with thioridazine, but, because of the above considerations, we feel that it is reasonable to substitute thioridazine in this situation. Clozapine, a neuroleptic not available in the United States, has the lowest incidence of associated parkinsonism and appears to be a more specific dopamine receptor blocker for the nucleus accumbens of the limbic system. In European studies, clozapine was reported to ameliorate tardive dyskinesia.

The low incidence of parkinsonism caused by thioridazine and clozapine has often been attributed to the ability of these agents to block central muscarinic receptors. The fact that these agents are both dopaminergic blockers and cholinergic blockers might suggest that they should have the highest likelihood of causing tardive dyskinesia. This is certainly not the case. No reports of clozapine-induced tardive dyskinesia have

yet appeared. From a variety of animal behavior studies it is clear that clozapine and thioridazine are not equivalent to another neuroleptic (e.g., haloperidol) plus an anticholinergic agent (72). The differences in incidence of parkinsonism may not be due solely to a neuroleptic's anticholinergic properties but may reflect a preferential ability to block specific striatal dopamine receptors.

With the exception of clozapine, the relative incidence of tardive dyskinesia with different neuroleptics is not definitely known; although thioridazine can cause tardive dyskinesia, we tend to feel safer using this drug when neuroleptic treatment is necessary.

PROGNOSIS

Shortly after neuroleptic withdrawal, tardive dyskinesia may worsen, presumably because of the better access of dopamine to striatal receptors. This exacerbation, however, is usually only a short-term effect. Tardive dyskinesias, in fact, are often reversible and may spontaneously remit following neuroleptic withdrawal. The earlier the symptoms are recognized and the drugs withdrawn, the better the prognosis for recovery (29). This is consistent with the view that tardive dyskinesia is a progressive disorder. The chance that tardive dyskinesias may become irreversible can be decreased by early detection and neuroleptic withdrawal; therefore, patients should be carefully examined for dyskinesias at frequent intervals. After the discontinuation of neuroleptic therapy, tardive dyskinesias can disappear, in some cases within one to two months. Larger series with more prolonged follow-up after the withdrawal of antipsychotic drugs have shown that patients can improve for up to two years and that mildly affected patients are more likely to experience complete remission (5, 6).

SUMMARY

The basic pathogenesis of tardive dyskinesia appears to relate to chronic pharmacologic denervation of specific dopaminergic receptor sites in the striatum. The pathophysiology of the disorder relates to the resultant denervation hypersensitivity. The mainstay of treatment includes withdrawal of neuroleptics when feasible and the use of dopamine-depleting agents. Enhancement of the striatal cholinergic input offers potential ancillary benefit to the alleviation of abnormal movements. The benefit of manipulating other neurotransmitters remains experimental. Treatment of tardive dyskinesia with neuroleptics themselves is clearly treatment with the presumed offending agent and should be avoided. This shortsighted therapy may temporarily abate the pathophysiology of the condition but serves to aggravate its pathogenesis.

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The Manfred S. Guttmacher Award

The American Psychiatric Association invites applications for its biennial Manfred S. Guttmacher Award for 1981. The award is now given in odd-numbered years for outstanding contribution to the literature of forensic psychiatry in the form of a book, monograph, paper, or other work, including audio-visual presentations (films, etc.), submitted to or presented at any professional meeting or published during the period from December 1, 1978, to December 1, 1980. A plaque and an honorarium of $750 will be presented to the award recipient at the Convocation of Fellows at the 1981 APA annual meeting. Please note that the award recipient is now required to give a lecture at the annual meeting.

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Opiate Use and Sexual Function

BY STEVEN M. MIRIN, M.D., ROGER E. MEYER, M.D., JACK H. MENDELSON, M.D., AND JAMES ELLINGBOE, M.D.

Although opiate addicts often equate the drug experience with sexual orgasm, diminished libido and impaired sexual performance are common sequelae of chronic use. Early clinical studies suggested that opiates may interfere with sex hormone secretion. The authors carried out three sequential studies which demonstrated that heroin use in man results in acute suppression of luteinizing hormone (LH) release from the pituitary followed by a secondary drop in plasma testosterone levels. The time course of these neuroendocrine events correlates well with the tension-reducing effects of heroin and suggests that drive reduction is an important component of opiate reinforcement.

The dramatic effects produced by an injection of heroin have been well described by both clinicians and users (1–4). A brief but very intense euphoria, followed by tension relief, seems to be the common denominator in the heroin experience. Chessick (5) called this sequence of events the "pharmacogenic orgasm," and addicts themselves often describe it in sexual terms. In the case of intravenous heroin the immediate postinjection "rush" is frequently equated to sexual orgasm, and subsequent feelings of tension relief and relaxation have been likened to "being wrapped in warm cotton," "returning to the womb," or similar allusions to warmth, security, and being cared for.

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In contrast to these retrospective accounts of the drug experience, untreated male heroin addicts and patients in methadone maintenance programs report that decreased interest in sex, impotence, and delayed ejaculation are common problems during opiate use (6–10), and opiate withdrawal has been associated with renewed sexual interest and premature ejaculation (11) in many patients. As a result of these observations, interest in the sexual sequelae of opiate use has shifted from an emphasis on intrapsychic factors to the study of the hormonal correlates of drug administration. In this context, a series of clinical and laboratory studies have explored the effects of opiates on the hypothalamic-pituitary-gonadal axis. This paper reviews a number of these studies in man, including some recent data from our laboratory. We will attempt to reconcile the curious dichotomy between the acute reinforcing effects of opiates, as expressed in the language of sexual gratification, and the neuroendocrine sequelae of chronic use, which appear to be somewhat less gratifying.

EFFECTS OF CHRONIC OPIATE USE ON PITUITARY-GONADAL FUNCTION IN MAN

Early clinical studies (12–14) of the effects of opiate use on pituitary-gonadal function were hampered by a variety of methodologic problems. In outpatient studies it was difficult to control for the amount of prior drug use across subjects as well as the amount of time elapsed between drug administration and hormonal measurements. Furthermore the pituitary gonadotropins and testosterone are secreted in a pulsatile fashion; thus even individuals with normal gonadal function have considerable moment-to-moment variation in the mean plasma level of these hormones (15). Consequently the infrequent plasma sampling (i.e., once or twice per day) that characterized earlier clinical studies made it difficult to accurately assess drug-induced changes in the episodic secretory pattern of these hormones. Finally, most early studies lacked the methodology for direct measurement of pituitary and gonadal hormones. More recent studies (16–21) have used modern radioimmunoassay techniques for the quantitation of these hormones.

In the face of these methodologic difficulties it is not surprising that the results of earlier studies often

proved contradictory. Nonetheless some general trends can be discerned. Azizi and associates (12) found lower than normal levels of serum testosterone in 8 of 16 heroin addicts and 2 of 6 chronic methadone users, although levels of estradiol, luteinizing hormone (LH), and follicle-stimulating hormone (FSH) were not consistently altered in these subjects. Cushman (13) also reported no change in LH secretion among active heroin users and abstinent former addicts. In a subsequent study (14), however, high dose methadone users (>40 mg/day) and some untreated heroin addicts had low plasma testosterone levels when compared with nonaddict controls; they also reported a variety of sexual problems during active drug use. In another study, Martin and associates (16) observed decreased plasma LH and FSH levels in 5 men maintained on 100 mg of methadone per day, but after 1 month of abstinence their gonadotropin levels were actually increased over predrug levels. In 1975 Mendelson and associates (17) studied a group of patients on heroin maintenance who, after receiving fixed doses of heroin at fixed intervals, reported diminished interest in sex and potency problems and had a significant decrease in plasma testosterone compared with normal nonaddict control subjects. These investigators also found lower than normal levels of plasma testosterone in untreated heroin addicts who had a recent history of heavy use and in patients on high dose (80–150 mg/day) methadone maintenance. In a subsequent study (18) Mendelson and Mello found diminished plasma testosterone levels in a group of chronic heroin users; after 1 month of abstinence, however, their testosterone levels were within the normal range for adult males. Finally, Cicero and associates (19) compared 29 patients maintained on methadone (mean dose=67 mg/day) with 16 active heroin addicts and 43 drug-free controls and found that methadone-treated patients had markedly impaired function of their secondary sex organs and significantly lower levels of serum testosterone compared with the other two groups.

Over the last 4 years our research group has carried out a series of studies on the acute and chronic effects of opiate administration on pituitary-gonadal function, attempting to avoid some of the methodologic problems found in earlier studies. As part of a multidisciplinary research project, detoxified heroin addicts were admitted to a four-bed research ward and allowed to self-administer intravenous heroin under a variety of experimental conditions. Their informed consent was obtained after the procedures had been fully explained. In all studies individual subjects or groups of subjects served as their own controls. Heroin dosage and the timing of plasma sampling for hormonal determinations was controlled by the experimenters. Since these studies, including the methods used to quantitate plasma levels of testosterone and pituitary gonadotropins, have been reported elsewhere (20–22), we will only summarize our findings here. The designs of our three experiments are presented in table 1.

Experiment 1

In our first study (20) we assessed the effects of chronic heroin use on plasma testosterone levels in 10 detoxified addicts, who were permitted self-regulated access to increasing doses of intravenous heroin over a 10-day period. Blood samples for plasma testosterone determinations were collected each morning at 8 a.m., approximately 6 hours after the previous heroin injection. The acute hormonal response to heroin or its effect on pituitary gonadotropins was not assessed in this first study. During study period 2, as the total daily dose of heroin increased, plasma testosterone levels fell. When the preceding day's heroin dose was between 45 and 60 mg, testosterone levels fell below 400 ng/100 ml, significantly lower than during study period 1, when subjects were drug-free ($p<.02$, matched t test). Following a period of methadone detoxification (study period 3), testosterone secretion gradually returned to predrug levels.

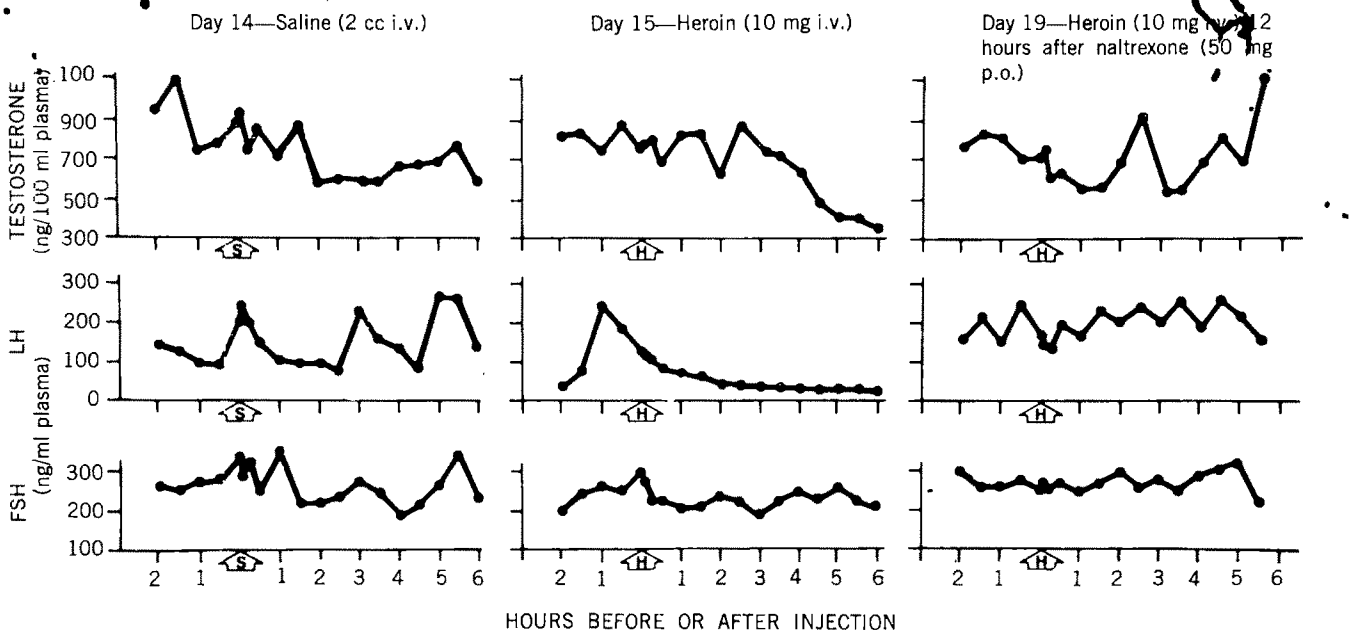
Experiment 2

In our second study (21) we sought to measure the acute effects of heroin on the pituitary-gonadal axis. After 2 weeks on a drug-free regimen and 1 saline control day (periods 1 and 2), 2 detoxified male addicts were given 10 mg i.v. of heroin. The 2 subjects had very similar hormonal responses. As illustrated in figure 1 (subject 1) the result was an almost immediate fall in the level of plasma LH. This acute response was not observed when subjects received saline, nor did it occur when subjects were pretreated with naltrexone, a narcotic antagonist with few agonistic effects of its own (23). In both subjects LH suppression was still observed 6 hours after heroin injection, although some recovery in LH secretion was noted in 1 subject. Interestingly, pretreatment with naltrexone, 12 hours before heroin administration, resulted in a mean 50% rise in plasma LH levels compared with levels on the saline control day. The number and frequency of LH secretory pulses also increased following naltrexone administration. Despite the rise in LH, however, testosterone secretion was only marginally increased in 1 subject and unaffected in the other. In none of the experimental conditions was FSH affected.

Experiment 3

In our third study (22) we examined the acute and chronic effects of unblocked and naltrexone-blocked heroin use on the pituitary-gonadal axis. Our goals were to replicate our findings from experiment 2 in more subjects and to better assess the time course of the acute suppressant effect of heroin on LH and testosterone. We also wanted to determine whether chronic heroin and/or naltrexone use altered the acute response to heroin. There were three different double-

FIGURE 1
Plasma Testosterone, LH, and FSH Levels Before and After Injection of Saline, Heroin, and Heroin Preceded by Naltrexone (22)[a]



[a]All injections were given at 10:00 a.m.

TABLE 1
Three Studies of the Acute and Chronic Effects of Opiate Administration on Pituitary-Gonadal Function (22)

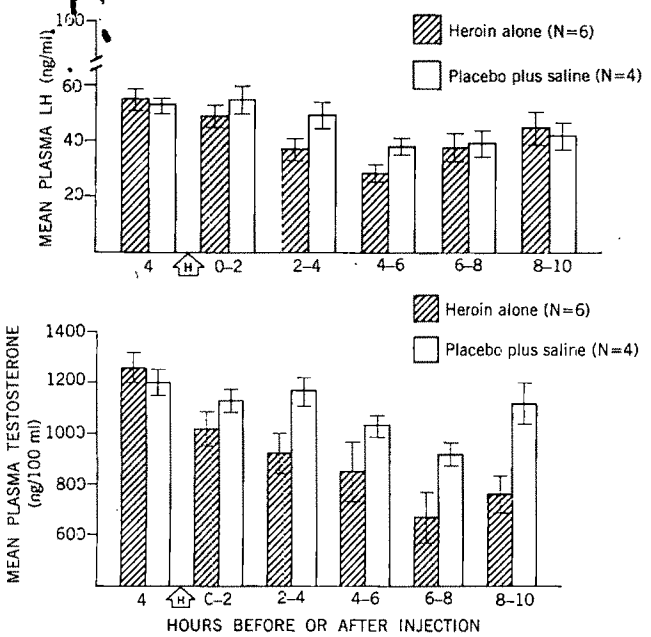
Study Period	Experiment 1 (N=10)		Experiment 2 (N=2)		Experiment 3 (N=8)	
	Day	Condition	Day	Condition	Day	Condition
1	1-7	Drug-free	1-13	Drug-free	1-7	Drug-free
2	8-17	Heroin[a]	14 (10:00 a.m.)	Saline, 2 ml i.v.	7 (2 p.m.)	Heroin, 10 mg i.v. (unblocked)
3	18-24	Methadone detoxification	15 (10:00 a.m.)	Heroin, 10 mg i.v. (unblocked)	10 (6 p.m.) 10 (10 p.m.)	Naloxone, 0.4 mg i.v. Naltrexone, 50 mg i.v., or placebo
4	25-34	Drug-free	18 (6 p.m.)	Naloxone, 0.4 mg i.v. Naltrexone, 50 mg p.o.	11 (2 p.m.)	Saline, 2 ml i.v., plus naltrexone, 50 mg p.o., or placebo
5	35-36	Narcotic antagonist	19 (10 a.m.)	Heroin, 10 mg i.v. (blocked)	12-23 (8 a.m.)	Naltrexone, 50 mg p.o., or placebo
6	37-46	Narcotic antagonist plus heroin	20-25	Naltrexone, 50 mg p.o.	13 (10 a.m.) to 23 (8 a.m.)	Heroin[a]
7	47-60	Narcotic antagonist alone			23 (2 p.m.)	Heroin, 10 mg i.v.

[a]Heroin was available any time but not more often than every 2 hours to prevent drug accumulation. The allowable intravenous dose escalated from 0.5 mg on day 8 to 5 mg on day 18 to accommodate tolerance. By waiting 4 hours the subject could double the allowable dose; after 6 hours he could triple it. Waiting longer than 6 hours resulted in no further increment. Subjects had the option of not self-administering heroin or of taking less than the allowable dose.

blind conditions. After 6 drug-free days 8 subjects received an injection of 10 mg of heroin; 4 days later all subjects received intravenous saline 12 hours after pretreatment with either naltrexone (N=4) or an equivalent volume of placebo (N=4). After 10 days of access to heroin (see table 1) naltrexone-treated and placebo-treated subjects received another 10-mg dose of heroin. Plasma levels of LH and testosterone were determined on integrated blood samples collected at 30-minute intervals from 4 hours before to 10 hours after each test injection. Blood samples were collected with the use of a constant exfusion pump; thus hormonal levels are expressed as the mean value for each 30-minute collection period.

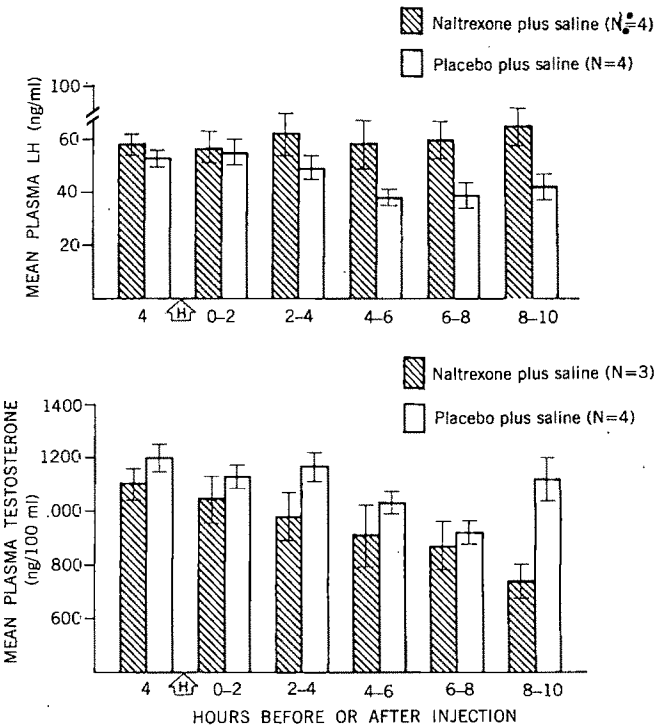
In general, pooled data for these subjects were consistent with our earlier findings. As illustrated in the top panel of figure 2 heroin-induced suppression of LH secretion was first evident 2–4 hours and was greatest 4–6 hours after unblocked heroin injection. In this study the degree of LH suppression was not as great as that observed previously. Since some subjects were given heroin after only 1 week on the research ward, compared with a 2-week drug-free period in the earlier studies, some may have been partially tolerant to the

FIGURE 2
Effects of Heroin (10 mg i.v.) Versus Isotonic Saline on Plasma Levels of LH and Testosterone (22)[a]



[a]In the unblocked heroin condition, accurate data were obtained in 6 of the 8 subjects; all injections were given at 2:00 p.m.

FIGURE 3
Effects of Heroin (10 mg i.v.) Versus Isotonic Saline on Plasma Levels of LH and Testosterone on Day 11 (Before Heroin Phase) (22)[a]



[a]Accurate data on plasma testosterone levels were obtained in 3 of the 4 naltrexone-treated subjects; all injections were given at 2:00 p.m.

effects of heroin on LH. The maximum decline in plasma testosterone occurred 6–8 hours after heroin injection (bottom panel figure 2). As in experiment 2, some recovery in LH secretion was noted 6 hours after heroin injection and was followed shortly by a rise in plasma testosterone.

As illustrated in figure 3 measurement of plasma LH in subjects on naltrexone or placebo also replicated our earlier finding—the naltrexone-treated subjects had consistently higher levels of LH, but their plasma testosterone levels were no different from the placebo-treated group. After 10 days of unblocked heroin use, there was some indication that tolerance developed to the drug's acute suppressant effect on LH and testosterone, but the timing of drug administration and blood sampling in this study did not allow for accurate assessment of this question. Investigation of tolerance development would require that the hormonal response to a single dose of heroin be measured on several consecutive days during a sustained period of fixed-dose, fixed-interval drug administration.

DISCUSSION

Opiate Effects on Sexual Functioning

There is ample clinical evidence that sexual drive and performance are diminished in male opiate addicts (6–11). Only recently, however, has a link been forged between these clinical observations and opiate-induced changes in the hypothalamic-pituitary-gonadal axis. Recent animal studies (24–26) and our data in man (21, 22) suggest that a prominent neuroendocrine effect of opiate administration is suppression of plasma LH secretion followed by a secondary decline in testosterone production. If the primary effect of heroin were on testosterone biosynthesis or metabolism, we would have expected a more rapid fall in plasma testosterone followed by an increase in plasma LH. This is the normal response to a fall in plasma testosterone and results from disinhibition of the tonic control exerted by testosterone on the release of luteinizing hormone releasing factor (LRF) from the hypothalamus. LRF is released in episodic bursts and, in turn, controls LH secretion from the pituitary (27–29). That acute heroin administration appears to completely inhibit episodic LH secretory pulses suggests a central effect on the release of hypothalamic LRF. In laboratory animals the regulation of LH (via LRF secretion) has been localized to ventral and medial areas of the hypothalamus (30), a region that also possesses numerous opiate receptor binding sites and a high concentration of endogenous opiate peptides (31).

In our studies pretreatment with a narcotic antagonist (naltrexone) not only blocked heroin-mediated suppression of LH release but actually increased the frequency of LH secretory bursts from the pituitary. As a result mean plasma LH levels were elevated in naltrexone-treated subjects. This finding can best be

explained in light of our advancing knowledge about the role of endogenous opioids (i.e., endorphins) and opiate receptors in the regulation of hormonal output. The endogenous opioids may play an inhibitory role in the normal regulation of LH secretion (32), and this inhibitory effect may be blocked by administration of a narcotic antagonist. One could also explain naltrexone's stimulation of LH release as a hormonal concomitant of precipitated abstinence; however, a recent double-blind study by Ellingboe and associates (33) revealed that naltrexone given to nonaddicts increased mean plasma LH levels by 50% over baseline, which again suggests the involvement of a hypothalamic-pituitary mechanism.

Finally, our studies indicated little effect of either heroin or naltrexone on plasma FSH secretion. Cicero and associates (24, 25) found the same dichotomous response to opiates in the rat. Their data suggest that those areas of the hypothalamus which influence the synthesis and release of pituitary LH may be more sensitive to the effects of opiates than those which mediate FSH levels. Although one study has demonstrated anatomic separation of the centers that control LH and FSH release in animals (34), there is no clear evidence of such a separation in man. Indeed, the decapeptide (LRF) that regulates pituitary release of LH probably plays a role in FSH release as well, although the pituitary response is usually of lower magnitude.

Correlation of Endocrine Changes with Mood States

Addicts often cite the acute euphorigenic and tension-relieving effects of opioids as important factors in the decision to initiate and maintain drug use. At the same time, laboratory studies of opiate use in man have consistently demonstrated that chronic administration of these substances is associated with the development of increased psychopathology and dysphoric mood states (35–38). Recently we have attempted to relate such events to changes in brain catecholamine levels (39), but the observed clinical phenomena are the result of many factors, including expectancy and the setting in which the data are gathered.

At present there are also sufficient data to explain the brief (30–60 seconds) but intensely pleasurable rush that immediately follows intravenous heroin administration. The mechanism most likely involves massive release of catecholamines at both central and peripheral sites. The rush is followed by a less intense, but more sustained, period of relaxation and tension relief lasting 2–4 hours. The strength and duration of this effect are influenced by the dose administered, the level of plasma morphine achieved (heroin [diacetylmorphine] is almost immediately converted to morphine by the liver), the rate of elimination of the drug, the degree of tolerance developed, and the conditioned and unconditioned stimulus properties of the environment (39–40). The time course of this effect closely parallels, and may be related to, the acute effects of heroin on neuroendocrine function—specifically, the acute suppression of LH release followed by a secondary fall in plasma testosterone. Indeed, heroin acts much like the antiandrogen compound cyproterone acetate, which suppresses the release of pituitary gonadotropins (especially LH), lowers testosterone levels, and reduces both sexual drive and performance (41). In low doses acute administration of cyproterone acetate alters the computerized electroencephalogram in a fashion similar to that of the tricyclic antidepressants; larger doses produce a profile much like the benzodiazepine anxiolytics (42). On the other hand, as with heroin, chronic administration of cyproterone acetate has been reported to produce apathy, restlessness, and depression (41). The similarity of these data to our observations in heroin users is striking. In contrast, acute administration of naltrexone raises LH levels in detoxified opiate addicts (21) and normal volunteers (33), while producing a dysphoric mood state and increased sexual drive (43).

Although no direct link has been demonstrated between sex hormone changes and temporally related alterations in mood, our data on the acute effects of heroin suggest a drive reduction model of opiate reinforcement, in which the drug acts as an antianxiety agent and reduces sexual drive and tension. Whether this effect is a direct consequence of acute suppression of LH and testosterone or is mediated through the same hypothalamic mechanisms that control LH release (i.e., alterations in catecholamine systems) is unclear. It is also possible that changes in other hormone systems (e.g., the hypothalamic-pituitary-adrenal axis) may also play a role.

With repeated heroin use, tolerance develops to the drug's acute positive effects on mood (38) and subjects manifest increasing apathy, irritability, depression, and social withdrawal (38, 40). This may be related to the development of tolerance to its acute suppressant effect on LH and testosterone or to alterations in the catecholamine systems that control mood and hormonal output. Nonetheless, chronic heroin users and patients on high doses of methadone still have low circulating levels of testosterone, a condition common to other groups who manifest high levels of anxiety and depression, such as soldiers under stress (44, 45), intoxicated alcoholics (46), and monkeys who have recently lost status (47). Although there are data which suggest that testosterone may exert a mood-elevating effect in man (48, 49), Sachar and associates (50) found no correlation between testosterone levels and mood.

The relationship between the subjective effects of opiate use and the neuroendocrine events that accompany these phenomena is still unclear. We do not yet understand the role of endorphins in the regulation of sex hormone secretion, the mediation of drive states, and the regulation of mood. We hope that future research will help to clarify these issues.

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Lithium Response in Good Prognosis Schizophrenia

BY JACK HIRSCHOWITZ, M.D., REGINA CASPER, M.D., DAVID L. GARVER, M.D., AND SIDNEY CHANG, M.D.

The authors report the results of a 2-week lithium trial using 31 patients meeting Research Diagnostic Criteria for schizophrenia or schizoaffective disorder who were also diagnosed according to DSM-III *criteria. One-third of the patients showed reduction of their schizophrenic-like symptoms during the lithium trial. Seven of the 9 responders (78%) met* DSM-III *criteria not for schizophrenia but for a schizophreniform disorder. Such patients also met McCabe and associates' criteria for good prognosis schizophrenia. This study lends further support to the growing body of evidence which suggests that schizophreniform disorder and good prognosis schizophrenia may be affective disorders with atypical schizophrenic-like features. The authors cautiously suggest that a lithium trial may be indicated in this subgroup of patients.*

There has been growing interest in the use of lithium in the schizophrenias, but reports on its efficacy in schizophrenic patients are conflicting (1, 2). One of the most patent difficulties in evaluating treatment results is the inconsistency in the diagnostic criteria used for schizophrenia and the so-called schizoaffective disorders. Alexander and associates (3), in a recently published study using the Research Diagnostic Criteria (RDC) of Spitzer and associates (4), demonstrated that some schizophrenic-like patients appear to respond to lithium; however, they were unable to find any RDC parameters that would discriminate responders. The present study is an attempt to identify diagnostically a subgroup of schizophrenic-like patients for whom a lithium trial is indicated. It is also, to our knowledge, the first study that has examined lithium response in relation to the *DSM-III* criteria for schizophreniform versus schizophrenic disorders.

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METHOD

Consecutive patients with a suspected diagnosis of schizophrenia were admitted to the psychobiology treatment unit. Only patients who then met the RDC for schizophrenia and schizoaffective disorder were admitted to the study. To meet the RDC, patients must have had at least two symptoms from a list that includes specific types of delusions, hallucinations, and thought disorder. The illness must not have been evidence of coarse brain disease, medical illness, or significant alcohol or drug abuse (5).

Study patients underwent a 2-week washout period. During the second week the baseline data, including the full Schedule for Affective Disorders and Schizophrenia (SADS), were collected. The SADS, developed by Spitzer and colleagues, is a standard interview that documents symptoms, both at the time of the interview and at the time during which the illness was at its worst for this episode, time course of illness, previous illnesses or episodes, and premorbid functioning. After completion of the SADS, a series of formal diagnoses was made for each patient by the research physicians according to the RDC (5), *DSM-III*, the good versus poor prognosis criteria of McCabe and associates (6, 7), and Taylor and Abrams' criteria for mania (8, 9). McCabe and associates' criteria emphasize differences in degree of recovery from previous episodes of major psychiatric illness. Taylor and Abrams' criteria for mania reclassified patients who met criteria for schizoaffective illness as manic on the basis of the presence of all four of the following features: hyperactivity; rapid or pressured speech; euphoric, expansive, or irritable mood with a broad affect; and absence of concurrent medical illness. Patients were excluded from the study if they met Taylor and Abrams' criteria for mania, resulting in a residual group of schizophrenic-like patients who would not typically be diagnosed as having mania or a variation of mania. Patients whose schizophrenic symptoms were mild as reflected by an initial modified New Haven Schizophrenia Index (NHSI) score of less than 12 (out of a possible score of 80) were also excluded.

because small changes in ratable symptoms in such patients could not be reliably estimated without artificial magnification and distortion of individual results.

Beginning during the second drug-free week, the Serial Modified NHSI was scored twice weekly by rating teams standardized with the instrument. The raters included a psychologist and social worker who did not participate in the original diagnostic workup. This instrument rates on a scale of 0–5, depending on the degree of severity, delusions, hallucinations, thought disorder (including bizarre reasoning, looseness or overinclusion, autism, and concreteness), disturbances of affect, confusion, paranoid ideation, and catatonic symptoms. The NHSI was originally designed for diagnostic purposes (10), but has been modified for use as a change rating scale (11, 12). The scale uses explicit criteria for identifying specific symptoms and rating their intensity. Furthermore, it has been used to rate psychotic thought disturbance at intervals of 20 minutes (13). The usefulness of the scale has been demonstrated in several studies that show relationships between blood levels of antipsychotic drugs and patient response (11, 12, 14). In addition to the NHSI, the Affective Disorders Rating Scale was scored twice weekly by a team of nurse-raters. Our interest in this latter instrument was primarily in the mania and depression scores.

On day 1 of the lithium trial, patients began a regimen of 1200 mg daily of lithium carbonate, which increased each second day by 300 mg until either toxicity developed or a plasma lithium level of 1.4 mEq/liter had been achieved. An attempt was made to maintain patients at between 1.1 and 1.4 mEq/liter. Blood levels of lithium were monitored three times weekly during the 2-week trial.

The absolute and percentage change during lithium treatment (days 1–14) was computed for each patient from the least squares linear regression of the biweekly NHSI (schizophrenic symptoms) and the Affective Disorders Rating Scale (affective symptoms scores). Improvement of greater than 30% on the NHSI was defined as response during the 2-week lithium trial. Analysis of variance was used to compare results among subgroups. Fisher's exact test was used to compare the relationships between lithium response and the diagnostic categories of the RDC, *DSM-III*, and McCabe and associates (6, 7).

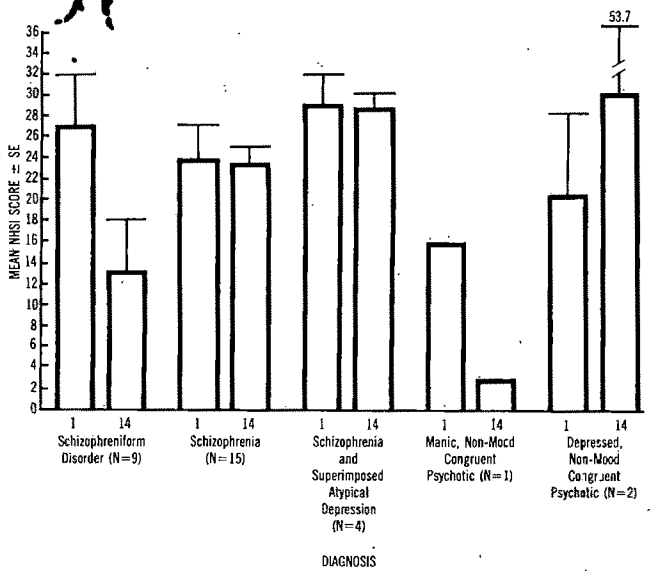
RESULTS

Of the 42 patients who entered the study, 11 were dropped before its completion. Two chronic paranoid schizophrenic patients were dropped because they had incorporated the medication into their delusional system and did not take medication on a regular basis, which resulted in fluctuating blood levels of medication. Five patients required more conventional antipsychotic drugs during the washout and baseline periods. Of these 5 patients, 3 had been symptomatic less than 6 months. Four patients required antipsychotic medication in addition to lithium during the early phase of drug treatment. All 4 had been ill for less than 6 months; all of them were subsequently discharged on lithium therapy, 2 of them on lithium alone. Such dropouts indicate that the study has been somewhat selective; we report data on only those patients who could tolerate 4 weeks without conventional antipsychotic drugs. Some of our most disturbed schizophrenic-like patients did not have the opportunity of a lithium trial.

The mean age of the 31 patients who completed the 2-week lithium trial was 25.3±1.1 years. There were 21 male patients and 10 female patients. During the baseline week the mean percentage change of schizophrenic symptomatology on the NHSI was 2.7±7.5%, which suggests that minimal change in symptoms occurred during the week before the initiation of lithium.

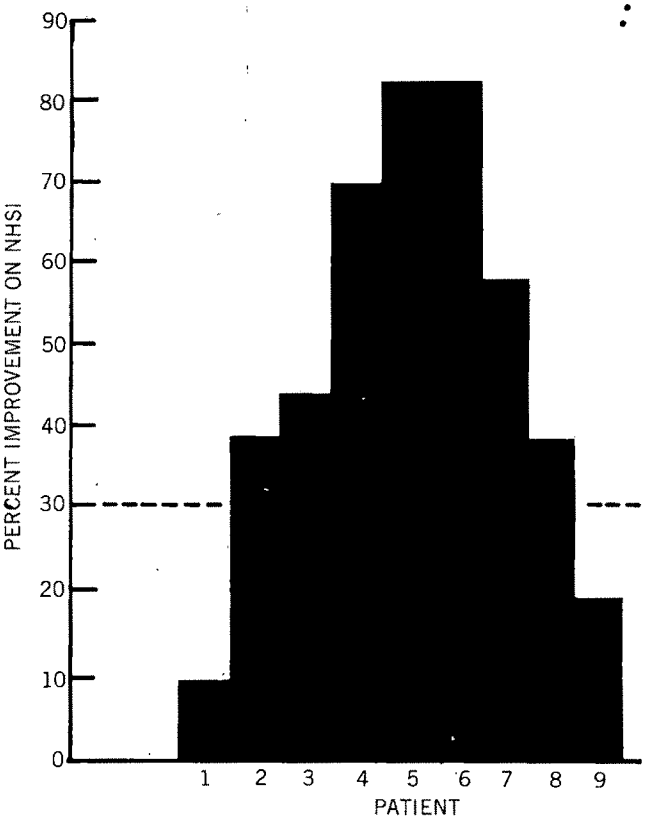
Figure 1 identifies the patients according to *DSM-III* diagnosis and shows mean NHSI scores for each diagnostic group on days 1 and 14 of the lithium trial. Nine patients met the *DSM-III* criteria for schizophreniform disorder. Schizophreniform disorder has the essential features of schizophrenia, except the duration is less than 6 months. This group showed a mean improvement score of 13 points, that is, a 49.7% reduction of baseline NHSI scores. The within-group variance of such reduction of NHSI scores for the schizophreniform group was significantly less than the total variance for all the groups ($p<.01$). Fifteen patients met the *DSM-III* criteria for schizophrenia. These criteria are essentially the same as those of the RDC, except that the illness must have lasted for at least 6 months. The mean improvement for this group between days 1 and 14 was 1 point on the NHSI, that is, a 0.6% reduction of baseline NHSI scores. Four patients met the *DSM-III* criteria for schizophrenia with a superimposed atypical depression. They met *DSM-III* criteria for schizophrenia but had for a short period of time a superimposed depressive syndrome. Once again there was minimal change for this group, with a mean deterioration of 1 point on the NHSI—that is, a 1.5% increase of baseline NHSI scores. The last two categories comprised 3 patients who although meeting the RDC for entrance into the study were rediagnosed according to *DSM-III* as having an affective illness rather than schizophrenia or schizoaffective disorders. These patients had so-called "non-mood congruent psychotic symptoms" superimposed on a major affective illness. Non-mood congruent psychotic symptoms refers to those symptoms which are schizophrenic-like but the dominant syndrome is an affective one. One patient was rediagnosed according to *DSM-III* as manic, non-mood congruent; he responded dramatically to lithium. The 2 remaining patients were depressed, non-mood congruent; one of them showed a non-

FIGURE 1
Mean Scores of 31 Patients on the New Haven Schizophrenia Index (NHSI) on Days 1 and 14 of Lithium Treatment, According to *DSM-III* Diagnosis[a]



[a]F=4.71, $df_1=4$, $df_2=26$, $p<.01$ (analysis of variance).

FIGURE 2
Individual Percentage Improvement on the New Haven Schizophrenia Index (NHSI) in 9 Schizophreniform Patients Between Days 1 and 14 of Lithium Treatment



significant improvement during the lithium trial, and the other showed a marked deterioration during the lithium trial.

Figure 2 illustrates the percentage change on the NHSI for each of the 9 patients given a diagnosis of schizophreniform disorder by *DSM-III* criteria. Seven of these patients showed a greater than 30% improvement on the NHSI during the 2-week lithium trial. All 9 were discharged on lithium therapy; 2 patients required neuroleptic medication in addition because of residual schizophrenic-like symptoms that appeared to persist with lithium alone. One patient who made an 8% improvement during the 2-week lithium trial subsequently improved dramatically during the third week while receiving lithium alone and was also discharged on lithium therapy.

Figure 3 illustrates the percentage change on the NHSI for each of the 19 patients diagnosed as having schizophrenia or schizophrenia with a superimposed atypical depression. Only 1 of these patients showed a greater than 30% improvement on the NHSI.

It is interesting to examine the relationship among the *DSM-III* diagnoses, the RDC diagnoses, and McCabe and associates' good versus poor prognosis groups. The 9 patients who met *DSM-III* criteria for schizophreniform disorder included 5 RDC schizophrenic patients and 4 RDC schizoaffective patients, 1 schizoaffective-manic and 3 schizoaffective-depressed patients. All *DSM-III* schizophreniform patients met McCabe and associates' criteria for good prognosis schizophrenia.

All 15 of the *DSM-III* schizophrenic patients also met the RDC for schizophrenia; all similarly met McCabe and associates' criteria for schizophrenia of poor prognosis.

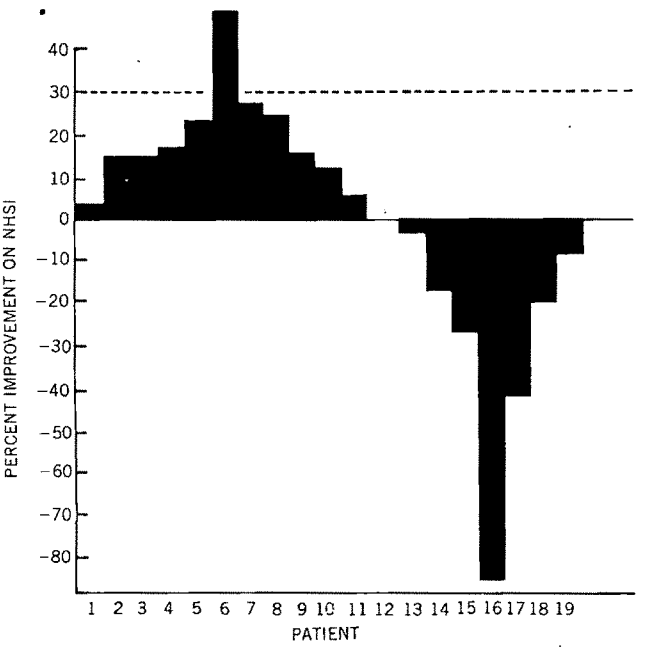
The 4 patients meeting *DSM-III* criteria for schizophrenia with superimposed atypical depression met RDC criteria for schizoaffective depressed and were poor prognosis schizophrenic patients according to the criteria of McCabe and associates.

There were no *DSM-III* schizoaffective patients in this sample of 31 schizophrenic-like subjects.

The single *DSM-III* manic, non-mood congruent psychotic patient was an RDC schizoaffective, manic patient and a good prognosis schizophrenic patient according to McCabe and associates' criteria. The 2 *DSM-III* depressed, non-mood congruent psychotic patients were RDC schizoaffective, depressed patients and poor prognosis schizophrenic patients according to McCabe and associates' criteria.

With respect to therapeutic response during the 2-week lithium trial, within the *DSM-III* groups (excluding the affective non-mood congruent groups) 7 of 8 lithium-responding patients could be identified as members of the schizophreniform group, while 17 of 20 lithium-nonresponding patients could be identified as having schizophrenia or schizophrenia with super-

FIGURE 3
Individual Percentage Improvement on the New Haven Schizophrenia Index (NHSI) in 19 Patients with Schizophrenia and Schizophrenia with Superimposed Atypical Depression



imposed atypical depression ($p<.0005$, Fisher exact probability test).

Using the RDC for schizophrenia or schizoaffective disorder to preselect patients for the subsequent good versus poor prognosis classification of McCabe and associates, 8 of 10 good prognosis patients responded during the lithium trial, while 20 of 21 poor prognosis patients had no response during a similar lithium trial ($p<.0001$, Fisher exact probability test, two-tailed).

In contrast, RDC diagnosis of schizophrenia versus schizoaffective disorder did not differentiate between lithium responders and nonresponders. Five of 20 schizophrenic patients responded during the lithium trial; four of 11 schizoaffective patients similarly responded ($p>.5$, Fisher exact probability test, two-tailed).

The depression and mania subscales of the Affective Disorders Rating Scale were examined for 6 of the 9 *DSM-III* schizophreniform patients who completed the lithium trial in order to determine whether the antipsychotic effect of lithium in this group of patients was paralleled by a reduction in depression or mania ratings. No relationship was found between changes in the NHSI and changes in either the mania or depression subscores of the Affective Disorders Rating Scale during the lithium trial.

DISCUSSION

Beginning with a group of 31 patients who met the RDC for schizophrenia or schizoaffective disorder, we rediagnosed such patients according to *DSM-III* and McCabe and associates' good versus poor prognosis criteria for schizophrenia. Each of the patients received a 2-week trial of lithium, with plasma levels greater than 1.0 mEq/liter. Good prognosis schizophrenic patients (McCabe and associates) and patients with schizophreniform disorder (*DSM-III*) showed amelioration of their schizophrenic-like symptoms during the lithium trial. More common diagnostic criteria, based primarily on symptoms, such as the schizophrenia versus schizoaffective distinctions on the RDC, failed to differentiate lithium responders from nonresponders.

Several studies reviewing the characteristics of nondeteriorating schizophrenia and schizoaffective syndromes have elicited the common features of these disorders (14–21). These include good premorbid adjustment, the presence of a precipitating event, acute onset, presence of confusion or excitement, and an affective heredity. In their recent review Pope and Lipinski (22) suggested that affective-like course (exacerbation and full remission) of schizophrenic-like symptoms and family history of affective disorders may differentiate those patients who may benefit from lithium better than symptoms do. Our work supports the finding that acute onset and good premorbid adjustment are indicators of lithium response, but we were not able to find that affective symptoms are in any way related. In addition, we found that *DSM-III*, which is a clinical diagnostic instrument, discriminated better than the RDC, which is a research diagnostic instrument, in predicting lithium response.

Thus there appears to be a growing body of evidence which suggests that schizophrenia of good prognosis and a substantial number of the schizophrenia disorders may be lithium-responsive affective disorders masquerading phenomenologically as schizophrenic-like illnesses. Our data support this notion and suggest that a lithium trial may be indicated in such patients. Firm conclusions and recommendations must await the evaluation of such schizophreniform illnesses and lithium response using double-blind, placebo-controlled methodology to rule out the possibility that spontaneous remission regularly occurs in such patients during weeks 3 and 4 of hospitalization.

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"Schizoaffective Disorder": An Invalid Diagnosis? A Comparison of Schizoaffective Disorder, Schizophrenia, and Affective Disorder

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The authors compared patients meeting widely accepted criteria for the diagnosis of schizoaffective disorder, manic type, with patients meeting rigorous criteria for manic disorder and schizophrenia, using three methods of validation: family history, short-term treatment response, and long-term outcome. No significant differences were found between patients with manic disorder and schizoaffective disorder. However, consistent and often highly significant differences separated patients with schizophrenia from those with manic disorder and schizoaffective disorder. The findings suggest that schizoaffective disorder, as currently defined, *is not a valid and independent entity. The authors suggest that psychotic disorders not diagnosable as manic-depressive illness or schizophrenia and without apparent organic basis would best be called "undiagnosed" or "atypical" psychosis. Further, while proposals for new diagnoses or for subtyping of schizophrenia or manic-depressive illness should be encouraged, these should undergo rigorous screening for validity before being accepted into clinical use.*

The diagnosis and nosology of psychotic disorders have been matters of interest and debate since Kraepelin's original delineation of manic-depressive illness and schizophrenia (1, 2). Although in the past it was largely of academic interest, the nosology of these disorders has now assumed greater importance, as the availability of newer and more specific treatments for the psychoses has required more accurate diagnosis and classification of patients. Furthermore, growing research into the etiology and treatment of the psychoses demands that carefully selected, homogeneous populations of patients be available for study. It is thus crucial to develop a nosology of these disorders in which each diagnostic entity is shown to be valid.

How does one establish the validity of a psychiatric diagnosis? Lacking the histological, pathological, or biochemical evidence used to validate diagnostic entities in most branches of medicine, psychiatry must rely on other validating criteria. These have been reviewed by Robins and Guze (3), who enumerated five criteria for establishing diagnostic validity in psychiatric illness: clinical description, laboratory studies, delimitation from other disorders, follow-up study, and family study. Only by the constant and rigorous testing of the validity of proposed diagnostic entities, using criteria such as these, can we develop a truly useful classification of psychiatric disorders.

Currently, fairly well-validated criteria exist for the diagnosis of schizophrenia and manic-depressive illness, including the criteria of Feighner and associates (4), the Research Diagnostic Criteria (RDC) (5), and the criteria of the 1978 and 1979 drafts of *DSM-III*. However, it is widely recognized that many patients manifest psychotic syndromes difficult to classify under schizophrenia or manic-depressive illness, for example, the patient who manifests an acute psychosis with both "affective" and "schizophrenic" symptoms. Should such patients' illnesses simply be classified as undiagnosed psychosis, should they be subsumed under an already validated diagnosis (i.e., schizophrenia or manic-depressive illness), or can they be shown to represent a valid diagnostic group in their own right?

Many diagnostic terms have been proposed to describe such patients; in the United States one of the most popular has been schizoaffective (6). Schizoaffective disorder has been variously proposed to represent a subtype of schizophrenia (as in *DSM-II*), a subtype of manic-depressive illness (7), or a separate entity (8, 9). The diagnosis of schizoaffective has been in use for nearly 50 years, despite a lack of evidence to establish its validity. Does schizoaffective disorder merit distinction as a valid diagnostic category?

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A useful design for testing the validity of the schizoaffective diagnosis is to select three groups of patients: 1) a study group of putative schizoaffective patients who display a defined combination of affective and "schizophrenic" symptoms and two narrowly defined reference groups of 2) "definite" schizophrenic patients and 3) "definite" manic-depressive patients. The study group is then compared with each of the two reference groups on a wide range of available validating criteria such as demographic indices, treatment response, family history, and long-term outcome. A finding of consistent, significant differences between the schizoaffective study group and a given reference group would suggest that the two groups represent different disorders, but a finding of minimal, nonsignificant differences would suggest that the two groups may represent the same disorder.

In a recent study that closely approaches this design, Abrams and Taylor (10) compared 78 patients who met the RDC for manic disorder (5) with 10 patients who also met the RDC for manic disorder but in addition displayed one or more of Schneider's first-rank symptoms (11). This latter group would be defined as having schizoaffective disorder, manic type by both the RDC and in the 1978 draft of *DSM-III*. No significant differences could be found between the manic and schizoaffective patients on any variables related to prognosis, premorbid history, family history, or treatment response. These results would seem to question the validity of the schizoaffective diagnosis and suggest that schizoaffective disorder might be indistinguishable from manic disorder.

A problem with this study noted by the authors is that it included only 10 schizoaffective patients. This causes a risk of a so-called type II error—failure, as a result of small sample size, to demonstrate significant differences between two groups when in fact real differences exist. A second problem is the lack of long-term follow-up: it would be of interest to know whether the two groups were distinguishable in any way several years later.

Three other studies used similar designs to investigate groups of patients who were diagnosed as schizophrenic but who displayed good prognostic criteria such as affective symptoms (12–14), an acute onset of illness (12–14), and/or a past history of a remitting illness (14). A number of these study patients would probably have been diagnosed schizoaffective in many centers. We summarized the results of these three studies in table 5 of a previous report (15). Briefly, all three studies found the good prognosis schizophrenic patients indistinguishable from reference groups of pure bipolar manic-depressive patients on virtually all indices, including demographic data, treatment response, and family history, but significantly different from the reference group of schizophrenic patients on many of the same indices.

However, the first two of these three studies were based on the same series of admissions as was the study of schizoaffective disorder previously described (10). Thus, three of the four studies represent the work of the same group at the same center, which makes the generalizability of the findings less certain.

Several additional studies have used the single validating criterion of family history to compare "schizoaffective" and similarly defined "good prognosis schizophrenic" patients with reference groups of poor prognosis schizophrenic patients. These are summarized in table 3 of our previous review (15). These studies consistently found a preponderance of schizophrenia in the relatives of poor prognosis schizophrenic patients, but a preponderance of affective illness in the relatives of schizoaffective and good prognosis schizophrenic patients, which again suggests that the latter groups represent a disorder separate from poor prognosis schizophrenia.

The present study further tests the validity of the schizoaffective diagnosis, using the widely recognized RDC (5) (which closely resemble the 1978 draft of *DSM-III* criteria) to define patient groups. In order to minimize the possibility of a type II error, we examined a relatively large number of schizoaffective patients. We also conducted a 1½- to 5-year follow-up for most patients. We examined whether the validating criteria of treatment response, family history, and long-term follow-up would significantly distinguish schizoaffective disorder from schizophrenia and/or from affective disorder. Alternatively stated, we asked whether, in patients displaying a full manic or depressive syndrome, the presence or absence of putative schizophrenic symptoms (especially the Schneiderian first-rank symptoms) would have any effect on treatment response, family history, or long-term outcome.

METHOD

We reviewed the charts of 219 consecutively admitted patients to Bowditch I, a research ward for the study of psychotic disorders at McLean Hospital in Belmont, Mass. This group represented all of the patients admitted during the period from January 1, 1974, to July 1, 1977. Charts contained detailed admission notes, mental status examinations, physicians' progress notes, case reports, and daily notes by the trained nursing staff of the research unit. In addition, most of the 219 patients were directly known to one or another of us from our work on the unit. Thus, in spite of our retrospective approach, there were extensive data on each patient.

We diagnosed the patients using the RDC (5) (which closely resemble the criteria of the 1978 draft of *DSM-III*). However, for the reference group of schizophrenic patients we required 6 months of illness, as noted in the *DSM-III* criteria, rather than only 2 weeks as required in the RDC. This was done in order to ensure

TABLE 1
Research Diagnostic Criteria Diagnoses of 219 Consecutively Admitted Patients

Diagnosis	N	%
Schizophrenia	41	18.7
Schizoaffective disorder, manic type	52	23.7
Schizoaffective disorder, depressed type	9	4.1
Manic disorder	34	15.5
Major depressive disorder	4	1.8
Unspecified functional psychosis	12	5.5
Other disorders (psychoses of possible organic etiology, factitious psychoses, psychoses of less than 1 week's duration, and nonpsychotic disorders)	67	30.6

that the reference group of schizophrenic patients would be as strictly defined as possible. The distribution of diagnoses of the 219 patients is shown in table 1.

Three diagnoses—schizophrenia, schizoaffective disorder, manic type, and manic disorder—collectively accounted for 57.9% of the patients. The paucity of depressed patients on the unit was due to the fact that most such patients were routinely sent to two other units. The large number of patients classified in the category of other disorders included many who were disqualified because of a possible organic etiology (such as abuse of hallucinogens or sympathomimetic substances), plus a not inconsiderable number of patients (N=13) who we felt displayed factitious psychotic disorders.

The 127 patients fulfilling RDC for schizophrenia, schizoaffective disorder, manic type, and manic disorder were selected for study. These groups were compared using three validating criteria: 1) family history of schizophrenia, affective disorder, and alcoholism; 2) short-term treatment response; and 3) long-term outcome at 1½ to 5 years after discharge.

Family history was rated by abstracting available information about ill relatives. These data were later scored by one of us (H.G.P.), who was blind to the diagnosis of the proband, using the criteria of Winokur and associates (16) for the diagnosis of family members.

Short-term treatment response was scored directly from the wording used in the physician's discharge summary in order to minimize any bias in our own retrospective assessment of short-term response. Patients were divided into three categories: 1) those rated "worse," "unimproved," or "slightly improved" by their physicians; 2) those rated "moderately improved"; and 3) those rated "markedly improved" or "in remission."

Long-term outcome was rated by one of us (D.T.A.), who was blind to the research diagnosis that had been assigned to the patient. However, blindness to diagnosis in this instance was only partial since the outcome rater had seen many of the patients earlier during their index admissions. Outcome data were obtained by telephone or personal contact with the patient, information from patients' therapists, and review of the records of hospital admissions subsequent to the index admission. Outcome was evaluated for the entire follow-up period since the index admission (i.e., 1½ to 5 years). Four indices were defined and rated:

1. Best social function at any time during the follow-up period.
2. Maximum occupational or academic level at any time during the follow-up period.
3. Lowest level of residual symptoms at any time during the follow-up period (residual symptoms were defined as the eight symptoms listed under part C of the *DSM-III* criteria for schizophrenic disorder).
4. Global assessment of outcome for the period as a whole.

Each of the four indices was rated on a 5-point scale (0=poor, 1=fair, 2=moderately good, 3=very good, 4=excellent).

Thus, by abstracting and blind scoring family histories, using treating physicians' ratings of outcome, and by partially blind scoring of long-term outcome, we attempted to minimize the effects of bias in any of the three validating criteria employed.

RESULTS

Family History

Table 2 indicates the number of patients in each of the three diagnostic groups (schizophrenia, schizoaffective disorder, manic type, and manic disorder) found to have a positive family history for schizophrenia, affective illness, alcoholism, and psychotic disorders for which data were inadequate to permit a diagnosis. In some instances, we felt that the family of a given proband contained a possible rather than a definite case of affective illness or alcoholism; such cases are shown separately in the table.

A positive family history for schizophrenia occurred in 9.8% of schizophrenic probands and none of the schizoaffective and manic probands. Using a one-tailed t test, this significantly differentiated schizophrenia from schizoaffective disorder, manic type ($p=.04$) and showed a similar but nonsignificant trend ($p=.09$) in comparing schizophrenia with manic disorder. The low incidence of familial schizophrenia in our study, even in the families of patients with schizophrenia, is consistent with the findings of a number of other investigations (11–14, 17).

A positive family history for affective illness significantly differentiated schizophrenia from schizoaffective disorder, manic type, and from manic disorder. However, there was no significant difference in family history of affective illness between schizoaffective disorder, manic type, and manic disorder. These findings hold true whether or not "possible" cases are included.

In summary, then, the validating criterion of family history suggests that schizoaffective disorder, manic

TABLE 2
Family History Findings in 127 Patients Diagnosed Retrospectively According to the Research Diagnostic Criteria

Diagnosis of Proband	Probands with Positive Family History[a]							
	Schizophrenia[b]		Affective Illness[c]		Alcoholism[d]		Psychotic Disorder; Inadequate Data to Rate	
	N	%	N	%	N	%	N	%
Schizophrenia (N=41)	4	9.8	4	9.8	4	9.8	3	7.1
Including relatives with possible cases	4	9.8	4	9.8	6	14.3	3	7.1
Schizoaffective disorder, manic type (N=52)	0		21	40.4	12	23.1	5	9.6
Including relatives with possible cases	0		26	50.0	13	25.0	5	9.6
Manic disorder (N=34)	0		11	32.4	11	32.4	1	2.9
Including relatives with possible cases	0		15	44.1	11	32.4	1	2.9
Schizoaffective disorder, manic type with 2 or more schizophrenic symptoms (N=33)	0		16	48.5	8	24.2	3	9.1
Including relatives with possible cases	0		19	57.6	9	27.3	3	9.1

[a]Some probands had a positive family history of more than 1 disorder.
[b]Schizophrenia versus schizoaffective disorder, p=.04; schizophrenia versus schizoaffective disorder with 2 or more schizophrenic symptoms, p=.09; schizophrenia versus manic disorder, p=.09 (all binomial theorem, one-tailed t test). All other differences were not significant.
[c]Schizophrenia versus both schizoaffective groups, p<.001 (regardless of whether possible cases are included); schizophrenia versus manic disorder, p<.05 (excluding possible cases) and p<.001 (including possible cases) (all by chi-square analysis). All other differences were not significant.
[d]Schizophrenia versus manic disorder, p<.05 (excluding possible cases) and n.s. (including possible cases) (chi-square analysis). All other differences were not significant.

TABLE 3
Short-Term Treatment Response Among 127 Patients Diagnosed Retrospectively According to the Research Diagnostic Criteria

Diagnosis and Treatment	Improvement						Total Number
	Minimal		Moderate		Marked		
	N	%	N	%	N	%	
Schizophrenia[a]	19	46.3	19	46.3	3	7.3	41
Neuroleptics	18		19		3		40
No medication	1		0		0		1
Schizoaffective disorder, manic type	6	11.5	8	15.4	38	73.1	52
Lithium	1		3		16		20
Lithium and neuroleptics	2		2		10		14
Neuroleptics	2		2		8		12
Lithium and antidepressants	0		0		2		2
ECT	0		1		1		2
No medication	1		0		1		2
Manic disorder	1	2.9	6	17.6	27	79.4	34
Lithium	0		1		12		13
Lithium and neuroleptics	0		5		12		17
Neuroleptics	1		0		2		3
ECT	0		0		1		1
Schizoaffective disorder with 2 or more schizophrenic symptoms	5	15.2	4	12.1	24	72.7	33
Lithium	1		1		9		11
Lithium and neuroleptics	2		1		8		11
Neuroleptics	2		2		5		9
ECT	0		0		1		1
Lithium and tricyclic antidepressants	0		0		1		1
No medication	0		0		0		0

[a]Schizophrenia versus all three other groups, p<.001 (chi-square analysis). All other differences were not significant.

type, is indistinguishable from manic disorder but significantly different from schizophrenia.

One possible objection that might be raised at this point is that schizoaffective patients are too loosely defined in the RDC; the presence of even one Schneiderian first-rank symptom, or preoccupation with a single delusion or hallucination unrelated to mood, is enough to shift the diagnosis from manic disorder to schizoaffective disorder, manic type. What if one were to examine the more narrowly defined subgroup of schizo-

TABLE 4
Outcome (1½–5 Years) Among 127 Patients Diagnosed Retrospectively According to the Research Diagnostic Criteria

	Outcome[a]					
	Poor-Fair		Good		Excellent	
Outcome Index and Diagnosis	N	%	N	%	N	%
Best social function						
Schizophrenia (N=27)[b]	18	66.7	8	29.6	1	3.7
Schizoaffective disorder, manic type (N=35)	3	8.6	7	20.0	25	71.4
Manic disorder (N=18)	0		6	33.3	12	66.7
Schizoaffective disorder, manic type, with 2 or more schizophrenic symptoms (N=24)	3	12.5	4	16.7	17	70.8
Best occupational or academic function						
Schizophrenia (N=27)[b]	15	55.6	8	29.6	4	14.8
Schizoaffective disorder, manic type (N=35)	1	2.9	9	25.7	25	71.4
Manic disorder (N=18)	0		5	27.8	13	72.2
Schizoaffective disorder, manic type, with 2 or more schizophrenic symptoms (N=24)	1	4.2	8	33.3	15	62.5
Minimum residual symptoms						
Schizophrenia (N=27)[b]	11	40.7	15	55.6	1	3.7
Schizoaffective disorder, manic type (N=35)	1	2.9	13	37.1	21	60.0
Manic disorder (N=18)	0		4	22.2	14	77.8
Schizoaffective disorder, manic type, with 2 or more schizophrenic symptoms (N=24)	1	4.2	10	41.7	13	54.2
Global assessment of outcome						
Schizophrenia (N=27)[b]	16	59.3	11	40.7	0	
Schizoaffective disorder, manic type (N=35)	3	8.6	23	65.7	9	25.7
Manic disorder (N=18)	1	5.6	9	50.0	8	44.4
Schizoaffective disorder, manic type, with 2 or more schizophrenic symptoms (N=24)	2	8.3	16	66.7	6	25.0

[a]Poor-fair outcome is defined as a rating of 0–1, good=2–3, and excellent=4 for best social function, best occupational or academic function, and global assessment of outcome. For minimum residual symptoms, poor-fair indicates severe symptoms, good indicates moderate symptoms, and excellent indicates no symptoms.
[b]Schizophrenia versus all other groups, $p<.001$ (chi-square analysis). All other differences were not significant.

affective patients who showed not one but several Schneiderian or mood-incongruent psychotic symptoms? Would this group still prove indistinguishable from patients with manic disorder?

To investigate the possibility, we examined the subgroup of 33 schizoaffective patients who displayed two or more of the Schneiderian or mood-incongruent psychotic symptoms from the list of symptoms considered suggestive of schizophrenia in criterion C of the RDC for schizoaffective disorder, manic type. Eighteen of these patients showed three or more of such symptoms and 6 showed five or more. However, even this more narrowly defined subgroup proved indistinguishable from manic disorder on all family history indices, but significantly distinguishable from schizophrenia on family history of manic-depressive illness ($p<.001$), and showed a nonsignificant trend on family history of schizophrenia ($p=.09$, one-tailed t test).

Short-Term Treatment Response

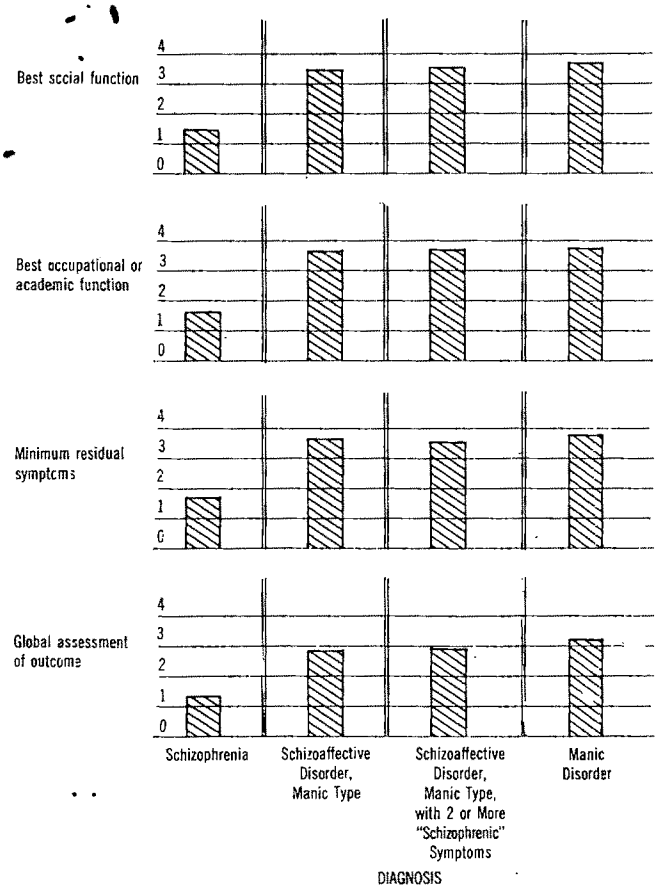
Table 3 presents data on short-term treatment reponse. Medications shown represent those the patient was receiving at the time of discharge. Based on this criterion, a highly significant difference separated the schizoaffective patients from the schizophrenic patients. However, no difference was found between schizoaffective patients and manic patients. Looking again at the subgroup of schizoaffective patients with 2 or more schizophrenic symptoms (i.e., Schneiderian first-rank or mood-incongruent psychotic symptoms) there was a highly significant difference between them and the schizophrenic patients, but not between them and the manic patients. Looking within the various groups at specific medication regimens (for example, lithium alone), there was little difference between the manic, schizoaffective, and schizoaffective patients with 2 or more schizophrenic symptoms.

Thus these findings, like the family history findings, support the similarity of schizoaffective disorder, manic type, and manic disorder, but they suggest the heterogeneity of schizoaffective disorder and schizophrenia.

Long-Term Outcome

Data on long-term outcome are shown in table 4 and figure 1. It is important to reiterate that the indices of social function, occupational or academic function, and residual symptoms rate best functioning at any time during the follow-up interval of 1½ to 5 years. Thus, an individual with several episodes of severe illness, separated by complete remissions, would tend to score high on these indices; an individual with chronic mild symptoms would score lower. On the other hand, global assessment of outcome represents an overall assessment of the entire follow-up period; an individual with several episodes of severe illness would thus

FIGURE. 1
Mean Ratings on Four Outcome Indices of 127 Patients Diagnosed as Having Schizophrenia, Schizoaffective Disorder, Manic Type, and Manic Disorder[a]



[a]0=poor, 1=fair, 2=moderately good, 3=very good, 4=excellent. Mean rating values are available from Dr. Lipinski on request.

score lower, however complete his or her remissions between episodes, than an individual with only a single major episode.

Of the 41 patients with schizophrenia, adequate information was obtained to rate long-term outcome in 27 of them (66%). Eighteen of the patients with manic disorder (53%), 35 of the patients with schizoaffective disorder (67%), and 24 of the patients with schizoaffective disorder with two or more schizophrenic symptoms (73%) could be rated for outcome. There was no significant difference between the proportions of patients in each group for whom adequate outcome data were obtained.

On all four outcome indices, the schizophrenic patients displayed strikingly and significantly poorer outcome than any of the other groups. However, no significant difference was found on any index between schizoaffective patients and manic patients. Nor did any significant differences emerge between the manic patients and the schizoaffective patients with two or more schizophrenic symptoms.

DISCUSSION

In order to test the validity of the diagnosis of schizoaffective disorder, manic type, patients meeting widely published criteria for this disorder (reference 5 and 1978 draft of *DSM-III*) were compared to patients meeting rigorous criteria (reference 5 and 1978 draft of *DSM-III*) for manic disorder and schizophrenia, using three validating methods: family history, short-term treatment response, and long-term outcome.

On all three validating criteria, no significant differences were found between manic disorder and schizoaffective disorder, manic type. No significant differences were found even in comparisons between patients with manic disorder and schizoaffective patients who displayed two or more schizophrenic symptoms (i.e., Schneiderian first-rank symptoms, preoccupation with mood-incongruent delusions or hallucinations, or thought disorder persisting in the absence of affective disturbance). On the other hand, on the same three validating criteria, consistent, often highly significant differences emerged separating patients with schizophrenia from both schizoaffective and manic patients.

Our findings suggest the following.

1. Schizoaffective disorder, manic type (as it is defined by two sets of well-known published criteria [reference 5 and 1978 draft of *DSM-III*]), is readily distinguishable from schizophrenia but indistinguishable from manic disorder using available validating criteria. This suggests that schizoaffective disorder, manic type, is not a valid and independent diagnostic entity but rather should be classified within manic disorder.

2. In patients displaying a full manic syndrome, the presence or absence of Schneiderian first-rank symptoms or of mood-incongruent psychotic symptoms was of no demonstrable diagnostic or prognostic significance in this study. This finding adds to the mounting evidence, from a number of recent studies (15), that Schneiderian first-rank symptoms and other putative schizophrenic symptoms are nonspecific symptoms, occurring frequently in both affective illness and schizophrenia and having little, if any, demonstrated significance in differential diagnosis.

Several limitations in the present study suggest avenues for further investigation. First, the number of depressed patients in our study (4 with major depressive disorder and 9 with schizoaffective disorder, depressed type) is too small to permit conclusions. Our findings for the two groups of depressed patients closely paralleled those for the patients with manic and schizoaffective disorder, manic type, but the numbers were far too small to reach significance. A larger study with depressed patients would be of interest.

Second, the restrospective design of the present study limits the accuracy attainable in some ratings. Some phenomenological or family-history data (especially familial alcoholism) may have been missed in the

retrospective approach. Complete researcher blindness was also impossible to achieve, although partial blindness was attained. To improve on this, we are currently conducting such a prospective study of consecutive admissions in which the admitting diagnosis, family history, short-term treatment response, and long-term outcome are determined by four independent observers, each blind to the ratings of the other three.

Third, the present study examines only one definition of schizoaffective disorder—that of the RDC (5) and the 1978 draft version of *DSM-III*. The final version of *DSM-III* redefines schizoaffective disorder as a residual category, without specific criteria. These narrower, but less specific criteria for schizoaffective disorder are more difficult to test, since it is much harder to get different observers to agree reliably as to which patients merit the newly defined schizoaffective diagnosis. Thus validation of these criteria may prove extremely difficult.

In summary, while the diagnostic separation of schizophrenia from schizoaffective disorder and manic disorder appears to be valid, the diagnostic separation of schizoaffective disorder from manic disorder does not appear to be valid as judged by available criteria. Since course, family history, and treatment response do not distinguish schizoaffective disorder as defined here from manic disorder, there would seem to be no adequate reason on clinical grounds to make the former diagnosis. Such a diagnosis can only lead to confusion.

From this discussion it should not be concluded that schizophrenia or manic-depressive illness necessarily represent unitary, homogeneous diagnostic entities, but rather that no other divisions or diagnostic groupings as yet appear to have proven diagnostic validity. Thus at present it appears that the diagnosis of schizoaffective disorder should be suspended and that individuals without toxic, metabolic, or factitious psychosis who do not fit the above categories would best be given a diagnosis of atypical psychosis or undiagnosed psychosis. Finally, proposals for new diagnostic groups and subgroups should be encouraged, but should undergo rigorous screening for validity before being accepted into clinical use.

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Neuropsychology in Alcoholic Men in Their Late Thirties: One-Year Follow-Up

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In a previous article the authors reported on two groups of alcoholic men in their late 30s who were remarkably free of neuropsychological impairment. In this article they present the results of a 1-year follow-up of many of these subjects. They found similarities in performance between alcoholic subjects who in the original study had been abstinent for 18 months and nonalcoholic control subjects who drank moderately. However, the group of alcoholic men who in the original study had been recently detoxified (about 3 weeks) failed to show practice-effect learning on follow-up. The authors interpret this failure as possibly indicative of a preclinical phase of what might ultimately become organic impairment.

In 1979 we reported on two groups of alcoholic men in their late 30s who were remarkably free of neuropsychological impairment (1). Furthermore, we found that we could not distinguish between alcoholic men who had been recently detoxified (3 weeks) and those who had been abstinent for 18 months and a control group of subjects who drank moderately on the basis of an extended Halstead-Reitan Neuropsychological Battery (2). We found no evidence from life history, neurological history, drug history, nutritional history, or drinking history (3) which suggested that our subjects were either at special risk for or would enjoy special immunity from alcohol-induced cerebral impairment. In this article we will present the results of a 1-year follow-up of these groups.

METHOD

A comprehensive description of the study design can be found in the initial report (1). In brief, we performed extended Halstead-Reitan neuropsychological examinations on three groups of men at the beginning of the study. Group 1 consisted of inpatients from a Veterans Administration alcoholism treatment unit who had been in the program for 3 weeks. Group 2 were abstinent alcoholic men who had remained sober for 18 months. Group 3 was composed of nonpatients who were recruited from various civic and educational organizations and screened against criteria that would eliminate subjects with "neuropsychological risk" conditions (3). Any subject with a history of neurological disorder, psychosis, or polydrug abuse was excluded, as were subjects older than age 46.

Approximately 1 year after the initial examination we repeated the neuropsychological tests and gathered interim data on health, accident, nutritional, alcohol, and drug abuse histories. There were 23 men in the follow-up group 1 (we will maintain the original group labels even though the "recently detoxified" men had been detoxified for 1 year and 3 weeks at follow-up). The mean age of the men in group 1 was 36.8±6.5 and the mean number of years of education was 12.9±2.0. Group 2 (who had originally been abstinent for 18 months) consisted of 25 men whose mean age was 36.5±6.3 and who had a mean of 13.2±2.7 years of education. In group 3 were 21 nonalcoholic men aged 36.9±5.9 with 13.3±1.7 years of education.

At the time they entered the study, we estimated the amount of ethanol consumed by all of the subjects in the preceding 10 years. The mean values were as follows: group 1, 695 kg, group 2, 675 kg, and group 3, 44 kg ($F=19.0$; $df=2, 66$; $p<.0001$). The number of years of very heavy drinking (defined as years in which ethanol consumption exceeded 560 g per week on the average) was 6.7 years for group 1, 6.5 years for group 2, and 0.2 years for group 3 ($F=35.4$; $df=2, 66$; $p<.0001$). The two alcoholic groups also used more marijuana (approximately three "joints" per week versus essen-

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TABLE 1
Mean Scores of Alcoholic Men and Controls on Neuropsychological Tests at Initial Testing and at 1-Year Follow-Up

Test	Recently Detoxified Alcoholics (N=23)		Abstinent Alcoholics (N=25)		Nonalcoholic Subjects (N=21)	
	Initial	Follow-Up	Initial	Follow-Up	Initial	Follow-Up
WAIS						
Verbal IQ	107.1	106.4	112.2	113.7	108.3	107.9
Performance IQ	107.3	110.2[a]	111.2	116.4[b]	107.6	113.1[c]
Full-scale IQ	107.4	108.5	112.4	115.7[c]	108.5	110.8
Information	12.0	11.9	12.7	12.9	12.1	12.2
Comprehension	10.8	10.3	11.9	11.4	11.8	11.4
Arithmetic	11.8	11.5	11.8	12.6[a]	11.5	11.1
Similarities	11.5	11.1	12.3	12.4	11.8	12.1
Digit Span	10.2	11.0	12.2	12.7	9.8	9.8
Vocabulary	11.5	11.0[c]	12.0	11.9	11.7	11.5
Digit Symbol	10.5	10.4	10.4	11.1[a]	10.6	11.2
Picture Completion	10.7	11.2	11.6	12.1[a]	10.8	11.5
Block Design	10.8	11.5	11.9	12.4	11.1	12.3[b]
Picture Arrangement	10.2	10.5	10.5	11.8	9.3	10.0
Object Assembly	10.3	11.0	11.1	11.9[a]	11.0	11.9
Halstead-Reitan battery						
Category Test[d]	36.3	33.1	34.9	22.8[b]	36.4	26.0[c]
Tactual Performance Test, dominant hand[d]	6.0	5.6	6.7	4.9[a]	5.6	5.0
Tactual Performance Test, nondominant hand[d]	4.5	4.5	4.2	3.5[a]	4.2	3.7
Tactual Performance Test, both hands[d]	2.3	2.7	2.4	2.5	2.5	2.2
Tactual Performance Test, total time[d]	12.6	13.0	13.0	10.7[c]	12.3	10.9[c]
Tactual Performance Test, memory	8.4	8.2	8.2	8.4	7.9	8.3
Tactual Performance Test, location	4.6	5.0	4.7	4.6	3.7	5.0[a]
Speech Errors[d]	6.1	5.7	4.3	4.0	5.3	4.6
Rhythm Errors[d]	3.7	4.5	3.2	2.2	4.3	4.5
Tapping, dominant hand	50.9	50.4	51.9	51.9	51.3	51.8
Tapping, nondominant hand	46.0	45.1	47.1	48.9[a]	47.4	48.2
Impairment Index[d]	.28	.28	.21	.16	.28	.23
Other tests						
Trails A[d]	26.6	28.2	22.7	28.8	25.8	22.7[a]
Trails B[d]	63.3	67.9	57.2	67.3	65.6	57.0
Pegboard time, dominant hand[d]	67.2	68.7	69.2	69.1	62.1	63.5
Pegboard time, nondominant hand[d]	74.0	74.3	71.8	69.5	69.1	69.2
Right-hand grip (kg)	51.9	53.1	57.4	57.1	60.6	59.2
Left-hand grip (kg)	45.8	45.2	52.3	53.9	54.1	54.3
Aphasia total errors[d]	1.74	2.13	1.52	1.12[a]	1.33	.76
Constructional Dyspraxia Rating[d]	1.00	1.30[a]	1.00	1.12	1.00	1.00
Memory tests[e]						
Immediate verbal	6.7	7.2	7.6	9.2[c]	6.7	7.8[c]
Verbal after 15-minute delay	5.9	5.6	5.9	7.0	5.2	6.5[c]
Rey diagram copy	34.0	32.4	32.4	33.0	32.2	32.7
Rey diagram after 15-minute delay	19.5	19.0	18.7	24.1[c]	21.0	23.7[c]

[a]The difference between initial and follow-up ratings was significant ($p<.05$).
[b]The difference between initial and follow-up ratings was significant ($p<.001$).
[c]The difference between initial and follow-up ratings was significant ($p<.01$).
[d]A higher score on this measure means more impaired performance.
[e]The group Ns for these tests are different because these tests were added after the study began. There were 8 men in the group of recently detoxified alcoholics, 15 in the group of abstinent alcoholics, and 19 in the nonalcoholic group.

tially none) and more cigarettes (10 versus 3 packs per week) than the nonalcoholic men. None of the subjects reported much experience with other drugs. Detailed comparisons did not reveal any significant differences between the follow-up groups and the original samples (demographic, medical, or substance use histories, nutrition, and initial neuropsychological test results).

The neuropsychological test protocols were grouped together, and one of us (I.G.), who was blind to group membership, rated the performance data on a 6-point scale ranging from excellent to severely impaired (1, 3).

RESULTS

At follow-up 9 men from group 1 (39%), 3 from group 2 (12%), and 3 from group 3 (14%) had mild to moderate neuropsychological deficits ($\chi^2=6.17$; $df=2$; $p<.05$). At the first examination 1 year before, 6 men in group 1 (26%), 5 men in group 2 (20%), and 4 men in group 3 (19%) showed such impairment (the difference was not significant). Initial and follow-up scores for selected neuropsychological tests are presented in table 1.

The alcoholic men who had been abstinent for at

least 18 months and the nonalcoholic subjects improved significantly on several tests and summary indexes including the Category Test, Tactual Performance Test total time, and some Wechsler Adult Intelligence Scale (WAIS) performance items. In contrast the recently detoxified group did not improve significantly on any test, although they did increase their overall performance IQ. This suggests that the recently detoxified group did not benefit as much from practice effect as did the other groups.

Sixty-three percent of the recently detoxified alcoholic men reported having had at least one drink in the follow-up periods, as did 20% of the long-term abstinent group and 48% of the nonalcoholic subjects. Most reports of drinking were extremely modest (a few drinks a month), although 1 man from group 1 reported drinking a quart of whiskey a day for a month 2 months before retesting. We related interim drinking (yes versus no) to follow-up neuropsychological results (unimpaired versus impaired). There was no reliable association. Similarly, neuropsychological status at the beginning of the study did not predict subsequent drinking.

There were no systematic between-group differences in nutritional status (ratings were made by a dietitian) during the follow-up period. Several group 1 subjects reported interim medical events: 1 man suffered a stroke (although he was rated as much more impaired neuropsychologically at follow-up, he was also mildly impaired before the stroke), another was given diphenylhydantoin because of a seizure, and a third suffered a concussion in an auto accident. The latter 2 subjects remained unimpaired at follow-up. No important medical events occurred in groups 2 or 3. Thus, the increased proportion of neuropsychological impairment in group 1 at follow-up cannot be explained on the basis of available interim drinking, drug use, or dietary or medical history.

COMMENT

Our initial results (1) suggested that there were no systematic neuropsychological differences between two groups of abstinent alcoholic men (those recently detoxified and those who had been abstinent for 18 months) and a nonpatient matched sample of men without a heavy drinking history.

Repeat neuropsychological assessment of these subjects at 1 year follow-up suggests that subtle changes may have occurred. Taken as a whole, our neuropsychological results show similarities in performance between longer-term abstinent alcoholic men (group 2) and the nonalcoholic control subjects (group 3). The recently detoxified group (group 1) seemed to perform differently from the other groups at follow-up.

The clinician's global ratings showed a trend toward improvement in the alcoholic men who had been abstinent for a longer time (group 2) and the nonalcoholic subjects (group 3); this trend was also reflected in improved scores on about one-fourth of the neuropsychological tests. This may reflect actual improvement in cerebral functioning, along with whatever small amount of variance is attributable to practice effects.

In contrast, the recently detoxified alcoholic men (group 1) not only failed to produce improved performance on retest, but in some cases they were actually more impaired according to the clinician's global rating.

These findings might plausibly be interpreted to mean that at initial examination the recently detoxified alcoholic men, although able to perform tests at an essentially normal level, nevertheless had difficulties in the kind of incidental learning that helped subjects in the other two groups do better when retested. Such a defect in incidental memory might reflect a persisting subclinical "intermediate duration organic mental disorder," evidence for which has been presented by Bennett and associates (4) in alcoholic subjects and by us (3, 5, 6) among polydrug users.

Some of our results are comparable to the results of Long and McLachlan (7), who studied a smaller sample of alcoholic men who had been abstinent for a year at follow-up. Their subjects were 8 years older, on the average, than our subjects and more intelligent as measured by the WAIS.

Direct comparison of the changes in Long and McLachlan's subjects and ours shows that five of the seven indicators that showed significant improvement in their study were also significantly improved in our abstinent alcoholic group. Among our nonpatient control subjects, four of the seven tests showed greater than chance improvement, but our recently detoxified alcoholic men had no test improvement comparable to Long and McLachlan's abstinent group.

Some procedural differences make direct interpretation of this comparison difficult. Long and McLachlan obtained differences between their older alcoholic subjects and volunteer control subjects at initial testing, but they did not retest their control subjects. Further, the period between detoxification and testing in their initial examination of the alcoholic subjects was about half that of our study (11 versus 21 days).

The fact that several of our recently detoxified subjects actually deteriorated over a year is puzzling. We found no relationship between medical history, nutrition, drug, or alcohol use in the interim period that would explain the neuropsychological differences among the three groups. There are no readily apparent subject selection factors (health and educational histories were comparable), and the retested samples did not differ from the three initial groups on any relevant dimension. It is, of course, possible that the recently detoxified alcoholic subjects drank more in the follow-up period that they admitted and that such interim in-

toxication contributed to a deficit. Although we cannot dismiss such an explanation, we believe its credibility is weakened by the fact that the alcoholic subjects who reported drinking very heavily since the initial assessment showed no neuropsychological deterioration at follow-up.

Deserving of further exploration is the possibility that some men in the recently detoxified group have entered a process of change in their neuropsychological abilities that is no longer correctable by abstinence.

We suggest that alcoholism-related organic mental disorder might have a latent phase similar to that described for many chronic medical illnesses such as diabetes. According to this model, people destined to develop neuropsychological impairments (either purely on the basis of alcoholism or because of some interaction among constitutional factors, drinking, and normal aging) must at some point in the evolution of their disorder experience early subtle changes in cerebral functioning that are not yet measurable even by extensive neuropsychological testing. A clue to this latent phase might be failure of incidental learning as reflected by lack of expected improvement in test performance at follow-up. With the passage of time, the process might advance to a subclinical phase. At this point a person would still appear "normal" in the usual clinical sense but show signs of neuropsychological deficit on comprehensive examination. Most neuropsychological studies of alcoholics reported in the literature thus far appear to have studied subjects with such a subclinical disorder (8–14). Eventually, some people would be expected to enter a clinical phase characterized by definite signs of cognitive decline, the final outcome of which is classical Wernicke-Korsakoff dementia (15).

Clearly, a report on a small number of subjects followed for a year can offer only tentative support for the model of the natural history of alcoholism-related cerebral disorder that we have proposed. The real test will come when we and others have examined larger and more diverse groups of alcoholic subjects over much longer time spans. Such investigations are time-consuming and costly. At the same time, we probably have reached a limit to the amount of new information that cross-sectional studies, no matter how elegant, can provide about the long-term effects of alcoholism on CNS function.

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Neuropsychological Deficits in Alcoholic Men in Their Mid Thirties

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The authors examined performances on 24 commonly used clinical neuropsychological tests for drug-free alcoholic men in their mid to late 30s either within 2–6 days of their last drink (N=59) or between 14 and 31 days after their last drink (N=20). Although mean scores were indicative of impairment on approximately half of the tests, no statistically significant overall difference in performance between individuals tested at these two time periods was observed. Recent and chronic alcohol consumption variables were found to interact with each other and with age and education in a nonlinear fashion in predicting neuropsychological performance. Increased consumption predicted decreased performance even on tests whose mean scores were in the normal range.

Although sober alcoholics do not possess overall intellectual impairment (1, 2), they have deficits in perceptual capacities, conceptual shifting, motor performance, memory function, and visual-spatial abstracting abilities (1, 3–6) and consequently have been compared to people with organic brain damage (7–11). These results become even more convincing when it is noted that many of the neuropsychological tests commonly used to assess alcoholism-associated brain dysfunction have been demonstrated to be valid (12, 13) and clinically reliable (14, 15). Reduced cognition in alcoholic patients has been related to length of alcoholism (9, 16, 17) and to pattern of alcoholic drinking even when mean test scores are in the normal range (6). Fine and Steer (18) reported brain damage coincident with early alcohol dependency, and Parker and Noble (19) demonstrated that the amount of alcohol consumption per drinking occasion was negatively related to performance by social drinkers on several cognitive tests. These findings are strikingly different from those of the study by Grant and associates (20), who opted for "cautious optimism that even very heavy alcohol use (80 g/day, or more) is not related to neuropsychological impairment in the alcoholic who is in his or her late 30s."

This conclusion by Grant and associates (20) has serious clinical implications, particularly in view of the enormity of alcoholism as a social problem. Specifically, if clinicians believe that no harm will result from drinking heavily to individuals who are less than 37 years of age, efforts to intervene in this large population might not be highly motivated. Thus, additional studies of this age group should be conducted so that the generality of the findings of Grant and associates (20) to other clinical populations can be assessed. We undertook this prospective study to determine whether recent and chronic alcohol consumption variables were related to neuropsychological performance in alcoholic men in their mid 30s.

METHOD

The subjects were 79 male volunteers who were between the ages of 21 and 46 years and were inpatients in the Alcohol Treatment Program of the Veterans Administration Hospital, Long Beach, Calif. These patients were an age-selected subsample of those alcoholic men between the ages of 21 and 60 years previously reported on by Eckardt and associates (6, 21). They were admitted with a primary medical diagnosis of alcoholism and had no other psychiatric disorder. Informed consent was obtained after the procedure had been fully explained. We used a table of random numbers to assign patients to one of two groups. Group 1 (N=59) was tested within 2–6 days of their last drink, and group 2 (N=20) was tested between 14 and 31 days after their last drink. Group designation was independent of the intensity of withdrawal. Group 1 contained more patients than group 2 because the

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latter was originally viewed as a control group for repeated-testing effects (21). The patients in group 1 had a mean age of 36.1±7.2 years, and those in group 2 were aged 34.4±7.9 years.

Neuropsychological testing was conducted by study personnel specifically trained and familiar with the tests used. Testing was conducted after the clinical symptoms associated with acute withdrawal (extreme tremulousness, ataxia, and diaphoresis) subsided. Patients were tested individually after it was determined that no psychoactive medication had been administered during the previous 48 hours. The battery of 24 neuropsychological tests was given over a 2-day period; the order of tests was determined randomly. Tests were selected on the basis of common clinical use and recent literature reviews on brain dysfunction in alcoholics and have been described elsewhere (6, 21).

Detailed information on recent and chronic alcohol consumption, drinking-related behavior, physical and mental health conditions that might influence neuropsychological performance, use of drugs, and socioeconomic characteristics was obtained from research questionnaires filled out by the patient approximately midway through the 21-day treatment program. Occasional discrepancies between this information and medical charts were resolved through an interview by study personnel, who were not members of the treatment staff.

RESULTS

Comparison of Group 1 and Group 2

There were no significant differences between the two alcoholic groups in age, education, marital status, race, personal monthly income, current employment status, recent and chronic alcohol consumption, or drinking-related behavior. One of the subjects in group 1 (2%) and none of the subjects in group 2 had completed grade school only, 28 in group 1 (47%) and 7 in group 2 (35%) had completed high school, and 30 in group 1 (51%) and 13 in group 2 (65%) had completed college. Thirty of the subjects in group 1 (51%) and 10 in group 2 (50%) were married, 17 in group 1 (29%) and 6 in group 2 (30%) were widowed or divorced, and 12 in group 1 (20%) and 4 in group 2 (20%) were single. Twenty-four subjects in group 1 (41%) and 12 in group 2 (60%) had an income of $100–$499, 16 in group 1 (27%) and 6 in group 2 (30%) had an income of $500–$899, and 19 in group 1 (32%) and 2 in group 2 (10%) had an income of $900 or more. The mean number of years of abusive drinking in group 1 was 12.0±7.6, and the mean for group 2 was 13.4±8.2. The mean lifetime consumption of 100% ethanol was 483±450 gallons in group 1 and 548±501 gallons in group 2. The mean number of grams of ethanol per kilogram of body weight per drinking occasion during the past 6 months was 2.4±0.7 in group 1 and 2.8±0.9 in group 2. The mean number of drinking occasions during the past 6 months was 337±200 in group 1 and 352±225 in group 2. Thirteen subjects in group 1 (22%) and 7 in group 2 (35%) had a history of delirium tremens, 7 in group 1 (12%) and none in group 2 had a history of seizures, 55 in group 1 (93%) and 16 in group 2 (80%) had a history of blackouts, and 25 in group 1 (42%) and 7 in group 2 (35%) had received treatment for alcoholism before.

TABLE 1
Neuropsychological Test Scores for Alcoholic Men Tested Within 2–6 Days of Their Last Drink (Group 1) and Those Tested Between 14 and 31 Days After Their Last Drink (Group 2)

	Group 1 (N=59)		Group 2 (N=20)	
Test	Mean	SD	Mean	SD
Tactual Performance Test, dominant hand (minutes)	7.2	3.0	6.4	3.1
Tactual Performance Test, nondominant hand (minutes)	6.1	3.3[a]	6.4	3.6[a]
Tactual Performance Test, both hands (minutes)	3.7	2.4[a]	3.7	3.0[a]
Tactual Performance Test, total time (minutes)	17.1	7.7[a]	16.6	8.7[a]
Tactual Performance Test, memory	7.4	1.3	7.2	1.4
Tactual Performance Test, location	3.3	2.5[a]	2.8	2.0[a]
Tapping, dominant hand	45.5	7.2[a]	45.4	6.8[a]
Tapping, nondominant hand	41.7	7.0[a]	43.5	5.5[a]
Trails A (seconds)	37.9	14.6[a]	34.4	9.2[a]
Trails B (seconds)	89.1	36.1[a]	96.9	41.0[a]
Categories	−66.9	27.0[a]	−67.0	29.2[a]
Speech Perception	50.7	7.2[a]	52.7	5.3
Seashore Rhythm	24.2	2.9[a]	25.4	3.6
Wisconsin Card Sorting Test	4.7	1.9[a]	3.9	2.5[a]
Digit Symbol	42.9	9.9	46.2	10.3
Digit Span	11.1	2.1	11.2	1.8
Block Design	30.6	7.9	34.2	7.6
Object Assembly	29.2	6.7	29.8	6.6
Rod and Frame, left tilt (degrees)	− 1.70	1.35	− 1.86	1.05
Rod and Frame, right tilt (degrees)	− 1.12	0.78	− 0.96	0.57
Benton Visual Retention Test	5.5	1.9[a]	5.4	1.9[a]
Shipley-Hartford, verbal	28.6	5.0	28.2	6.3
Shipley-Hartford, abstract	10.5	4.4	10.8	3.9
Shipley-Hartford, conceptual quotient	83.3	13.4	86.1	18.2

[a]Performance at a level associated with brain dysfunction according to descriptive references in Eckardt and associates (6, 21).

The subjects' performance on the 24 neuropsychological tests are summarized in table 1. It is apparent that mean scores were at levels indicative of brain dysfunction on 13 of 24 tests for group 1, tested within 2 and 6 days of their last drink, and 11 of 24 tests for group 2, tested between 14 and 31 days after their last drink (see table 1). These results are similar to those reported by other investigators (1, 3, 5) and differ markedly from those of Grant and associates (20).

Multivariate analysis of variance procedures demonstrated that performance over all 24 tests did not differ as a result of past or present medical ailment. These ailments were dizziness, severe head injury, headaches, anxiety, depression, nervousness, excessive sleepiness, unconsciousness, difficulty in concen-

tration, memory loss, abnormal blood pressure, blackouts, delirium tremens, seizures, shakes, or difficulty in sleeping. In addition, neither the use of medication nor illicit drug use over the 6 months preceding treatment was related to neuropsychological performance. Of the patients who used illicit drugs, 2 (2.5%) used glue, 4 (5.1%) used heroin, 6 (7.6%) used LSD, 6 (7.6%) used cocaine, and 26 (32.9%) used marijuana; the average frequency of use of these compounds among users was never more than 1–2 times per month.

Multivariate analysis of covariance, with age and education as covariates, revealed no significant differences between the two groups compared over all 24 tests; i.e., there was no demonstrable improvement in neuropsychological performance between means (±SD) after 5.0±1.3 and 20.8±4.0 days of abstinence. Similar findings in patients between 21 and 60 years of age have already been reported (21). Even though the two groups did not differ significantly in cognitive performance, they were analyzed separately to include a group that approximated the time of testing used by Grant and associates (20).

Analyses for Group 1 (N=59)

We have reported that polynomial regression models involving recent and chronic drinking variables, age, and education significantly predicted neuropsychological performance in alcoholic men (6). Therefore, we conducted a similar analysis using age, education, and four alcohol-consumption variables—current dose (grams of ethanol per kilogram of body weight per drinking occasion), how often the maximum number of drinks was consumed, lifetime gallons of 100% ethanol, and years of abusive drinking at current or greater levels. The analysis consisted of first expanding these variables into a full polynomial equation of degree two. This quadratic equation had the usual linear terms[1] employed by Grant and associates (20) plus the square of each linear term and all combinations of the linear terms in order to represent more complex relationships. This quadratic equation was then inserted into the BMDP9R all-possible subsets regression program, which finds the "best" regression equation for prediction. "Best" is defined in terms of minimizing the standardized total mean squared error of estimation (22). All combinations of independent terms, linear and nonlinear,[2] were considered in the selection of the best equation. We used actual test scores of the 59 alcoholic men tested within 7 days of their last drink as dependent variables rather than using overall clinical ratings as Grant and associates (20) did. We did this to satisfy mathematical concepts inherent to the statistical model and the customary hypothesis testing that is performed.

Of the 24 tests, 21 were significantly predicted by this procedure ($p \leq .05$) with an average R^2 (i.e., the amount of variability or variance explained by the independent variables) of .38, including 8 tests for which the mean score was in the normal range. The only tests not significantly explained were Tactual Performance Test-Memory, Block Design, and left-tilt Rod and Frame. Determination of the amount by which R^2 was reduced if drinking variables were removed from the equations revealed that alcohol consumption was involved in 70% of the explained variance, age contributed 20%, and education contributed 10%. Approximately half of the variance related to alcohol consumption was associated with chronic variables and half with recent variables. The resulting equations were similar to those reported by Eckardt and associates (6), which consisted of linear and nonlinear combinations of variables; no equation contained only linear terms.

We conducted additional regression analyses in which we incorporated the quadratic expansion of age and one of the recent or chronic consumption variables at a time to determine the neuropsychological consequences of different drinking patterns. In the 15 tests for which the models predicted significant amounts of variance, increased consumption was predictive of decreased performance for all tests except Tapping, dominant and nondominant hands ($p \leq .05$). The trend of decreased performance with increased consumption was apparent even on the tests for which the mean score was in the normal range: Tactual Performance test, dominant hand; Digit Symbol; Digit Span; Object Assembly; and the Shipley-Hartford, verbal, abstracting, and conceptual quotient.

Analyses for Group 2 (N=20)

Because there were only 20 alcoholic men tested between 14 and 31 days after their last drink, similar polynomial regression analyses that included the quadratic expansion of age, education, and the four consumption variables could not be accomplished; therefore, only analyses for age and one of the recent or chronic consumption variables at a time were conducted. We found that 13 tests had a recent or chronic drinking variable in the resulting best equation; consumption and age each contributed approximately 50% of the explained variance. In the 11 tests for which a conclusion was possible, increased consumption predicted decreased neuropsychological performance on all tests except right-tilt Rod and Frame and Shipley-Hartford, verbal. The finding of decreased performance with increased consumption held even for tests with mean scores in the normal range, such as Tactual Performance Test, dominant hand; Digit Symbol; and Shipley-Hartford, conceptual quotient. No test score was predicted solely by linear terms.

[1]Of or involving terms of the first degree, such as X_1 and X_2.
[2]Of or involving terms of more than the first degree, such as X_1^2, X_2^2, and $X_1 \times X_2$.

DISCUSSION

Because the reduced level of neuropsychological performance that we observed in this study is similar to levels previously reported (1, 3, 5), our sample is not atypical. We demonstrated that recent and chronic alcohol consumption variables interact with age and education in a nonlinear fashion and that alcohol consumption is involved in explaining significant amounts of variance for commonly used clinical neuropsychological tests. It is clinically not surprising to find that these higher order statistical models were more sensitive than the standard linear models used by Grant and associates (20), when one considers the complexity of the relationships between multiple cognitive functions and recent and chronic drinking history.

Grant and associates (20) reported normal neuropsychological test scores for a group of alcoholic men; we also observed that mean scores for our samples on approximately half of the tests were in the normal range. However, the fact that we demonstrated significant nonlinear relationships between recent and chronic alcohol consumption and normal test scores necessitates discussion of what is implied by the concept of normalcy. It is commonly concluded that when no impairment is found on clinical neuropsychological tests, there is no brain dysfunction. This line of reasoning assumes our clinical assessment instruments are infinitely sensitive and ignores the findings that the brain appears to have a neurological reserve or plasticity (23), which, particularly in younger individuals (24), may allow the brain to reorganize rapidly to a "normal level" of cognitive functioning as measured by clinical neuropsychological tests. Thus, it is difficult to determine the impact of ethanol on brain function at any given point in time because we rarely know what the level of functioning for a particular individual was before heavy and/or chronic alcohol consumption. Accordingly, it is possible to obtain "normal" results for any given person that are actually abnormal in terms of the original (i.e., before heavy alcohol intake) capacity. This concept of "abnormal normals" among alcoholics gains support from our investigation and from the study by Eckardt and associates (6), both of which showed that cognitive performance among alcoholic patients was significantly predicted by nonlinear combinations of recent and chronic consumption variables, even on tests whose mean scores were in the normal range. Additional support derives from the finding of significant negative correlations between normal test scores and current dose of alcohol consumed per drinking occasion by social drinkers (19). In our study, as well as in studies by Eckardt and associates (6) and Parker and Noble (19), increased alcohol consumption was usually associated with decreased cognitive performance.

The observed degree and speed of recovery in pathological states that return to "normal" is obviously related to the time of observation after cessation of the pathogenic insult. If the intensity of insult is small and/or the duration of exposure is short, the period of abnormality may be minimal. Grant and associates (20) gave their patients a clean bill of health on the basis of neuropsychological test scores obtained after 3 weeks of abstinence. Although it is possible that their patients may have been malfunctioning cognitively for the 3 weeks before testing, a recent investigation (21) found no improvement in the cognitive performance of patients tested after 1 week of sobriety and those tested after 2–3 weeks of sobriety other than that resulting from having taken the same set of tests twice; we have confirmed this finding in the present investigation. Nevertheless, the period of cognitive "malfunction" directly related to alcohol abuse, even if it is less than 1 week, is clinically very important. An individual who goes into cardiac failure for even 1 hour because of a known drug reaction is not given a clean bill of health when he or she recovers but is told to avoid the drug and is then monitored clinically. Moreover, if repeated insults that have a neurological impact occur, they might be likely to have a cumulative effect over time. Clinical laboratory tests such as liver function tests become "abnormal" early in excessive chronic alcohol drinking (25); therefore, if such tests were routinely available for brain function, similar early adverse consequences of heavy alcohol consumption could be found. A crucial difference in this analogy is that brain tissue does not have the regenerative capacity of the liver either before or after a person reaches the mid 30s.

Intensity, chronicity, and receptivity to any pathological process are generally acknowledged to be important determinants of outcome. Although both our samples and the one reported on by Grant and associates (20) were from Veterans Administration hospitals and of similar ages (i.e., receptivity), our patients drank significantly more alcohol (i.e., intensity) and had more years of abusive drinking (i.e., chronicity). In fact, the patients in Grant and associates' study (20) ingested relatively little alcohol for a short period of time. Our patients had a significant incidence of delirium tremens, seizures, blackouts, and previous treatment for alcoholism; on the other hand, Grant and associates did not mention these characteristics in their sample. We conclude that the patients in Grant and associates' study (20) had not been drinking sufficient amounts (i.e., intensity and chronicity) to allow the researchers to observe significant dose-effect relationships for neuropsychological testing with the age (i.e., receptivity) of their sample and the statistical procedures used. In contrast, our investigation demonstrated significant nonlinear relationships between recent and chronic alcohol consumption and neuropsychological performance which indicated that increased consumption was associated with decreased cognition.

Accordingly, instead of "grounds for cautious optimism that even very heavy alcohol use is not related to neuropsychological impairment in men in their late 30s" (20), we conclude that alcohol abuse in this age group is associated with clinically detectable cognitive impairment.

ADDENDUM

In this issue of the *Journal,* Adams and associates (26) conclude that their failure to show improved neuropsychological performance in alcoholic men in their late 30s when tested 3 weeks after their last drink and again 1 year later indicates "a preclinical phase of what would ultimately be seen as a more clear organic impairment." In that 63% of their alcoholic patients reported posttreatment alcohol consumption, a recent report by Eckardt and associates (27) would support another interpretation, namely, that verified posttreatment alcohol consumption has adverse neuropsychological consequences and might serve to maintain cognitive performance at original levels.

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DSM-III: Psychiatric Diagnosis in a University Population

BY RONNIE S. STANGLER, M.D., AND ADOLPH M. PRINTZ, PH.D.

Using DSM-III *the authors reviewed psychiatric diagnoses in a sample of 500 students at a university psychiatric clinic. They present prevalence data and associated age and sex characteristics of this population in relationship to their diagnostic findings and discuss these in the context of earlier epidemiologic studies and their impact on university mental health services.*

The third edition of the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* holds promise for increasingly discrete, uniform, and reliable identification of clinical entities. With this new diagnostic language, clinicians have the capacity to more accurately assess the prevalence of psychiatric disorder; such data are essential to treatment planning, program development, and psychotherapeutic and psychopharmacologic outcome research.

Although epidemiological studies have previously focused on university populations (1–9), the emergence of *DSM-III* warrants fresh examination of this patient group. We present data that identify diagnostic prevalence and associated age and sex characteristics for a sample of students who used a psychiatric clinic. Our findings offer additional empirical information about new diagnostic categories first presented in *DSM-III;* we highlight those data which are consistent with or contradict the suggested descriptors of both old and new categories presented in the field trial manuals.

METHOD

The University of Washington Psychiatric Clinic for Students is an outpatient service housed in a comprehensive outpatient medical facility, Hall Health Center, serving a university population of 37,000 students. All undergraduate, graduate, and professional school students are eligible for the clinic's services. We provide crisis intervention and time-limited psychotherapy to approximately 1,200 students per year. The clinic staff is composed of psychiatrists, a clinical psychologist, psychiatric social workers, and trainees in all these disciplines in conjunction with the training program of the university's Department of Psychiatry and Behavioral Sciences.

During the study, our staff recorded age, sex, ethnic status, and the multiaxial *DSM-III* diagnosis (Axis I, clinical syndromes, Axis II, personality and specific developmental disorders, Axis III, physical disorders, Axis IV, severity of psychosocial stressors, and Axis V, highest level of adaptive functioning) for all patients. We initiated data collection on 500 consecutive patients during our participation in an NIMH-sponsored field trial of *DSM-III*. Although our diagnostic protocol overlapped with a number of *DSM-III* draft revisions, all diagnoses reported in this study have been corrected to agree with the final *DSM-III* terminology.

RESULTS

Of the 500 students evaluated, 63.6% were women and 36.4% were men. The mean age of the students was 25.4 years (SD=5.71, range=17–53); the modal age was 21 years. The ethnic-racial composition of the group (according to a classification recommended by NIMH) was 88.2% white, 5.6% Asian, 3.6% black, 2.4% Hispanic, and 0.2% American Indian.

The diagnostic sample yielded a total of 726 entries on Axes I and II: 548 on Axis I and 178 on Axis II. The majority of patients (63.4%) received only one diagnosis (regardless of axis), 29.2% received two, 6.4% three, and the remainder received four or more.

On Axis I, 75.4% of the sample received only one diagnosis of a clinical syndrome, 8.8% received none, and the balance had three or more such diagnoses. On Axis II, 65.8% of the sample received no diagnosis of a personality or developmental disorder, 32.8% received one diagnosis, and 1.4% received two diagnoses. Axis III was not tabulated for this study and will not be discussed further.

After conclusion of formal participation in the

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TABLE 1
Most Frequent Axis I Diagnostic Classes Used for 500 University Students, Including the Most Prevalent Single Diagnoses in Each[a]

Diagnostic Class	Number of Diagnoses	Percent of Total Patient Sample (N=500)	Two Most Prevalent Single Diagnoses: Diagnoses	Number of Patients	Percent of Diagnostic Class
Adjustment disorders	145	29.0	Adjustment disorder with depressed mood	60	41.4
			Adjustment disorder with mixed emotional features	53	36.6
Other specific affective disorders	85	17.0	Dysthymic disorder	79	92.9
			Cyclothymic disorder	6	7.1
Conditions not attributable to a mental disorder (V codes)	73	14.6	Marital problem	27	37.0
			Other interpersonal problem	19	26.0
Anxiety disorders	46	9.2	Generalized anxiety disorder	11	23.9
			Panic disorder	10	21.7
Major affective disorders	42	8.4	Major depression[b]	33	78.6
			Bipolar disorder, depressed[b]	4	9.5
Psychological factors affecting physical condition	36	7.2	Psychological factors affecting physical condition[c]	36	100.0
Eating disorders	22	4.4	Bulimia	19	86.4
			Anorexia nervosa	3	13.6
Other disorders of infancy, childhood, or adolescence	19	3.8	Identity disorder	19	100.0
Psychosexual dysfunction	12	2.4	Inhibited sexual desire	4	33.3
			Inhibited sexual excitement	4	33.3

[a]Diagnostic classes were included only if there were 10 or more subjects in that class; since many patients received more than one Axis I diagnosis, the final tally of Axis I diagnoses exceeds the actual number of patients.
[b]The levels of severity for these diagnoses were collapsed for a uniform data count.
[c]*DSM-III* classification provides only one diagnosis in this diagnostic class.

TABLE 2
Sex and Age Distribution in Most Frequent Axis I Diagnoses for 500 University Students[a]

Diagnosis	Number of Diagnoses	Percent of Total Patient Sample (N=500)	Women: N	Women: Percent of Diagnostic Class	Men: N	Men: Percent of Diagnostic Class	Age (years): Mean	Age (years): SD
Dysthymic disorder	79	15.8	59	74.7[b]	20	25.3	25.6	6.4
Adjustment disorder with depressed mood	60	12.0	35	58.3	25	41.7	25.1	7.0
Adjustment disorder with mixed emotional features	53	10.6	37	69.8	16	30.2	26.2	7.4
Psychological factors affecting physical condition	36	7.2	23	63.9	13	36.1	25.0	4.1
Major depression[c]	33	6.6	19	57.6	14	42.4	24.5	5.0
Marital problem	27	5.4	20	74.1	7	25.9	26.9	3.2
Other interpersonal problem	19	3.8	13	68.4	6	31.6	26.5	5.0
Bulimia	19	3.8	17	89.5[b]	2	10.5	21.4[d]	2.6
Identity disorder	19	3.8	17	89.5[b]	2	10.5	23.4	4.8
Adjustment disorder with anxious mood	18	3.6	11	61.1	7	38.9	27.2	6.3
Phase of life problem or other life circumstance problem	14	2.8	10	71.4	4	28.6	23.4	5.7
Adjustment disorder with work (or academic) inhibition	11	2.2	6	54.5	5	45.5	26.7	7.3
Generalized anxiety disorder	11	2.2	8	72.7	3	27.3	26.0	4.1
Panic disorder	10	2.0	8	80.0	2	20.0	24.7	3.9

[a]Diagnoses were included only if there were 10 or more subjects in that subsample.
[b]The difference between men and women was significant ($p<.05$, corrected chi-square).
[c]The level of severity for this diagnosis was collapsed for a uniform data count.
[d]The difference in mean age between the subsample and the balance of the total sample was significant ($p<.05$, t test).

NIMH field trial, several staff members independently began to omit Axes IV and V from their multiaxial diagnoses. These omissions were predominantly in response to a subjective sense of minimal variability in the ratings on these axes. As a result, only 397 of our 500 patients received ratings on Axes IV and V. Data on these axes for the patients so diagnosed revealed that ratings for both scales clustered about the scale

TABLE 3
Sex and Age Characteristics of Most Frequent Axis II Diagnoses for 500 University Students[a]

Diagnosis: Personality Disorder	Number of Diagnoses	Percent of Total Patient Sample (N=500)	Women		Men		Age (years)	
			N	%	N	%	Mean	SD
Borderline	29	5.8	21	72.4	8	27.6	26.5	5.9
Atypical, mixed, or other	28	5.6	19	67.9	9	32.1	24.5	6.1
Compulsive	24	4.8	9	37.5	15	62.5[b]	25.4	5.7
Dependent	20	4.0	12	60.0	8	40.0	26.5	5.8
Histrionic	18	3.6	18	100.0[b]	0	0.0	25.0	6.6
Avoidant	18	3.6	9	50.0	9	50.0	24.5	5.2
Schizotypal	12	2.4	5	41.7	7	58.3	26.1	4.0

[a]Diagnoses were included only if there were 10 or more subjects in that subsample.
[b]The difference between men and women was significant ($p<.05$, corrected chi-square).

midpoints. On Axis IV (severity of psychosocial stressors), 61.2% of this subsample was rated as 3 (mild) or 4 (moderate). On Axis V (highest level of adaptive functioning), 62.5% were rated as 3 (good) or 4 (fair). We do not further detail Axes IV and V in our statistical reporting. Assessment of the clinical utility of these scales necessitates correlation with external criteria not included in this diagnostic study (e.g., outcome).

Axis I diagnostic classes with at least 10 diagnoses each are rank-ordered in table 1. Within each of these classes, the two diagnoses with the highest frequency and what percentage they represent of their respective classes are also presented in the table. For all classes except anxiety disorders, the two most frequent diagnoses account for at least 60% of each class's diagnostic total.

Diagnostic classes with fewer than 10 diagnoses each are represented as follows: substance use disorders, 9 diagnoses; schizophrenic disorders, 8; somatoform disorders, 7; other psychosexual disorders, 7; atypical affective disorders, 5; dissociative disorders, 5; psychotic disorders not elsewhere classified, 3; paranoid disorders, 3; gender identity disorders, 2; disorders of impulse control, 1; paraphilias, 1; substance-induced organic mental disorder, 1; other disorders with physical manifestations, 1; and stereotyped movement disorders, 1.

The most prevalent Axis I diagnoses (those with at least 10 cases) are shown in table 2 with their respective sex and age distributions. We tested male-female differences using a corrected chi-square ($p=.05$). The mean age for any given diagnosis was tested against the mean age for the balance of the total sample with t tests ($p=.05$).

A significant age difference was found for only one diagnosis, bulimia: the patients with this diagnosis were younger ($p=.002$). Significant sex differences were found for bulimia ($p=.032$), identity disorder ($p=.032$), and dysthymic disorder ($p=.035$); there were more women than men with all of these diagnoses.

Table 3 extends the same tabulation and statistical procedures to Axis II diagnoses, which, for our sample, included only personality disorders. We found no significant age differences here, but two clear sex differences emerged: histrionic personality disorder was exclusively diagnosed in women ($p=.002$), and compulsive personality disorder was significantly more common in men ($p=.02$).

Personality disorders with fewer than 10 diagnoses per category included passive-aggressive, with 8 diagnoses; narcissistic, with 8; paranoid, with 7; antisocial, with 4; and schizoid, with 1.

DISCUSSION

It is clear from these diagnostic findings that depressive symptoms, irrespective of severity, presence of clear precipitants, pattern of recurrence, or chronicity, account for the majority of this population's diagnostic variance. This global statement is essentially consistent with the findings of earlier university mental health studies (9, 10), but we used *DSM-III*'s greater differentiation of depressive syndromes to provide a more qualitatively and quantitatively accurate picture.

The high frequency of adjustment disorder with depressed mood is to be expected in this population as sequelae to the many stressors of the university experience: relationship conflicts and loss, academic demands and failure, and separation from home and family. Since there is little basis in the literature for predicting the prevalence of major depressive disorder in the student population (7), we can only report our findings and wait for further data from other colleges and universities.

We found the prominence of dysthymic disorder quite surprising. Given that this diagnosis requires a minimum of two years of depressive symptoms, its high frequency in our age-restricted sample supports a late adolescent-early adult onset model. The singularly high prevalence of this chronic form of depression challenges the resources of university mental health facilities, many of which are mandated to provide only short-term psychotherapeutic interventions.

Consistent with the finding of the *DSM-III* draft, dysthymic disorder was significantly more common among women than men. However, this sex difference

did not hold for any other category of depression. Although the draft also predicted a greater prevalence of major depression among women, our data revealed no such difference for this diagnosis or for the adjustment disorders.

Our finding that women constituted the majority of our clinic sample (64%) is in keeping with previous reports (3, 6, 8), but no university mental health research has addressed the issue of sex differences for clearly delineated individual diagnoses. Two new diagnoses, identity disorder and bulimia, were significantly more common among women. For the personality disorders, only histrionic and compulsive disorders revealed clear sex differences: more women had the former diagnosis and more men had the latter. Although *DSM-III* predicted that women would be more likely to fit the categories of borderline, dependent, and avoidant personality disorders, we noted no significant sex differences in these diagnoses. These initial sex prevalence data will need to be further explored in future studies, particularly in the light of changing sex roles and their consequent emotional correlates.

The emergence of bulimia as a diagnostic entity and its striking frequency in our sample serves as one example of the complicated sociocultural issues surrounding femininity. A 1978 study of one variant of this diagnosis, bulimarexia (11), suggested that issues of "female role definition" are central to this disorder's genesis in adolescent women. We believe that our prevalence finding for bulimia represents a conservative accounting of this syndrome in the university population. In addition to patients given this diagnosis, we noted that other cases of bulimia were revealed during therapy; our data entry system had no provision for the inclusion of new diagnoses after intake. It is interesting to note that bulimia was the only diagnosis in our study with a significant age difference. Although patients with bulimia were younger women, their mean age of 21.4 years falls within the usual undergraduate age spectrum.

Conspicuous by their absence in our results, the schizophrenic disorders accounted for only 1.5% of our diagnoses. Although previous studies have attempted to survey the prevalence of psychotic illness in university populations (4, 6), only rarely has attention been paid to factoring out the affective and paranoid psychoses from schizophrenia. Our low frequency of schizophrenic disorders requires further investigation, but the altered diagnostic criteria of *DSM-III* may partially explain this finding. The newly defined diagnoses of borderline and schizotypal disorders may now include patients previously diagnosed as having variants of schizophrenia (e.g., latent, simple, residual) in the *DSM-II* nomenclature.

The high frequency of psychophysiologic disorders and conditions not attributable to mental disorder (V codes) in our population is striking. As Reifler and associates (3) suggested, we may anticipate encountering a wider spectrum of problems in living in university psychiatric clinics as services become increasingly available. Apart from the issue of treatment availability, additional factors that contribute to the appearance of these diagnoses include increased public and professional acceptance of intervention in such areas, the emergence of short-term, more visible, and clearly defined treatment approaches (e.g., biofeedback training, couples therapy), and an increased focus on preventive strategies in psychiatry.

As university populations become increasingly heterogeneous with respect to age, sex, ethnicity, and socioeconomic status, the range of psychiatric diagnoses available for study extends toward an approximation of the general population and offers a more viable base for clinical inquiry. The cutting edge of *DSM-III* will expose our "unweeded garden" and challenges us to be more precise in our diagnostic formulations, more focused in our treatment planning, and more accountable to our supporting institutions.

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Age, Dementia, Dyskinesias, and Lithium Response

BY JONATHAN M. HIMMELHOCH, M.D., JOHN F. NEIL, M.D., SUSAN J. MAY, R.N., DR.P.H., CARILYN Z. FUCHS, PH.D., AND SANDRA M. LICATA, R.N.

Nineteenth-century neuropsychiatrists felt that the aged bipolar patient usually developed chronic mania, which eventually turned into dementia. The authors' elderly patients seemed to experience few such denouements, so they evaluated the course and treatment response of 81 bipolar patients over the age of 55. Fifty-six responded well to lithium. Advanced age had no effect on course or outcome. However, with increased clinical evidence of neurological illness there was an increased incidence of chronic mania, a poorer response to lithium, and more frequent and severe neurotoxicity. Extrapyramidal syndromes were particularly devastating.

King David and King Solomon led merry, merry lives
With many, many concubines and many, many wives.
But when old age came over them with its many, many qualms,
King Solomon wrote the proverbs and King David wrote the psalms.

—*Shul* ditty

The effects of aging on the presentation, course, and outcome of manic-depressive illness have never been thoroughly described. In the 19th century both Kraepelin (1) and Schüle (2) observed that in older patients a chronic manic state could develop out of acute manic excitement. This chronic mania contained not only confusional elements but a certain "psychobiological rigidity" (3) that foreshadowed the almost inevitable development of full-blown dementia. Yet even within the extremely ill populations cared for by late 19th-century alienists, this sequence of events—acute mania, followed by chronic mania, ending in dementia—was not the lot of every elderly manic-depressive patient. Mendel (4), for example, pointed out that most manic psychoses did not end this way and felt that the chronic manic states described by Kraepelin and Schüle were simply dementia with excitement, totally unrelated to true mania.

The past two decades have afforded a better opportunity to increase our rather incomplete understanding of the effects of aging on manic-depressive illness. Increased longevity has produced more elderly patients. The availability of lithium salts has not only provided effective treatment but has led to the creation of lithium clinics, where large numbers of elderly subjects with both early and late onset manic-depressive illness are treated. Moreover, not all of these patients are extremely ill, like those of Kraepelin's time, so that the effects of aging can be studied within a spectrum of severity. It is from such a sample that the effects of aging on the course of illness and the treatment response can best be studied and the true incidences of both chronic mania and neurological deterioration be gleaned.

This paper describes a detailed investigation of 81 patients between the ages of 55 and 88 who were treated with lithium salts at the Affective Disorders Clinic of the University of Pittsburgh.

METHOD

We studied the charts of 81 patients, who represented 81 consecutive admissions (from 1973 through 1976) of patients over age 55 with manic-depressive illness. Diagnoses were made using the Kupfer-Detre system (KDS) (5). All 81 patients met Feighner's criteria (6) for primary bipolar affective disorder. The Schedule for Affective Disorders and Schizophrenia-Research Diagnostic Criteria (SADS-RDC) (7) were not used at the clinic until late 1976, but it is probable that all 81 patients would also fulfill these criteria. Seven of the 81 patients were disabled only during their depressive phase and therefore merit the diagnosis of bipolar II (8). The remaining 74 were diagnosed bipolar I.

Data were collected on the following variables: sex, present age, age of onset of manic-depressive illness, drug or alcohol abuse, dementiform features, extrapyramidal syndromes, neurotoxic reaction to lithium, development of chronic mania, and significant medical

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illnesses. Lithium response was rated on a six-point scale of clinical efficacy. Ratings were defined as follows:

1. Optimum—remission of symptoms within three weeks of the start of lithium therapy without subsequent relapse.
2. Good—remission of symptoms within three to eight weeks of the start of lithium therapy without relapse.
3. Adequate—remission of symptoms during lithium therapy but subsequent relapse usually associated with poor compliance; manageable on an outpatient basis.
4. Poor—incomplete remission of symptoms during lithium therapy and/or at least one relapse during lithium therapy and requiring hospitalization.
5. Minimal—little symptomatic response to lithium therapy.
6. None—no response or worsening during lithium treatment, frequently the result of neurotoxicity at very low lithium doses and low plasma lithium concentrations.

Habitual drug and alcohol use was defined as in previous investigations (9). A patient was considered to be a habitual drug user if he or she used four or more standard doses of sedatives, minor tranquilizers, or psychomotor stimulants per day, three months or longer, and had had difficulty stopping these medications because of withdrawal symptoms. Alcohol abuse was defined according to criteria in *DSM-II*, i.e., "alcohol intake great enough to damage physical health or personal or social functioning; or to be the prerequisite of normal functioning."

Dementiform features were divided into two types: 1) the presence of confusion and/or disorientation only during an affective episode (episodic confusion), and 2) the presence of mild residual dementiform symptoms on mental status examination or on neuropsychological testing between, as well as during, affective episodes (dementia always present). Dementiform symptoms included poor memory, dysphasia, and/or concrete thinking or perseveration, but no frank disorientation to time, place, or person.

Extrapyramidal dysfunction in this sample included only parkinsonism and/or facial dyskinesia. Parkinsonism was defined as the presence of parkinsonian symptoms on the basis of idiopathic Parkinson's disease or on the basis of atherosclerosis, but not on the basis of ingestion of neuroleptic drugs. Facial dyskinesias were probably all neuroleptic related but had to persist after discontinuation of major tranquilizers in order to be recorded as present.

RESULTS

The most important overall result of this study was that 56 of the 81 elderly bipolar patients responded well (ratings of 1–3) to lithium carbonate. However, it is notable that 23 of the 25 poor responders (ratings of 4–6) had manifest neurological illness. Nineteen patients developed chronic mania. All were poor responders; 17 had hard neurological illness (12 had extrapyramidal syndromes, and 5 chronic dementia).

The relationship of lithium response to other variables is shown in tables 1 and 2. Table 1 shows no significant differences between good and poor responders related to present age, age of onset, duration of manic-depressive illness, or presence of medical illness. Abuse of drugs appeared to significantly interfere with lithium response. There was a significantly greater incidence of poor treatment response among men. However, this finding disappeared in later analyses that controlled for neurological status and drug abuse (see below and table 3), probably because of the known higher incidence of both alcohol abuse and cerebral vascular disease among men.

Table 2 shows the devastating effect of worsening neurological status on response to lithium treatment. Neurologically normal patients and those confused only during affective episodes all had some response to lithium; the great majority of those with episodic confusion had "good" or "adequate" response. No neurologically well patient developed chronic mania. Patients with dementiform features tended to have a poorer lithium response, with 2 patients showing signs of toxicity at low serum lithium levels and 5 patients developing chronic mania. Patients with extrapyramidal syndromes had a profoundly worse response to lithium, with 12 patients developing chronic manic states. Eleven of the 15 patients with extrapyramidal syndromes developed neurotoxicity at low doses and low serum levels of lithium. In all but one case neurotoxicity developed at such low serum lithium levels (0.45–0.65 mEq/liter) that increase of lithium dose during affective relapses was precluded.

We performed stepwise discriminant analyses to determine which characteristics most clearly distinguish good from poor treatment response and which distinguish absence from presence of neurotoxicity. Table 3 summarizes the results of these two analyses and illustrates that only three of the six variables entered into the analyses were used to discriminate fully. Neurological illness constitutes almost the entire weight in determining treatment response, with minor additional weights contributed by drug abuse and medical illness.[1] The discriminant function is extremely powerful, correctly classifying over 95% of the cases and resulting in a canonical correlation of .85. A three-way analysis of variance was performed on treatment response ratings to estimate the significance of the effects of neurological status, drug abuse, and medical

[1]Forty-seven patients had 88 medical illnesses; the predominant illnesses consisted of hypertensive, arteriosclerotic heart disease (43 cases), endocrine disease (12 cases), diabetes (4 cases), and thyroid disease (8 cases).

TABLE 1
Lithium Treatment Response and Other Characteristics in 81 Elderly Bipolar Patients

Variable	Total Sample (N=81)	Good Response Patients (N=56)[a]	Poor Response Patients (N=25)[b]	Significance
Age (years)				
Mean	63.27	62.82	64.28	n.s.[c]
SD	6.93	6.78	7.30	
Age of onset (years)				
Mean	43.00	43.05	40.64	n.s.[c]
SD	13.69	12.10	12.60	
Duration of manic-depressive illness (years)				
Mean	20.98	19.76	23.64	n.s.[c]
SD	13.54	13.79	12.54	
Medical illness				
Not ill				
Number	34	27	7	n.s.[d]
Percent		79.4	20.6	
Ill				
Number	47	29	18	
Percent		61.7	38.3	
Dug abuse				
Not abusing				
Number	54	41	13	p=.04[d]
Percent		75.9	24.1	
Abusing				
Number	27	15	12	
Percent		55.6	44.4	
Sex				
Female				
Number	39	31	8	p=.03[d]
Percent		79.5	20.5	
Male				
Number	42	25	17	
Percent		59.5	40.5	

[a]Ratings of 1-3.
[b]Ratings of 4-6.
[c]t test.
[d]Fisher exact probability test.

TABLE 2
Lithium Treatment Response and Neurological Status in 81 Elderly Bipolar Patients[a]

Neurological Status	Treatment Response[b]						Total
	Optimum	Good	Adequate	Poor	Minimal	None or Worse	
Normal	28	11	4	1	0	0	44
Row percent	63.6	25.0	9.1	2.3			
Column percent	96.6	64.7	40.0	12.5			54.3
Episodic confusion	1	5	5	1	0	0	12
Row percent	8.3	41.7	41.7	8.3			
Column percent	3.4	29.4	50.0	12.5			14.8
Dementia always present	0	1	1	5	3	0	10
Row percent		10.0	10.0	50.0	30.0		
Column percent		5.9	10.0	62.5	37.5		12.3
Extrapyramidal syndrome and episodic confusion	0	0	0	1	2	5	8
Row percent				12.5	25.0	62.5	
Column percent				12.5	25.0	55.6	9.9
Extrapyramidal syndrome and always-present dementia	0	0	0	0	3	4	7
Row percent					42.9	57.1	
Column percent					37.5	44.4	8.6
Total	29	17	10	8	8	9	81
Row percent	35.8	21.0	12.3	9.9	9.9	11.1	

[a]χ^2=116.678, df=20, p<.0001.
[b]Optimum response had a rating of 1; good, 2; adequate, 3; poor, 4; minimal, 5; none or worse, 6.

TABLE 3
Discriminant Analyses: Standardized Coefficients for Variables Entered into Stepwise Analyses

Variable	Discriminant Functions: Good (56 patients) Versus Poor (25 patients) Treatment Response	Discriminant Functions: Absence (68 patients) Versus Presence (13 patients) of Neurotoxicity
Neurological status	1.03	.95
Drug abuse	.16	.32
Medical illness	.20	Eliminated
Age	Eliminated	.19
Sex	Eliminated	Eliminated
Illness duration	Eliminated	Eliminated
Canonical correlation	.85	.73
Percent of patients correctly classified	95	89

illness. Main effects of neurological illness ($F=80.63$, $df=4$, $p<.00005$) and drug abuse ($F=12.35$, $df=1$, $p<.001$) were highly significant. There was no significant effect of medical illness, nor were there any significant two- or three-way interactions.

The discriminant function for neurotoxicity was slightly less powerful (see table 3) due to the limited number of patients with neurotoxicity. Nevertheless, the major weight was again contributed by neurological illness. Drug abuse contributed substantial additional weight. There was a minor residual contribution made by age. However, univariate F tests on these three variables were significant only for neurological illness ($p<.0001$) and drug abuse ($p<.05$). Drug habituation and tolerance are, in fact, a type of reversible neuropathology, and in the above analyses they acted like mild neurological illness in their adverse effects on treatment response, their facilitation of lithium-induced neurotoxicity, and their association with chronic mania.

DISCUSSION

Since the end of the 19th century it has gradually become accepted that age has important effects on the clinical manifestations, course, and outcome of manic-depressive illness. Investigations have shown that older bipolar patients not only have a tendency to experience shallower and more frequent cycles (10), but also are more likely to develop unipolar or chronic mania (11). Our data make it clear that neurological status, not age, is the critical factor in determining the natural course of manic-depressive illness in the elderly. Neurological status not only determines the evolution of chronic mania, it also predicts poor treatment response and lithium-induced neurotoxicity. Shopsin and associates (12) observed that patients with an "abnormal baseline neuropathy [may] exhibit a decreased threshold . . . to lithium in that it exposes them to central nervous system toxicity at moderate to low levels of serum lithium," and Hays (13) demonstrated that patients who are older at the onset of bipolar illness have a reduced hereditary predisposition but an increased incidence of EEG abnormalities. The studies of Shopsin and associates and Hays may be integrated with our findings to make the following prediction: the worse the neurological illness, 1) the greater the probability of the development of a chronic manic state, 2) the less the likelihood of a therapeutic response to lithium, and 3) the higher the incidence of neurotoxic complications.

An extrapolation of these trends allows us to define a spectrum that begins with pure primary bipolar affective disorder, moves through a range of bipolar cases complicated by increasingly severe neurological illness, and ends with chronic manic states that are pure neurological illness. This endpoint is probably what Mendel described as "dementia with excitement." Indeed, in the light of this hypothesis, all three 19th-century protagonists—Kraepelin, Mendel, and Schüle—were each accurately describing a different part of this spectrum.

The most theoretically interesting segment of this spectrum is that delineated by the intersection of extrapyramidal syndromes with bipolar illness. It is not too surprising that neurotoxicity and chronic mania would somehow be associated with an overall decrease in neuronal mass, but their predominant relationship to damage of such a specific neuroanatomical structure as the extrapyramidal system suggests that there is an important relationship between the dimly understood biologies of the extrapyramidal system and of manic-depressive illness itself.

What is important for the practitioner is to detect the true culprit, which is not age, manic-depressive illness, or lithium, but neuronal destruction and deterioration. The majority of elderly patients are spared a 19th-century nihilistic denouement by virtue of the fact that they do not suffer from neurological illness. As a result, most elderly manic-depressive patients may be treated in the same ways as younger patients are. Even severe medical illness, providing it does not involve the kidneys or the central nervous system, adds little to predicting outcome. The physician may use the same guidelines for administering lithium salts to the aged as he or she does for any other bipolar patient. For example, the physician should expect the usual adverse effects of drug and alcohol abuse on treatment response. Conversely, the physician can look forward to excellent results from lithium treatment of uncomplicated bipolar illness in elderly patients.

Since the determining factor in lithium treatment of the elderly is neurological status rather than age, the physician should be warned about the necessity of conducting an accurate and comprehensive neurological evaluation of elderly lithium candidates. Any subtle evidence of chronic dementia, particularly of

extrapyramidal movement disorders, should be a warning that lithium treatment must be used very cautiously in order to avoid the possibility of either producing neurotoxicity or of markedly worsening preexisting motor dysfunction. It is primarily in this respect that Van der Velde's warnings (14) that older people require lower lithium doses and respond at lower serum lithium levels is useful. Finally, since some investigators (15–17) have suggested lithium carbonate in the treatment of tardive dyskinesia, our data should serve as a warning that lithium salts must be used with great care in this population of patients.

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Validity of the MMPI with Native Americans

BY DAVID POLLACK, M.D., AND JAMES H. SHORE, M.D.

A study was conducted to determine the validity of the MMPI for 142 American Indian patients from Pacific Northwest tribes. All the MMPI profiles were similar and had significant elevations in the Sc, Pd, and Pa scales. There was no significant difference between diagnostic groups on many clinical scales. Nonpsychotic, depressed Indian patients could not be distinguished from schizophrenic patients on any clinical scale, and there were no significant differences between antisocial-alcoholic patients and those with situational reactions. There were minimal differences between the Northwest Coast, Plateau, and Plains cultural groups. The authors believe that the similarity of all subgroup profiles demonstrates a significant cultural influence on the results of the MMPI in this population of American Indians.

The validity of psychological assessment of minority groups has been challenged because of the significant cultural differential in test response. Nonetheless, many patients from minority cultures are evaluated by standard psychological methods when they seek care in the community mental health system. This evaluation often includes traditional psychological testing, which has been standardized on the majority population. In a clinical assessment psychological test results may have a significant influence on the evaluation of the patient's behavior and problems and on the formulation of the treatment plan. In this article we report the results of an investigation on the validity of the Minnesota Multiphasic Personality Inventory (MMPI) with a population of 142 American Indians.

In a widely quoted study of scholastic failure and personality conflict among American Indian students Bryde (1) used the MMPI to evaluate personality characteristics. He studied eighth-grade American Indian students who had fallen behind white students and compared the personality variables of the two groups. Bryde identified 26 significant differences out of 28 personality variables. On each of these measures the total Indian group revealed greater personality disruption and poorer adjustment. Notable among the more meaningful variables were feelings of rejection, depression, anxiety, withdrawal, and alienation. The American Indian students who dropped out of school showed significant personality differences from the Indian students who remained in school. Kline and associates (2) used the MMPI to assess 30 male native American alcoholics in an inpatient treatment program. They concluded that the general elevation of the profiles suggested that the study group as a whole was very seriously disturbed. Of the 30 profiles, 10 had five or more T scores over 70, and 20 had three or more T scores over 70. The F, Pd, and Sc scales were consistently elevated, and the greatest pathology was found in the group with a major elevation on the Sc scale.

Hoffman and Jackson (3) used the Differential Personality Inventory to study 25 North American Indians hospitalized for the treatment of alcoholism. These patients scored significantly higher than a comparison non-Indian group on 7 of 27 clinical scales measuring cynicism, self-depreciation, and somatic complaints. However, there were no significant differences on scales reflecting character disorder. Hoffman and Jackson concluded that separate norms should be developed and that they should have standardized measures of psychopathology for specific ethnic groups. Padilla and Ruiz (4) reviewed the literature on personality test assessment of Mexican-Americans. They found a paucity of research and concluded that problems involving fluency in English obscured the findings. Davis (5) studied the influence of race on MMPI results. He pointed out that when educational level was controlled there were no race differences on any of the 11 MMPI scales. Davis felt that social and economic variables were as significant as racial differences in affecting the interpretation of the MMPI.

METHOD

This study was conducted to determine the validity of standard MMPI psychological assessment for the

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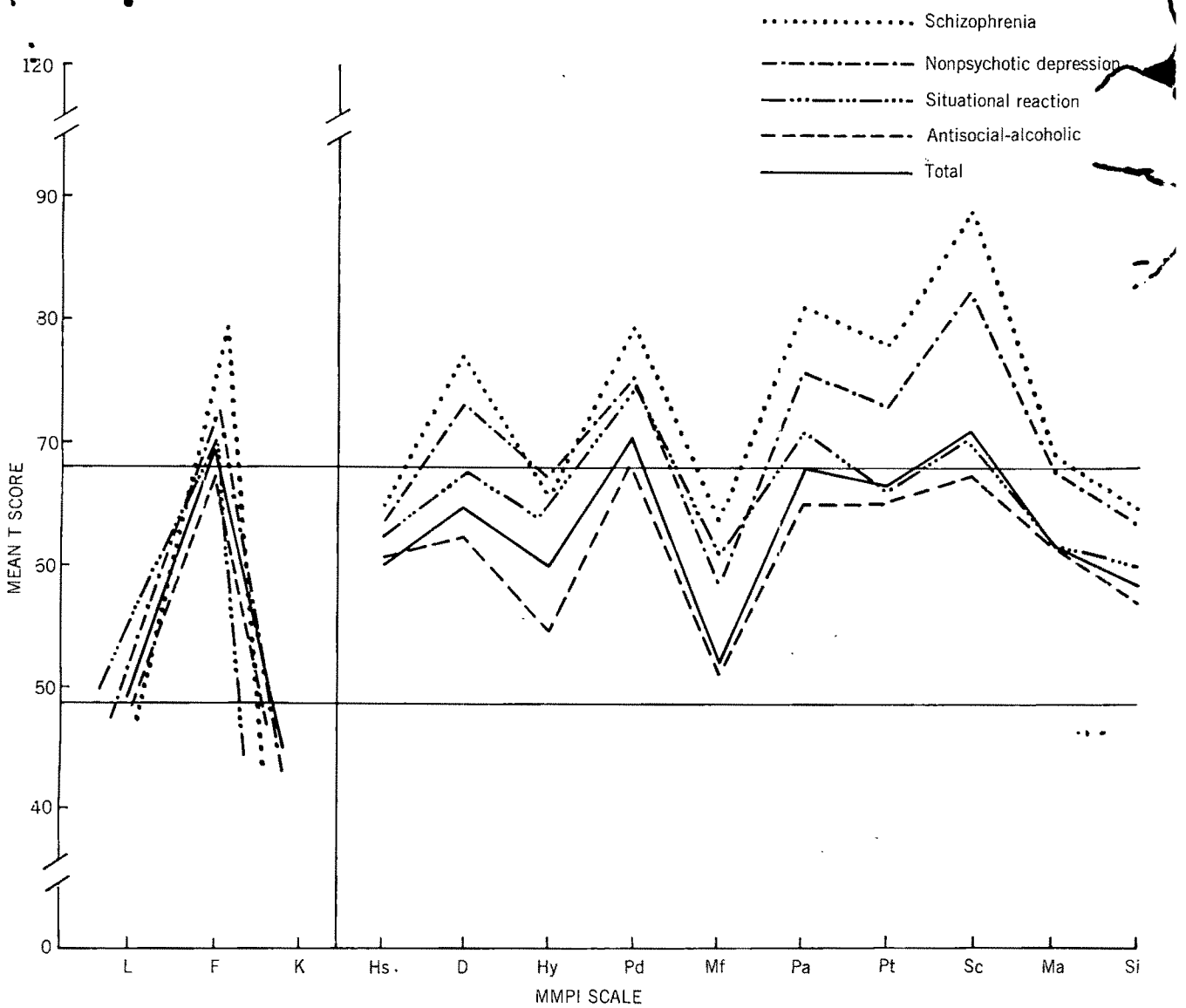
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FIGURE 1
MMPI Profiles of 53 Native Americans, by Diagnosis



Mental Health Program of the Portland Area Indian Health Service, a branch of the U.S. Public Health Service. This regional mental health program for American Indians was initiated in 1969 with an interdisciplinary mental health team to develop services for 10 American Indian reservation communities in Oregon, Washington, and Idaho. In 1971 the mental health staff began to use a standardized, computer-scored MMPI. From 1971 to 1974, 142 MMPIs were completed by American Indian patients from Pacific Northwest tribes. The tribal groups belonged to one of three distinct cultures: Northwest Coastal, Plateau, and Plains. The psychological test results were used by local mental health staffs.

In 53 of the 142 cases the patients were independently evaluated by one of three senior mental health consultants, who made a psychiatric diagnosis by *DSM-II* criteria. Twenty-one of these 53 patients were imprisoned for antisocial behavior related to alcohol abuse. Seventeen patients were given a diagnosis of situational reaction, 9 of nonpsychotic depression, and 6 of schizophrenia. The three consultants met weekly to discuss the patient evaluation and diagnostic criteria. Many of the 53 patients were evaluated by at least two of the consultants. We calculated the means of K-corrected T scores from the MMPI profiles and compared them by age, sex, diagnosis, and cultural grouping.

RESULTS

The most impressive finding was the similarity among MMPI profiles (see figure 1). The mean profile for all 142 patients shows significant elevations in the

TABLE 1
Rank Order of Mean MMPI Scale Scores[a] for 142 Native Americans

Subgroup	MMPI Scale									
	Hs	D	Hy	Pd	Mf	Pa	Pt	Sc	Ma	Si
Age										
Under 29 (N=86)	9	5	8	2[b]	10	3[b]	4[b]	1[b]	6	7
29 or older (N=56)	7	4	8	1[b]	10	3	5	2	6	9
Sex										
Male (N=68)	8	3	9	2	10	5	4	1[b]	6	7
Female (N=74)	9	5	7	2[b]	10	3[b]	4	1[b]	6	8
Culture[c]										
Northwest Coastal (N=28)	8	5	6	1[b]	10	4	3	2[b]	7	9
Columbia Plateau (N=72)	8	5	7	2[b]	10	3[b]	4	1[b]	6	9
Plains (N=16)	8	6	9	3[b]	10	2[b]	4[b]	1[b]	5	7
Diagnosis[d]										
Schizophrenia (N=6)	9	5[b]	8	3[b]	10	2[b]	4[b]	1[b]	6	7
Nonpsychotic depression (N=9)	8	5[b]	7	3[b]	10	2[b]	4[b]	1[b]	6	9
Situational reaction (N=17)	9	6	8	1[b]	10	3[b]	4	2[b]	5	7
Antisocial-alcoholic (N=21)	7	6	9	1	10	5	4	2	3	8
Total	8	5	7	2[b]	10	3	4	1[b]	6	9

[a]1=scale having highest mean score; 10=scale having lowest mean score.
[b]Score greater than 70.
[c]For the three primary cultures represented (N=116).
[d]Of patients who received a diagnosis (N=53).

F, Pd, and Sc scale scores. The Pd and Sc scales are among the three scales with the highest scores for each subgroup. Table 1 summarizes the profile patterns for the 11 subgroups. A significant amount of pathology is indicated by the high incidence of mean T scores above two standard deviations (mean score of 70) on the Pd, Sc, and Pa scales. The Pd and Sc scales are above 70 in 9 of the 11 subgroups, and the Pa scale is above 70 in 7 subgroups. Younger subjects had significantly higher scores on the F, Pd, Pt, and Sc scales. Women scored significantly higher than men on the Hy, Pd, Pa, and Si scales, and men scored higher on the Mf scale (masculine-feminine identification).

There were no significant differences between the antisocial-alcoholic patients and the patients with situational reactions or between the schizophrenic and nonpsychotic depressive patients. The combined profile of the schizophrenic and nonpsychotic depression patients was compared to the combined situational and antisocial-alcoholic patient subgroups. The schizophrenic-depressed subgroup scored significantly higher on the D, Hy, Pa, Pt, and Sc scales, but the differences between subgroups was only in the degree of pathology, not in the profile pattern.

There were no significant differences between the MMPI scale scores of the patients from the Coastal culture and those of the patients from the Plateau culture. Patients from the Plains culture had significantly higher F (validity) and Hy scores than the other two cultural groups.

DISCUSSION

The similarities of all the subgroup profiles demonstrate that there is a significant cultural influence on the results of the MMPI in this population of American Indians. It appears that cultural influence overrides individual pathology and personality differences in influencing the pattern of the MMPI. This cultural response is demonstrated by the similarity of profiles between younger and older patients and between men and women, and across diagnoses and cultural subgroups. The higher Sc, Pd, and Pa scale scores may reflect a group that has been defined as bizarre or deviant by the dominant culture. In a recent report on MMPI interpretation, Graham (6) stated that in white Americans the "[48 of 84] individuals do not seem to fit into their environments. They are nonconforming and resentful of authority, and they often espouse radical religious or political views." The responses are compatible with those of an individual raised in a nonwhite minority culture. An American from the dominant culture who has taken the MMPI may meet all the designated criteria of a particular diagnosis, both behavioral and clinical, and be diagnosed correctly by this psychometric method. However, the patient from the dominant culture is being compared on a profile that has been developed by MMPI responses of known cases from the same culture. In the current application of the MMPI to American Indian subjects there has been no such cultural standardization.

The higher scores for women on 6 of the 10 clinical scales point to an important sex differential in response to stress where psychiatric symptomatology is measured by the MMPI. The statistically significant higher score for Indian women on the Pd scale is surprising and is further indication that this deviance is sociocultural rather than psychopathic.

The profiles of the schizophrenia and nonpsychotic depression subgroups do not differ significantly on any clinical scale. Because of the small number of patients

in these two diagnostic subgroups, this finding needs confirmation by additional research. However, the data point to the possibility that cultural influences significantly overshadow clinical and pathological differences in the MMPI response. American Indians with nonpsychotic depression appear to describe their symptomatology in a manner identical to that of schizophrenic Indian patients. This interpretation is supported by independent clinical observations that many American Indian patients with a grief reaction experience auditory hallucinations, that is, hear the voice of the deceased person. An inexperienced diagnostician usually will label these nonpsychotic grief symptoms as schizophrenic.

Significant differences were found between the "more impaired" (schizophrenic and nonpsychotic depressive) patient groups and the "less impaired" patient groups (situational reaction and antisocial-alcoholic). However, these differences were ones of degree, not pattern. Also, the profiles of patients with situational reactions were no different from those of the antisocial-alcoholic imprisoned group. The similarity of profiles for schizophrenic and nonpsychotic depressed patients in the American Indian population indicates that there is no valid diagnostic assistance with this test in confirming a major psychosis. Since higher subscale scores are significantly influenced by cultural differences in test response, the clinician must be cautious in using the MMPI as part of the diagnostic evaluation of American Indian patients. The higher test scores and cultural specificity introduce the likelihood of overdiagnosis and misinterpretation of the clinical status.

While the staff in this study did not find language skill to be an overt problem in the subjects' completion of the MMPI, cultural specificity in the comprehension and interpretation of word meanings can be a significant barrier to cross-cultural psychological testing and psychiatric evaluation. Leff (7) demonstrated that patients from different cultures show a differential ability to distinguish between various affective feelings and that these differences can be documented with a structured psychiatric examination that contains many items similar to various MMPI questions. Leff tested his hypothesis by using material derived from the International Pilot Study of Schizophrenia. In that study of psychiatric patients in nine different countries, a standardized interview schedule was used for assessing the mental state. It was translated from English into the five Indo-European languages (Czech, Danish, Hindi, Russian, and Spanish) used by the countries involved and the two non-Indo-European languages (Yoruba and Chinese). Patients who speak Indo-European languages can differentiate anger from depression and anxiety, whereas non-Indo-European language patients have difficulty differentiating all three emotions from each other.

From another perspective, Glott (8) gathered data to evaluate the MMPI by translating it into German, Spanish, and French. He then administered it to bilingual patients, who took the test in English and in one of the other three languages. The results showed that certain scales (pathology, deviance, and peculiarity) were likely to indicate more abnormality in the translations than in English. None of the American Indian subjects in our study took the test in a second language, but they were raised in cultures removed from a non-Indo-European language by only one or two generations. To our knowledge, no one has evaluated the relationship of English language usage and emotional states in American Indian cultures, but it is possible that like the non-Indo-European patients of Leff's report, the American Indian subjects have difficulty discriminating certain emotional states as defined in the MMPI. This difficulty could raise the score and inaccurately emphasize selected profiles.

Adebimpe and associates (9) recently reviewed MMPI diagnoses of black psychiatric patients and concluded that black-white differences in MMPI norms also may lead to significant misclassification of black patients. Their review demonstrated that blacks scored significantly higher on the validity (F), schizophrenia (Sc), and hypomania (Ma) scales, leading to false positive diagnosis in schizophrenia. While American Indian patients show a different pattern of elevated scales, the impact of the MMPI profile on the diagnostic formulation could be much the same.

Brislin and associates (10), in a comprehensive review of cross-cultural research methods, emphasize the need for extensive investigation in this area:

> The cross-cultural possibilities with the MMPI continue to be intriguing, though there are many who blanch at the sight of the instrument. It would be folly and a waste to dismiss all United States and other MMPI data, suggesting that the only reasonable approach with the MMPI would be to develop cultural norms. It would be equally foolish to tread in this jungle of clinical and psychometric data without utmost caution. No previous MMPI translation or cross-validation study across cultures with commendable accuracy can be found in the literature. What is critically needed is a series of cross-cultural cross-validation studies with the MMPI that document empirically the measured similarities and differences of clinical diagnosis and personality assessment as a function of culture.

While our study focuses on cross-cultural validation of the MMPI and identifies errors of interpretation when using the standard instrument without cross-cultural norms, it may be erroneous to conclude that a modified cultural norm will correct the MMPI for American Indian patients. It is not only cultural norms for standard tests that need development but cultural research that identifies culturally appropriate instruments from the outset. Certainly the use of the MMPI in a cross-cultural setting amplifies the risk of a false perception of the patient's psychological state. While clinical judgments may also be erroneous, a di-

agnostic interview can include techniques that may be more reliable. For example, direct diagnosis can be strengthened by the participation of mental health workers experienced with American Indians (professionals, paraprofessionals, and people involved in traditional medicine) in the evaluation process to provide a cultural perspective for the psychiatric diagnostician. If expert cross-cultural consultation is not available, a healthy skepticism about such testing should replace any standard clinical use of the MMPI until additional research demonstrates a valid clinical application.

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EDITORIAL

Sex and Heroin

The relationship between sex and heroin has been the subject of much research and more lucubration. As early as 1926, Rado (1) had developed the concept that equated narcotics injection with sexual orgasm; thus drive reduction in the form of relief of sexual tensions was postulated as the mechanism responsible for the appeal of narcotics.

Although there is ample evidence for this hypothesis in the language and folklore of the street, scientific confirmation has been lacking. In the past the lack of chemically pure and legal heroin has been a major obstacle. However, the shift from a religious-legal to a scientific-therapeutic approach to drug abuse now makes it possible for some of us to study the mode of action of heroin, catching it *in flagrante delicto* by replicating the pharmacologic aspect of the street scene in the laboratory. The availability of pure heroin coincides with the development of improved and sophisticated techniques of analysis (radioimmunoassays) for the quantification of pituitary gonadotropins and testosterone.

Mirin, Meyer, Mendelson, and Ellingboe in their article "Opiate Use and Sexual Function" in this issue provide convincing biochemical evidence of the relation between sex and heroin and for the drive reduction hypothesis. These studies show that the intravenous injection of heroin results in an immediate fall of luteinizing hormone (LH). This response was abolished by pretreatment with naltrexone. Chronic administration of heroin leads to a fall in plasma testosterone, which may account for the persistent dysphoria of the chronic addict. The authors have discovered in yet a third experiment that "heroin-induced suppression of LH secretion was first evident 2–4 hours and was greatest 4–6 hours after unblocked heroin injection." The maximal decline in plasma testosterone occurred 6 hours after injection of heroin.

The authors postulate on the basis of correlative animal work that acute heroin administration has a central effect on the release of hypothalamic luteinizing hormone releasing factor (LRF). The regulation of LH via LRF has been localized to ventral and medial areas of the hypothalamus, areas possessing many opiate receptor binding sites and a high concentration of endogenous opiate peptides.

Again, in an effort to synthesize clinical and physiological knowledge, the authors postulate that the intensive pleasurable "rush" which follows intravenous injection of heroin involves massive release of catecholamines at both central and peripheral sites. The authors are unable to document this suspicion. Fortunately, they have left some work for the rest of us.

In thus tying together laboratory and clinical findings, the authors have done the field of addiction a considerable service and lend support to the drive reduction theory of addiction. These results should command the attention not only of all workers in the field of the addictions but of all those interested in the relation of human function to behavior.

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BRIEF COMMUNICATIONS

The Use of a Correctional Setting for Follow-Up Care of Psychiatrically Disturbed Adolescents

BY DOROTHY OTNOW LEWIS, M.D., AND SHELLEY S. SHANOK, M.P.H.

The authors studied the first 88 adolescent boys who were placed on the secure unit of a correctional school during an 18-month period and found that 52 (59.1%) of the boys had previously received psychiatric residential treatment or psychiatric hospitalization. The authors hypothesize that many of these boys may have been transferred from a psychiatric setting to a correctional facility during adolescence when long-standing aberrant behaviors became more threatening to staff. At present the number of psychiatric hospital beds is decreasing and the criteria for hospitalization of children are becoming more stringent. The data indicate that correctional facilities are now expected to function as psychiatric treatment centers for disturbed adolescents no longer welcome in therapeutic settings.

The purpose of this paper is to report the transfer of psychiatrically disturbed children from psychotherapeutic settings to correctional settings as they reached adolescence. It documents the previous placements of a sample of seriously delinquent incarcerated adolescent boys. It also documents the fact that the recognition of serious psychopathology in delinquent children was not the idiosyncratic observation of clinicians seeing the children in adolescence, allegedly a turbulent period of development. Rather, it documents the repeated recognition of severe psychopathology by numerous clinicians who saw these children at different stages of their development throughout childhood.

THE LITERATURE

Several studies (1–3) have reported the subsequent serious psychopathology of formerly antisocial and/or delinquent children. Conversely, Silverman (4) has documented evidence of serious childhood psychopathology in the histories of adult prisoners. Other investigators (5–8) have focused on the early delinquent behavior of psychotic adults. To the best of our knowledge, with the exception of Lauretta Bender's paper (9) on pseudopsychopathic schizophrenia, there has been little in the literature regarding the previous psychiatric placements of delinquent children.

CLINICAL OBSERVATIONS

The present study sprang from the clinical observation that many of the children placed on a secure unit of a correctional school in Connecticut during an 18-month period had previously been identified as psychiatrically disturbed and had been in psychiatric residential treatment or had been psychiatrically hospitalized. For example, one youngster, adopted in infancy, had serious school difficulties in early childhood in spite of what was described as a "high average" intelligence. Throughout childhood he received private outpatient psychiatric treatment and was placed in a special supportive remedial school program. When, at adolescence, he got into a high-speed chase with police while driving the family's car, he was psychiatrically hospitalized. He was unhappy at the hospital (where he was given the diagnosis of borderline schizophrenia) and ran away on several occasions. He was eventually

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committed to the correctional school, where he stayed through his 16th birthday.

Another boy, who was the child of two psychotic parents, was first placed in a state receiving home at age 10 when he was found by police wandering the streets aimlessly, "involved in dangerous behaviors." His parents were found guilty of neglect, and the boy was subsequently placed in a psychiatric residential treatment facility. There he was described by clinicians as being "unable to differentiate between reality and fantasy." Over the course of several years he had multiple placements and received multiple psychological and psychiatric evaluations. At age 12, he was described by one psychologist as "filled with poorly formed fearful ideas." A psychiatrist described him at age 14 as "a very disturbed youngster emotionally" who was suffering from a "latent schizophrenia." He was eventually sent to a state psychiatric hospital after having set fires both in a residential treatment facility and in his aunt's apartment while on home visits. After a 30-day evaluation in the hospital, he was transferred to the correctional setting in spite of a judge's recommendation that he be placed in a special therapeutic setting because of his serious psychiatric problems and his "insubstantial delinquency record."

Another child was psychiatrically hospitalized 8 times, then transferred to the correctional school. And still another youngster, who had spent 4 years in a psychiatric hospital because of dangerous psychotic behavior and evidence of neurological dysfunction, was ultimately sent to the correctional school when, on discharge from the hospital at age 12, he wandered the streets, grabbing at women's breasts. His hospital discharge summary indicated that he was returned home after 4 years in order that he not become "institutionalized."

These were but four of the many children who were sent to the secure unit of the correctional school and who had previously been in residential psychiatric care. We wondered what percentage of the children sent to this unit had previously been in psychiatric residential treatment or had been psychiatrically hospitalized.

METHOD

Our sample of incarcerated children consisted of the first 88 boys who were placed on the secure unit of a correctional school during an 18-month period of time. Admission to the unit was determined by school administrators and judges based on the severity of delinquent behaviors.

The major source was the correctional school record, which almost invariably contained information regarding previous placements. In fact, correctional school records often contained copies of actual hospital or residential facility clinical reports. In addition, the boys in the study were interviewed and, whenever possible, a parent or guardian was contacted in order to verify information.

FINDINGS

Of the 88 securely incarcerated boys, 52 (59.1%) had previously been either in residential psychiatric treatment or psychiatrically hospitalized. Twenty-nine children had been in residential psychiatric treatment only; 9 children had been psychiatrically hospitalized only; and an additional 14 children had been both psychiatrically hospitalized and in residential psychiatric treatment.

In addition to having been psychiatrically hospitalized and/or in residential treatment, 28 of the 52 treated children had also spent time in foster homes or group homes. Only 9 of the 88 boys had been in foster or groups homes only. Twenty-seven boys had never been placed outside their homes.

Considering, now, the entire sample of 88 boys, the average number of placements a child had experienced was 1.818. This average included those children who had never been placed outside their homes. When the mean number of placements for boys ever placed outside their homes was calculated, the average number of placements was 2.622.

DISCUSSION

Our present data indicate that many seriously delinquent incarcerated adolescents have, during childhood, manifested signs and symptoms of severe psychopathology leading to inpatient psychiatric care. Many had also spent time in foster or group homes, which suggests that their parents were often unable to care for them. These epidemiological findings are consistent with two of our previously reported findings. First, we found that many delinquents were the children of seriously impaired parents (10). Second, we found that the majority of violent incarcerated delinquent boys whom we studied suffered from a variety of neuropsychiatric disorders that impaired their ability to adapt appropriately to their environments (11). The finding that 59.1% of the incarcerated boys had, prior to incarceration, required residential psychiatric care indicates that clinicians other than ourselves recognized the degree of psychiatric impairment in this population. In fact, they had made the effort to obtain residential treatment for these children, an indication of their concern.

One must therefore ask why these seriously disturbed youngsters were subsequently transferred to a correctional facility. A review of the children's early clinical records revealed that most of the youngsters with some psychotic symptoms and/or signs of neuro-

logical impairment posed serious behavior problems from early childhood onward. Nevertheless, as small children they were able to secure acceptance at residential facilities and hospitals. As these children matured physically, they continued to pose the same kinds of behavior problems that they had manifested earlier. Although in a few cases dramatic behavioral changes in adolescence could be documented, for the most part assaultiveness, temper tantrums, firesetting, and stealing were long-standing problems that during early childhood, along with neurological and often psychotic symptoms, were recognized as manifestations of psychopathology and were handled within the therapeutic settings. It was only when a child's increasing size made his aberrant behaviors more threatening to staff and other children that he was discharged to his home or transferred to the correctional school. The seriously disturbed adolescent was perceived as more violent and, indeed, his outbursts were potentially more dangerous because of his increased strength.

Discharge to the child's home often had the same effect as transfer to corrections—that is, parents who had been unable to manage their children when the children were younger were no better equipped to deal with the youngsters during adolescence. Hence, discharged children came into conflict with the law and eventually made their way to corrections. Noteworthy was the tendency of hospitals and treatment centers to change diagnoses at the time a child reached adolescence, although the child's symptoms remained relatively the same as they had been in childhood. Thus, children formerly given a diagnosis of borderline psychotic or minimally brain damaged were redefined as "aggressive acting out" or "antisocial" adolescents. In one instance, even previously diagnosed psychomotor epilepsy in a child with a documented temporal lobe focus of abnormality was rediagnosed as "explosive personality" for purposes of transfer. In essence, potentially treatable disorders such as schizophrenia, minimal brain dysfunction, and epilepsy in children with aggressive symptoms were redefined as ostensibly less treatable sociopathic disorders in order to justify the children's transfer to the correctional school.

The finding that seriously psychiatrically disturbed children are often transferred from therapeutic settings to correctional facilities at the onset of adolescence is particularly ominous today, when hospital beds are decreasing in number and the criteria for the psychiatric hospitalization of children are becoming increasingly strict. In the recent past, psychiatrists and attorneys have focused much energy and attention on the rights of juveniles *not* to be hospitalized (e.g., the Mental Health Law Project) (12). However, little if any attention has been given to the rights of psychiatrically disturbed youngsters to continue to receive necessary residential or hospital care through adolescence, when their disturbed behaviors are acknowledged to be dangerous to others. Since the criteria for the incarceration of children are extremely lenient (a child need only have committed a nonstatus offense of one sort or another), we can expect even greater numbers of seriously disturbed children to be sent to correctional schools in the future. Tragically, once a child has been designated delinquent (much less incarcerated), it is almost impossible to reinstitute residential psychiatric treatment for him in a reputable facility.

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The Psychiatrist's Role in Aerospace Operations

BY WILLIAM H. SLEDGE, M.D., AND COL. JAMES A. BOYDSTUN, MC, USAF

This paper presents two unique aspects of aerospace psychiatry: the influence of the specialized stressors and occupational requirements of an aviation career and the ambiguous role of the aerospace psychiatrist. Aerospace psychiatrists have multiple, sometimes conflicting, responsibilities to the organization and society (the social control task) and to the individual aviator (the humanistic and medical tasks). In the two case reports below the authors describe airmen who had vasovagal syncope and how the psychiatrist intervened and resolved these conflicting tasks.

Aerospace psychiatry is the practice of occupational psychiatry in the context of aviation and space operations. Two aspects of aerospace psychiatry present unique challenges to practitioners. First, the specialized occupational environment of aerospace operations entails particular psychosocial stresses, which result in a variety of altered priorities and paradoxes; for instance, benign medical disorders may have quite serious occupational implications. Second, the role of the psychiatrist is ambiguous because the psychiatrist must simultaneously serve multiple tasks, some of which may be in conflict.

AEROSPACE OPERATIONS AND STRESSORS ON AVIATORS

Advances in aerospace technology demand efficient and error-free functioning because errors are especially amplified by the high-performance capability of aircraft. Furthermore, the high-performance capability of aircraft creates conditions that have special stressors for the aviator. Some of these stressors are acceleration forces, transmeridian travel with jet lag, high-altitude flying, and flying in extremes of temperature. We will not address space travel here.

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An increased number and range of tasks can be performed by flight crews because of automated and computerized flight aids. However, the complexity of aircraft and the enormous increases in air traffic have resulted in more rigidly controlled safety requirements. These include precise procedures and regulations for negotiating weather and high-density traffic centers, more exacting medical surveillance of flight crew, and increased proficiency checks and adherence to training needs. Psychosocial factors of crew interaction, separation from family, fatigue-rest cycles, and time-zone travel are potential stressors. Consequently, professional aviators are caught in a series of demanding paradoxes; they must be able and willing to submit to numerous regulations, procedures, and rules in their professional lives, yet they must maintain a sense of responsibility and initiative in the execution of their work. They must be calm, thoughtful, and free of impulsive tendencies; however, they must be quick to react adaptively to emergent situations. Aviators must be able to tolerate stretches of long hours of work alternating with long periods of rest. Frequently, they and their families must be able to endure long periods of separation alternating with long periods of being idle at home. They must be able to make quick, effective decisions when given incomplete information, but they must always be looking for more facts about their present situation. They must also have a healthy, enduring love of flying tempered by a realistic respect for its dangers. They must be self-confident and convinced of their ability to survive dangerous situations; yet they must not be so grandiose that they take foolish chances or so unsure of themselves that they constantly must prove their mastery and invulnerability.

The typical flyer is achievement-oriented, with a high need to perform well. Competitive and aggressive needs are adaptively channeled into work and mastery of the flying environment (1, 2). The average flyer uses action to deal with ambiguity and psychic conflict and to achieve gratification. As a group, these men and women have obsessive-compulsive tendencies; however, these traits are usually flexibly deployed and do not necessarily represent neurotic conflicts or immutable styles.

Perhaps the most important personal trait for a flyer who wants a successful and safe aerospace career is motivation, particularly in terms of its coherence and fidelity with past experiences, its formal characteristics, and its content in organizing drives and needs

into appropriate adult activity (3). One theory (4) suggests that a successful flying career is an integration of infantile erotic and aggressive urges with the appropriate cognitive and affective attributes. The aircraft may become a powerful instrument of aggressive potential that is experienced unconsciously as a narcissistic extension of the self. The death-defying aspects of a flying career, coupled with the competitive rivalry with others, enhances the narcissistic gratification inherently involved in competent, masterful performance.

THE PSYCHIATRIST'S ROLE

Inevitably there will be failures and breakdowns in human functioning. When dysfunction occurs, the psychiatrist should be involved at three functional points in the aeromedical evaluation: diagnostic assessment from a clinical disease perspective, functional/adaptive assessment in terms of how the disorder has been influenced by and may continue to influence the job, and treatment.

In terms of role functioning, aerospace physicians are in a position of potential task conflict. Physicians who have primary contact with the patient must not only have patient care as their main function but they must also have a social control function whereby they work as agents of the organization to ensure mission completion and/or as protectors of public safety by identifying health conditions in the patient that threaten job functioning. This social control function of psychiatrists has been well characterized by Astrachan and associates (5). In addition the aerospace psychiatrist has the humanistic task of fostering the patient's development and opportunity and maximizing the patient's potential, as well as the medical task of diagnosing and treating the patient's disease; these may all conflict with the social control task.

Diagnostically, the aerospace psychiatrist assists other aerospace physicians and clinical specialists by helping them rule out functional disorders and clarify the relationships between psychosocial elements and altered biology. In addition to providing standard nosological or diagnostic categorization, the psychiatrist contributes by making a thorough evaluation of possible antecedent psychological and social factors in the onset of the disorder. In view of our research findings on syncope (6, 7), a careful appraisal of work-related stressors is particularly relevant.

As a behavioral scientist, the aerospace psychiatrist is in a good position to make a functional/adaptive assessment of how a particular disorder may influence job performance. The aerospace psychiatrist needs a clear understanding of possible psychosocial precipitants and the psychosocial consequences of the disorder. Ideally, the two perspectives on treatment outcome—the individual's interest in relief of suffering and the organization's interest in safe and reliable functioning—will not be in conflict. However, if there is conflict, the needs of the organization for safety and reliability must take precedence over the needs of the individual for employment and professional growth. This dilemma for the aerospace psychiatrist may occur in situations where the aviator has a form of intrapsychic conflict that is about to reach expression. Then the psychiatrist is faced with the problem of opting for either short-term public safety, by intervening to bolster the patient's repressive defenses, or a more therapeutic approach, by making the patient's conflict more manifest, which may lead to further temporary work dysfunction. Although this type of dilemma arises at times, usually the patient's interests and tolerance for psychological work will determine how the psychiatrist intervenes.

When the psychiatrist represents the needs of the organization, there are various potential problems in the interventions that stem directly from his or her social control task. These problems are most obviously clear in the doctor–patient relationship, in which the usual potential obstacles and resistances may be exacerbated by patient wariness and distrust. For instance, the patient may be reluctant to cooperate and will consciously withhold data that he or she thinks may be condemning in the eyes of the psychiatrist. Usually an explicit early acknowledgment of this process is helpful in enhancing meaningful collaboration. Most aviators fear they will be grounded for an obscure reason that they believe is irrelevant to their safe and reliable function. At the same time most of them are highly safety conscious and are not willing to fly with a condition that *they* believe is dangerous. Along these lines collaboration can usually be maintained. In more intermediate or long-term treatments in which exploration, uncovering, and insight are treatment goals, the treating physician's attitude toward the social control task must be kept very clear.

Another potential problem area is the psychiatrist's attitude toward the dual tasks of individual care and social control function. A balanced double perspective requires the psychiatrist to resist overidentifying with the patient at the expense of ignoring the organization's needs and to resist rigid application of organization rules (thereby prematurely foreclosing exploration) as a result of hostile countertransference feelings.

We will report two cases of vasovagal syncope to highlight the multiple aspects of the aerospace psychiatrist's functioning. Vasovagal syncope is a benign disorder from a health perspective, but it may present a threat to reliable professional functioning, depending on its context of onset and repetitions. There is good evidence that the onset of vasovagal syncope is dependent on a variety of psychosocial elements (6–8), including the specific context in which it occurs and broad nonspecific factors such as personality or character, antecedent psychological stress, negative feelings about work, and illness.

CASE REPORTS

Case 1. Mr. A, in his mid 20s, was a fighter jet pilot who fainted while having his blood pressure taken during an annual physical examination. A thorough medical and neurological workup revealed no disease or pathological condition as the basis for his fainting. He had fainted previously during an annual flight physical examination, and throughout his life medical procedures had made him uneasy. Furthermore, watching others clean and dress killed game after hunting made him queasy. However, he had been in situations where he and/or others had been injured or in pain and had had no syncope-like symptoms, for instance, viewing a dead friend's body and participating in his child's birth. There had been no clear psychosocial antecedents such as separations, loss, or job stress before this latest episode.

He gave a history of unconflicted competition and ambition to do well. Mr. A impressed the examining Air Force psychiatrist as an active, outgoing, action-oriented man. He had had a successful flying career, and his interest in and motivation for a flying career seemed to be appropriate and well integrated.

Although intially somewhat wary, Mr. A was able to appreciate during the course of several interviews with the examiner how physical manipulation of his body, however benign, together with a stance of enforced passivity and resignation were critical antecedents in his syncopal episodes. He was able to overcome this particular situation by assuming a more active orientation toward the numerous medical procedures and examinations being carried out during his aeromedical evaluation. An in-depth understanding of the dynamics of his "neurosis" was not attempted.

Although there were neurotic components in Mr. A's fainting response, this aspect of his psychopathology seemed discrete and limited and did not interfere with his flying; furthermore, the condition seemed influenced by education. It was recommended that Mr. A return to his flying duties.

In this case the psychiatrist was able to support the diagnosis of vasovagal syncope and rule out psychosocial factors that may have interfered with continuing job reliability and safe functioning. Furthermore, Mr. A and the psychiatrist were able to elaborate an understanding of the important psychodynamic elements in his syncopal episodes so that he was able to do something about the problem. His recurrent syncope was believed to be occupationally benign.

Case 2. Mr. B, a navigator in his mid 20s, fainted during a venipuncture procedure during an annual flight physical examination. A thorough physical and neurological evaluation revealed no somatic pathological basis for the syncopal episode. He had fainted three times before during medical or dental procedures that entailed receiving an injection. He almost fainted on many other occasions: during his wife's labor when she received an injection, when he came upon an accident and saw people injured and bleeding, and when he saw two people killed. Furthermore, he had had in-flight episodes of syncope-like symptoms during a variety of emergent and dangerous in-flight events, but he had never lost consciousness at these times.

Although there were no discrete antecedent psychosocial stressors before his last episode of fainting, Mr. B indicated that he had grave reservations about his suitability for a flying career. He wanted to serve out his commitment, but he found the work tedious, unfulfilling, dangerous, and frightening. During the evaluation he was found to be perfectionistic, overly conscientious, and severe with himself. He was not clinically depressed.

Mr. B was grounded and disqualified from flying on the basis of his recurrent episodes of syncope against the background of inflight episodes, conflicted feelings about his work, and questions about motivation for a flying career as a navigator. He was opposed consciously to the grounding and refused to accept a trial of psychotherapy after which time he could be reevaluated. The psychiatrist, therefore, recommended he be disqualified for flying. The social control function took precedence.

DISCUSSION

These two patients resembled one another superficially, but the relationship of the illness to flying was different. In both cases the psychiatrist was able to support the diagnosis of vasovagal syncope and make an assessment of the importance of antecedent psychosocial factors. Mr. A did not have a significant work-related dysfunction, and there were no apparent secondary gain benefits. The conflict was dealt with through rationalization and education. This approach permitted Mr. A to establish mastery over his symptoms, but he probably had no insight in the usual psychotherapeutic sense. The benign disorder of vasovagal syncope presented a possible threat to Mr. A's reliability as an aviator. The psychiatrist's assessment, however, suggested a focal neurotic process concerning enforced passivity during medical examinations; it did not reveal a dysfunctional response to the psychosocial stressors of an aviation career nor an inappropriate or conflicted motivation for a flying career. There was no task conflict for the psychiatrist.

Mr. B's medically benign disorder of vasovagal syncope did pose a threat to operational reliability and safety. The psychiatrist's evaluation revealed a more diffuse neurotic process involving a strong vagal response to many situations that were perceived as threatening, at least unconsciously, and that could not be countered. Furthermore this neurotic process was sensitive to the dangers and stressors of a flying career. He had an additional problem of conflicted motivation for flying and a character structure that compounded these problems. In Mr. B's case the psychiatrist experienced an element of task conflict: his social control function dictated that this patient be grounded and the humanistic function pointed toward trying to make it possible to work out his problems. The social control function clearly took precedence when Mr. B refused to accept grounding and a trial of psychotherapy.

The primary function of the psychiatrist who works as a part of the aeromedical evaluation process is to assess the relation of the patient's disorder to the occupation's psychosocial stressors and to assess how

the disorder relates to occupational functioning. In order to do this work effectively, the aerospace psychiatrist must not only know the aviator's character, motivation, psychopathology, and life events, but he must also understand the unique characteristics of aerospace operations. Furthermore, aerospace psychiatrists must be aware of the ambiguity of their own role in that they have responsibilities to the organization and society (the social control task) and to the aviator (the humanistic and medical tasks).

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Psychiatric Participation in the Hootch Case: Effects on Alaska Native Education

BY JOSEPH D. BLOOM, M.D., AND JACQUELINE L. BLOOM, M.S.

The Hootch case was a class action suit that established the right of Alaska Native students to attend secondary school in their home communities. The authors review the history of Native education, give the background of the suit, and present behavioral science data on school adjustment of Alaska Natives and American Indian students. These data, included in an amicus curiae brief, were incorporated into the court decision. The authors consider this case a positive example of psychiatric involvement in social policy matters.

The 1972 case of *Molly Hootch et al v Alaska State-Operated School System* (1) dramatically changed secondary education for Alaska Native students. In this paper we will review the history of the educational system for Alaska Native students, including the problems that led to the Hootch case, and will focus on the psychosocial effects of this system of education.

The Hootch case was a class action suit brought by Molly Hootch, a representative of Alaska Native village adolescents, who was attempting to secure educational opportunities for them. Specifically, Hootch and her fellow students were seeking to establish their right to attend secondary school in their home communities. The case involved no direct psychiatric testimony. However, psychiatric data on the effects of the secondary education system on Alaska Native students were provided through an amicus curiae brief that was submitted jointly by the local Alaskan chapters of the American Psychiatric Association, the Alaska State Medical Association, and the American Academy of Pediatrics (2).

ALASKAN NATIVE SECONDARY EDUCATION: HISTORICAL DEVELOPMENT

In 1884, 17 years after the Alaska purchase, Congress passed legislation that incorporated the new territory into the United States as an administrative juris-

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diction (3). Within the confines of this act, Congress set up an administrative procedure for the provision of education to the newly formed territory through the Bureau of Education. The first agent of the United States Bureau of Education in the territory was Dr. Sheldon Jackson, a Presbyterian minister. Jackson sought to interest religious denominations in establishing church schools and missions throughout the territory. Jackson was instrumental in establishing the Moravians in southwest Alaska, the Catholics on the Yukon, the Episcopalians at Point Hope, the Presbyterians in Barrow, the Congregationalists at Wales, and the Friends at Kotzebue. In 1905 Congress combined the educational programs for Alaska Natives with those for American Indians in a law stating the Alaska students would have the right to enroll in any of the boarding schools established for American Indian children. The government gradually discontinued supporting church-run schools and began constructing village-based day schools. In 1931 administration of the education programs passed from the Bureau of Education to the Bureau of Indian Affairs within the Department of Interior. From this time until World War II availability of grade school education was still limited, and secondary education was almost nonexistent for the Native population.

After the war, government services in Alaska accelerated rapidly. In 1955 health care was shifted from the Bureau of Indian Affairs to a newly created health authority within the U.S. Public Health Service. This agency was created because of the dire health status of the Native people, who were plagued by epidemically high rates of tuberculosis. The introduction of antituberculosis medication together with a vigorous medical program brought the disease under control rapidly. By the late 1950s and early 1960s the population was increasing at a dramatic rate; during the 1950–1960 decade the population increased 27% (4). This population explosion greatly affected the resources of the educational system in the late 1960s and in the 1970s.

The educational system developed rapidly from 1950 to 1975. Day schools were built in villages and teachers encouraged students to go on to high school. To do so meant that the majority of students had to leave their home communities and reside at Mt. Edgecumbe School at Sitka, in southeast Alaska. As the demand for places in school increased, students were sent further away to schools within the larger Bureau of Indian Affairs system. In the late 1960s students from Alaska were placed in boarding schools at Chemawa, Oregon, and at Chilaco, Oklahoma.

Statehood was granted Alaska in 1959. Within a decade significant changes were made in the system of secondary education. Unlike most reservation Indians, the Alaska Native peoples had full equality under the Alaska constitution. The state of Alaska was thus responsible for providing educational programs for Native peoples just as it provided them for other residents. The Bureau of Indian Affairs programs were gradually transferred to the state of Alaska. Although there is still a dual educational system, the state has increasingly assumed the leadership in establishing programs and policy. The state moved vigorously into its own version of boarding school education. All high school students remained in Alaska, and secondary education was provided through a network of boarding programs both rural and urban.

During the decade from 1965 to 1975 new schools were built. Existing school districts, like Anchorage, developed programs for Native students within urban high schools. Students were kept in Alaska but were in a hodgepodge of federal, state, and local jurisdictions, which had limited coordination. Students, especially troubled students, often rode a circuit from one high school to another. Many rural boarding programs were located in rural transitional towns such as Bethel or Nome. Because these towns were in transition from villages to cities (5–7), their social climate generated problems for many adolescents. The urban boarding programs were more stable, but the urban schools were almost universally unprepared for large numbers of Native students. In addition, the private boarding homes were poorly screened and poorly supervised by state agencies. Thus, although the students had been separated from the Bureau of Indian Affairs secondary school system, the learning environment had not really improved.

In 1971 Congress passed the Alaska Native Claims Settlement Act (8). The act extinguished the Native claims against the federal government with a cash and land payment to the Native groups. Regional Native corporations were organized to handle the settlement. The Claims Settlement Act focused on a regional entity for community organization and ultimately provided administrative structures that could be used for institutional change (9).

THE HOOTCH CASE

Molly Hootch was an Eskimo from Emmonok, a large village at the mouth of the Yukon River near the Bering Sea. In order to attend secondary school, Molly Hootch, as well as the other 3,000 Alaska Native school-age children, would have had to leave her home and family and travel a substantial distance to attend high school. The case was based on a constitutional argument that stated that the constitution of Alaska guaranteed public school as a "fundamental right to all Alaskan children, and further as a right it must be provided on equal terms to all children." The case was lost in the superior court in 1974 and was appealed immediately to the Alaska Supreme Court. The superior court's decision acknowledged that the state was obliged to make educational opportunities available to students and stated that it did so through

the state boarding programs. Availability of high school programs in every village was not regarded as a basic right and, further, was considered financially prohibitive.

In preparing their appeal to the Alaska Supreme Court the attorneys for the villagers asked the Alaska Psychiatric Association to detail the psychosocial effects of boarding school education on the children and their parents. Mental health professionals and educators had for years seen that the educational system resulted in dropouts, suicide attempts, antisocial acting out, alcohol problems, identity diffusion, poor vocational planning, and only a small number of students going on to advanced training institutions.

In the amicus brief a general statement was made regarding adolescent development in the context of family and cultural life. Given the cross-cultural problems faced by Alaska Native children, the maintenance of parent-child contact was viewed as crucial through the teenage years. The brief cited studies concerning the effects of boarding schools on Alaska Native students. Kraus (10) found a definite over-representation of younger people in suicide attempts and suicide. He concluded that school separation was a definite factor in the suicide problem. Kleinfeld (11, 12) studied Eskimo students in four different school environments: an urban boarding home program, a rural boarding home program, a rural transitional town boarding school, and a village-based church-affiliated school. Using a variety of measures, Kleinfeld found that rates of emotional disturbance were high in all of the schools but particularly so in the two rural town programs and the urban boarding school. The amicus brief concluded by supporting the position of the plaintiffs against the State Department of Education and recommending to the state supreme court that secondary education be moved to the villages.

The suit was settled in 1976 through a negotiated agreement in favor of the village children. The combination of the original constitutional issues and the behavioral science data was a crucial element in the court's findings, as shown by its decree:

> Members of the plaintiff class enrolled in the boarding program have experienced accelerated drop-out rates, psychological and social problems, including disruption of family life and loss of identity, and failure to live up to educational potential. Studies of drop-out rates indicate far higher rates among Native children attending boarding programs than among Native children residing at home while attending secondary school. . . . Also the sending of Native children to secondary schools outside of their villages has had harmful effects on village cultural and family life and on the student's relationship to each. When the student who attends a boarding program returns to his village in the summer, he finds it difficult to readjust to village life because of the increasing differences between himself and the other members of his village. He is unable to fully identify with either the town or village way of life. Such students are in the process of becoming what anthropologists term "marginal. . . ." (1)

The language cited reflects the court's consideration of the views expressed in the amicus brief. *Indian Family Defense* (13), published by the Association on American Indian Affairs, cited the conclusion of the case as a historic victory affecting Native American children and looked for ramifications that would affect Native American boarding programs in other areas of the United States.

DISCUSSION

The outcome of the Hootch case was based on the state's recognition of the right of children to be educated in their home villages. The court case was able to focus in one forum both the constitutional and human outcome arguments. It is interesting that most of the same issues have long been known and debated in professional circles and within government. Krush and associates (14) coined the term "psychological nomadism" to describe the objectless, anomic existence characteristic of the boarding school experience. Hammerschlag and associates (15) described one southwestern boarding school and concluded that the educational system contributed to the sense of powerlessness and government dependency so prevalent among Indian peoples. Shore and Levy[1] described high dropout rates in a northwest Indian boarding school. They reported a dropout rate of 26% in 1970–1971. In another report, Shore (16) described the difficulties and frustrations involved in providing mental health consultation in the same boarding school. In an editorial in the *American Journal of Psychiatry* (17) Beiser pointed to the problems of Indian boarding schools and called for changes in programs that separated children from their parents. Based on data gathered from a boarding school project in the southwest (18), the editorial suggested improvements in the schools that involved improving staff/pupil ratios by employing more Indian persons in dormitories and as instructional aides. The ill effects of Indian boarding schools were summarized in a publication of the Association on American Indian Affairs titled *The Destruction of American Indian Families* (19).

From the perspective of the physicians and behavioral scientists involved in the amicus brief the outcome was very positive. As indicated above, clinicians had for years seen a wide variety of problems that attested to the negative influence of the educational system on both Alaska Native and Indian children. In Alaska it had become painfully clear that the system of education was not improving after the state moved to

[1] J. Shore, J. Levy: Dropout epidemic at an Indian boarding school. Department of Psychiatry, University of Oregon Health Sciences Center, 1975 (processed)

replace the Bureau of Indian Affairs as the dominant system in education.

The amicus brief offered a vehicle by which scientific evidence directly effected a major social change. An implied hypothesis in this report is that the willingness to be involved in court proceedings of this type is an important consideration for psychiatrists interested in social change, decreased psychiatric morbidity, and in primary prevention. Psychiatry's involvement in social issues is certainly widely debated today within the profession. We consider the material described in this paper as a positive example of psychiatric participation in an attempt to solve an important social problem affecting the next generation of Alaska Native students.

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Treatment of Depression in the Medically Ill Elderly with Methylphenidate

BY WAYNE KATON, M.D., AND MURRAY RASKIND, M.D.

Three depressed geriatric patients had a marked therapeutic response to the psychostimulant drug methylphenidate. These patients either had been unable to tolerate tricyclic antidepressants or had a medical illness that contraindicated tricyclic therapy. The lack of adverse effects in our elderly patients and methylphenidate's effectiveness as an antidepressant were consistent with the findings of other investigators. These results suggest that psychostimulants deserve further evaluation as antidepressant agents in the geriatric population.

Although tricyclic antidepressants are the drugs of choice in the psychopharmacologic management of depression (1), their adverse anticholinergic and cardiovascular effects can limit their usefulness in elderly depressed patients, particularly those suffering from a concomitant serious medical illness. Given the high prevalence of depression in the elderly (2), finding an antidepressant regimen for geriatric patients who cannot tolerate tricyclics is a significant problem. One seldom considered alternative psychopharmacologic agent is methylphenidate, one of the psychostimulant drugs. Psychostimulants were briefly used as antidepressants before the introduction of tricyclics, but legitimate questions concerning long-term efficacy and potential for drug abuse contributed to a marked decrease in their use as antidepressants. On the other hand, the psychostimulants are effective and widely used in child psychiatry in the long-term management of the minimal brain dysfunction syndrome (3). In the pediatric population, adverse effects, tolerance, and drug abuse have not been significant problems (4, 5).

The usefulness and safety of psychostimulants in the pediatric population suggest that age may be an important variable in the effectiveness of this class of drug. In fact, several reports suggest that psychostimulants may have a place in the psychopharmacology of another special age group, the geriatric population. In a placebo-controlled but nonblind study (6), methylphenidate was significantly superior to placebo in the treatment of 54 elderly patients suffering from depression. Two studies have demonstrated methylphenidate to be effective in treating secondary depression in patients in whom the affective signs and symptoms were superimposed on an underlying dementia (chronic organic brain syndrome). In the first study (7), depressed dementia patients responded better to methylphenidate than they did to the tricyclic antidepressant protriptyline. In the second (8), methylphenidate was compared to placebo in the treatment of a large group of depressed elderly patients with underlying dementia. Methylphenidate was clearly superior to placebo in this double-blind 8-week study, and the methylphenidate group was remarkably free of significant adverse effects. Faced with the dilemma of instituting an effective therapeutic regimen for elderly depressed patients who were intolerant of tricyclic antidepressants, we elected to treat several of these patients with methylphenidate. Following are illustrative case reports.

CASE REPORTS

Case 1. Mr. A, an 82-year-old retired professor, had a one-year history of poor mental concentration, loss of interest in his environment, decreased energy, insomnia, episodes of tearfulness and pessimism, decreased appetite, and an insidious 7-kg weight loss. Psychometric testing and a complete neurological evaluation showed his cognitive functioning to be within normal limits. His primary physician had diagnosed depression and had prescribed amitriptyline, 50 mg/day. Unfortunately, acute urinary retention had developed and amitriptyline treatment was discontinued, without any change in Mr. A's psychiatric status. Supportive psychotherapy by the primary physician had also failed to alleviate Mr. A's symptoms, and he was referred for psychiatric evaluation.

We confirmed the diagnosis of depression and elected to start a course of desipramine. We hoped that the relatively low anticholinergic activity of desipramine would lower the risk of urinary retention (9). Unfortunately, after the dose had been gradually increased to 75 mg/day over 2 weeks, urinary retention again developed, without improvement in Mr. A's depressive signs and symptoms. After a drug-free

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period of 10 days, we prescribed 10 mg of methylphenidate twice a day. Two days later Mr. A reported that his mood was brighter and his ability to concentrate on everyday tasks was improved. Over the subsequent month his mood, enthusiasm, energy, and appetite markedly improved and he returned to part-time work. Methylphenidate treatment was continued for another 3 months and Mr. A maintained his therapeutic gains. Urinary retention or hesitancy were not problems, and there was no suggestion of psychostimulant habituation or abuse. Methylphenidate was discontinued after the 4-month treatment course and Mr. A remained free of signs and symptoms of depression at 1-year follow-up.

Case 2. Ms. B, a 73-year-old nursing home resident, had adult onset diabetes mellitus and bilateral amputations below the knee for peripheral vascular disease. She developed saddened mood, decreased appetite, weight loss, social withdrawal, and loss of interest in her usual activities over a 3-month period. Her private physician prescribed a 4-week course of imipramine, 100 mg/day in divided doses. The imipramine regimen was associated with unpleasant tremor, constipation, nocturnal sweats, and increased dysphoria. Imipramine treatment was discontinued and amitriptyline was prescribed at 75 mg/day in divided doses. During the subsequent week Ms. B became increasingly lethargic and fell twice from her chair. Severe orthostatic hypotension was documented at the time of her falls and treament with amitriptyline was discontinued, with no evident improvement of her depression. After a 1-week drug-free interval, we began to administer methylphenidate, 10 mg twice per day. Ms. B noted an increased sense of well-being within 3 days and the nursing staff reported that she had increased energy, brightened mood, and increased participation in social activities. Over the next month Ms. B's signs and symptoms of depression resolved and she was judged to have returned to her previous level of function. Methylphenidate treatment was continued for another 2 months. During treatment Ms. B's blood pressure was stable and she did not experience any adverse effects from the methylphenidate. She was free of signs and symptoms of depression at 6-month follow-up.

Case 3. Ms. C, an 85-year-old woman, was hospitalized for treatment of a dislocated right hip suffered in a motor vehicle accident. She was withdrawn, apathetic, and somnolent. Her primary physician became increasingly concerned about her inadequate caloric intake, progressive weight loss, and refusal to participate in physical rehabilitation. After a thorough medical and neurologic workup failed to provide an explanation for her deteriorating condition, we felt that depression most likely was responsible for Ms. C's symptoms. Although both Ms. C and her husband denied that she had a history of affective disorder, they did acknowledge that she had been much less active and enthusiastic about general activities over the past 2 years, after a traumatic fall in her bathtub. Her husband stated that she was "just getting back to herself" when the motor vehicle accident occurred. During psychiatric examination Ms. C appeared lethargic, apathetic, and depressed and had psychomotor retardation and increased latency of response. She was fully oriented, her past and present memory were normal, and she interpreted proverbs well. Several times during the examination she stated that she was very tired and needed to be left alone.

Electrocardiogram revealed first-degree atrioventricular block and a right bundle branch intraventricular block. Ms. C's primary physician felt that the cardiac conduction abnormalities contraindicated the use of tricyclic antidepressants. Therefore methylphenidate was prescribed at 10 mg twice per day. Within 3 days Ms. C was eating well participating in physical activity, and sitting up most of the day. She was alert, her mood was much improved, and she showed renewed interest in her rehabilitation and discharge from the hospital. She left the hospital 10 days later to continue her convalescence in an intermediate care facility, and her improvement in mood and activity level persisted.

DISCUSSION

We have presented three cases of depressed geriatric patients who either could not tolerate tricyclic antidepressants or whose medical status was felt to contraindicate the use of tricyclics. Each of these patients appeared to have a marked therapeutic response to low-dose methylphenidate. Although positive case reports certainly do not establish the antidepressant efficacy of a drug, these three cases illustrate several aspects of treatment with methylphenidate which suggest that this drug deserves more careful scrutiny as an antidepressant agent in the geriatric population. Of note was the rapidity of the therapeutic response, in each case beginning during the first few days of treatment. Of perhaps greater importance was the absence of significant adverse effects from methylphenidate in these three patients. The same patients who either could not tolerate tricyclic antidepressants or for whom tricyclics were felt to be contraindicated easily tolerated a course of low-dose methylphenidate. We did not observe the adverse effects that have been attributed to psychostimulants (10), such as appetite suppression, hypertension, palpitations, cardiac arrhythmias, nervousness, headaches, dyskinesias, precipitation of paranoid ideation or overt psychosis, insomnia, rapid tolerance to euphoriant effects, and abuse of these agents. This absence of adverse effects may have been fortuitous or secondary to the low dose of medication, but it is consistent with the low incidence of adverse effects secondary to methylphenidate in previous geriatric studies (6–8, 11–14).

Ms. C demonstrated another property of the psychostimulants, which Hackett and Draper (11) described as particularly relevant to consultation-liaison psychiatry. This property is its usefulness in the differentiation of depression from enfeebling medical illness. A sustained improvement of mood, energy, and function with psychostimulant therapy suggests a large component of reversible depression in the medically ill.

Before the initiation of treatment with methylphenidate, the consulting psychiatrist attempted supportive psychotherapy in each of the three cases, with little benefit. However, after methylphenidate treatment had begun the patients' affects brightened and their energy levels increased, which facilitated the formation of a strong therapeutic alliance with the psychiatrist

and subsequent beneficial supportive psychotherapy. Jacobson (6) also noted that methylphenidate increased the efficacy of psychotherapy in depressed geriatric patients.

Our suggestion that methyphenidate be considered as a therapeutic approach to the patient who cannot tolerate tricyclic antidepressants is not meant to imply that other approaches do not exist. Electroconvulsive therapy is effective and reasonably safe for severely depressed geriatric patients and may even be the treatment of choice for those with clear-cut delusions or for those whose nutritional status cannot be maintained (15). On the other hand, posttreatment memory impairment can be a problem in the elderly, and the idea of electroconvulsive therapy is frequently unpalatable to either the patient or the patient's family. Also, the use of electroconvulsive therapy is often administratively difficult in the nursing home population because the patient would have to be transferred to a hospital and would require the services of an anesthesiologist. The monoamine oxidase inhibitor drugs are effective antidepressants, but the risk of serious adverse effects has led several authorities (16) to discourage the use of monoamine oxidase inhibitors in the elderly patient.

Our data and those of earlier investigators suggest that methylphenidate is a reasonable candidate for closer scrutiny as an antidepressant drug for the elderly depressed patient. Well-designed clinical studies would clarify the role of methylphenidate in this important patient group, especially in those who cannot tolerate tricyclic antidepressants.

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Traumatic Neurosis in the Etiology of Alcoholism: Viet Nam Combat and Other Trauma

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Traumatic neurosis from Viet Nam combat or other sources includes many symptoms that can be effectively self-medicated with alcohol, at least initially. These symptoms include chronic anxiety and restlessness, insomnia, and recurrent frightening dreams. Repeated self-medication with alcohol results in tolerance and a need to increase the amount consumed. Attempts to decrease consumption or to abstain can lead to alcohol withdrawal symptoms similar to and exacerbating the initial symptoms of traumatic neurosis. Continuing alcohol use, with the establishment of a vicious circle, can follow. The authors present three case examples. They note that treatment of alcoholism under the conditions described requires specific attention to the underlying traumatic neurosis.

> Wine drunk with an equal quantity of water puts away anxiety and terrors.
>
> —Hippocrates, *Aphorisms*

The perspective of a Veterans Administration chemical abuse program has allowed us to observe a relationship between traumatic neurosis and alcoholism in some patients. This relationship is seen in Viet Nam combat-traumatized veterans and in other combat veterans, and may well occur in association with traumatic neurosis from noncombat sources. A variety of postcombat syndromes have been described (1, 2); the clinical picture is often not "pure." Of particular note in this paper are the symptoms associated with classical traumatic war neurosis, i.e., chronic and at least moderately severe anxiety; recurrent frightening dreams, usually about the traumatic event or events; and insomnia, irritability, agitation, depression, and somatic symptoms and preoccupations. Often the insomnia is secondary to the feared dreams. Various abusable substances can be used to suppress these symptoms, e.g., minor tranquilizers, barbiturates, and alcohol. In this report we focus on alcohol, a substance readily available and at least implicitly encouraged among armed forces personnel in the United States (3).

The acute administration of alcohol relieves many of the symptoms of classical traumatic neurosis. It is effective in inducing sleep and suppressing anxiety and can ease muscle tension and sometimes depression, irritability, and agitation. In most patients alcohol initially suppresses REM sleep (4–6), with which most posttraumatic terrifying dreams are associated (7). (It apparently does not provide such suppression in all patients [8].) Some of these terrifying dreams are probably stage IV night terrors; the effect of alcohol on them is not firmly established, but alcohol may have a suppressant effect similar to that of diazepam (7). The repeated drinking of alcohol leads to tolerance and the need to increase the amount consumed. Even then, with chronic alcohol use disturbing dreams can break through the suppressing effects (7). In addition, the abrupt cessation of significant drinking leads to its own problems. These are the well-known symptoms of the alcohol withdrawal syndrome and include many symptoms similar to those of traumatic neurosis, e.g., anxiety, motor restlessness, agitation, insomnia, and an increase in frightening dreams (4–6, 8). Thus, what can begin as innocent self-medication for symptoms of traumatic neurosis can lead to a vicious circle. Repetitive symptom reduction by means of alcohol leads to tolerance and an increase in alcohol consumption to maintain symptom reduction, which leads to problems from the alcohol use itself (e.g., hangovers, gastritis, marital difficulties, and arrests for driving while intoxicated) and attempts to reduce or abstain from alcohol consumption. This leads to the alcohol withdrawal syndrome, with an exacerbation of many of the initial traumatic neurosis symptoms; this stimulates further drinking, closing the vicious circle.

It is important to note here that the initial use of alcohol is effective self-medication for many posttraumatic symptoms. After chronic use, attempts to stop the self-medication lead to exacerbation of the initial symptoms. This use of alcohol is different in de-

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The authors wish to thank the many veterans who through their difficulties have been our teachers.

gree, if not also in kind, from the more frequent use of alcohol to relieve general psychological dysphoria; in the latter case the cessation or decrease of chronic alcohol use does not lead to withdrawal symptoms that are as synergistic with the initial psychological difficulties. In addition, alcoholism secondary to traumatic neurosis calls for particular therapeutic strategies with these alcoholic patients.

CASE REPORTS

The patients described here were all treated in our intensive residential alcohol rehabilitation program. The first patient had a classic traumatic neurosis; the second patient showed symptoms of traumatic neurosis mixed with other postcombat symptoms (2); and the third patient had an attenuated traumatic neurosis that had persisted for over 30 years.

Case 1. Mr. A, a 30-year-old veteran, was admitted because of alcoholism associated with interpersonal, work, and health problems. Although well detoxified on admission, he was extremely anxious, restless, "couldn't remember" much of his background or experience in Viet Nam, and either kept considerably to himself or sought staff contact for a variety of physical and psychological complaints. He slept very poorly and had recurrent terrifying dreams.

Mr. A had been adopted as an infant, and his natural parents were unknown. His adoptive parents were free of alcoholism and had no history of psychiatric treatment. He reported that he drank during high school and used marijuana but that this had not created problems; his family corroborated this information. He entered the army at age 19 and was sent to Viet Nam as a foot soldier. While patrolling and preparing to settle for the night, his patrol of 27 members was ambushed by machine-gun fire and all but a few of his group were killed in a matter of seconds. No one who was hit survived. He and a few others at the end of the line of soldiers were not hit. He felt extremely helpless during this catastrophe. He was subsequently very "uptight" and began to be awakened regularly by terrifying dreams in which he relived the ambush. He began to try to go to bed later and later and used drugs and alcohol more extensively; he found they relaxed him and helped him sleep. After discharge from the service he ceased using most drugs except alcohol, but alcohol began to cause him progressively more problems. His admission to our unit was his second treatment program for alcoholism.

Mr. A's treatment with us included thrice-weekly psychotherapy using support and hypnotherapy focusing on relaxation, recall of the traumatic event, and some catharsis. As often occurs, the combat trauma was found to be related to prior experiences, including early childhood experiences of being helpless and overwhelmed; these experiences were also examined. Hydroxyzine, 50 mg three times a day, was used to help decrease the patient's extreme level of anxiety and marked resistance to exploring the traumatic events. With 2 months of treatment the patient's severe anxiety level abated considerably, and he slept well without frightening dreams. At discharge he was taking disulfiram and hydroxyzine and felt confident that he could manage without alcohol.

At follow-up a month later Mr. A was doing well, was sober, and, as his father reported, was "calmer than he's been in many years." His sleep continued to be very much improved, although he had had at least one night of disturbing dreams.

Case 2. Mr. B, a 29-year-old veteran, was admitted for treatment of alcoholism after prior detoxification in another hospital. On admission he was depressed, had fears of attack and death, was concerned over losing control of his anger, and described repetitive frightening dreams showing the faces of two Vietnamese he had killed. He described himself as "a small town boy" jolted by his military experience.

Mr. B's family background showed no evidence of alcoholism or psychiatric problems. He was in the military from ages 18 to 20 and began drinking significant amounts of alcohol toward the end of that time. He felt that drinking represented an attempt to cope with his fears of imminent attack and death, sorrow over the loss of friends, rage at the Vietnamese enemy, and guilt over his killing others. He said that he had problems from alcohol within his first year of drinking. He acknowledged experimenting with other substances, but without difficulties. Before his treatment with us he had been detoxified from alcohol in 1974 and 1975.

Mr. B's treatment on our unit included short-term individual and group psychotherapy with emphasis on exploration, catharsis, and reintegration of his Viet Nam experiences. By discharge he was much improved and was free of frightening dreams. No follow-up data were available.

Case 3. Mr. C, a 59-year-old World War II fighter pilot, was intoxicated when admitted for his first treatment for alcoholism. Following detoxification he was anxious, restless, dejected, and complained of poor sleep with recurrent frightening dreams.

Mr. C's history showed that his father had had problems with alcohol beginning when the patient was a teenager. While in the service the patient was shot down over Japan shortly before the end of the war and was beaten by peasants before being taken into custody by the local police. From that time onward he had a recurrent dream that awakened him in terror in a cold sweat just at the point of being shot down. He said he used alcohol to sleep, to avoid dreaming, and "to settle my nerves." Although alcohol did not fully stop the dreams, when he tried to abstain from drinking the dreams became worse and he lost more sleep.

Mr. C's treatment included hypnotherapy with abreaction of the traumatic event after abreaction of earlier traumatic life experiences related to the combat trauma. He progressively slept better and soon slept through the night without frightening dreams. His overall condition in spite of its long history was much improved at discharge. On follow-up several weeks later his wife noted many ways in which he had changed; he was sociable, patient, had a sense of humor, was relaxed, and slept well. He was maintaining sobriety.

DISCUSSION

We do not wish to imply that other factors may not also have had important etiological significance in these cases of alcoholism. However, from our evaluation the traumatic neurosis-type symptoms were crucial and precipitating causes, and diligent attention needed to be directed to them to satisfactorily treat the alcoholism. Whether or not we have obtained enduring and significant help for the patients' posttraumatic

symptoms or for the alcoholism remains to be seen, but it appears to us that a treatment approach for such cases of alcoholism which ignores the historical development will have less chance of success. The possibility remains that alcoholism which begins largely secondary to a traumatic experience might persist after the traumatic neurosis is adequately treated. This is a researchable question.

The relationship between traumatic neurosis and alcoholism appears not to have been previously recorded in the psychiatric literature. Our computer searches of the literature for the past 10 years failed to find any material on this subject, as did an examination of older war-related literature (but see hints in reference 1, p. 413). It is hoped that our drawing attention to this relationship may encourage others to carefully look for it, provide appropriate treatment, and perhaps further study this area.

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A Multi-Tiered Screening System for the Least Restrictive Setting

BY RICHARD H. ALLEN, DR.P.H., MAXINE WEINMAN, DR.P.H., RONALD LORIMOR, PH.D., AND JAMES L. CLAGHORN, M.D.

The authors evaluated the placement process in a large psychiatric facility with both inpatient and outpatient services. They examined 71 inpatient and 90 outpatient new admissions using standardized tests of psychopathology and sociodemographic status. After correcting for response bias, the authors found, in contrast to other studies, that the level of morbidity in the inpatient service was significantly different from that in the outpatient unit. The measures of sociodemographic data indicate that inpatients were more dependent on social support systems and that this finding was not influenced by response bias.

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A psychiatrist's decision to hospitalize a patient is a profound moment in that patient's life. Apparent immediate medical desirability notwithstanding, the patient will most assuredly experience the stigmatizing effects of psychiatric hospitalization. In contrast to outpatient placement, such an event can compromise a patient's effectiveness in securing employment, joining a union, or entering a trade or professional school. For these reasons, the process for hospitalization should be carefully designed to ensure that inpatient placement is essential to the total well-being of the patient. To that end, a step has been interposed at our facility before hospitalization. Our facility requires that all patients who are hospitalized must have been interviewed first in an outpatient setting. Those patients who might be treated effectively in an outpatient setting are then so assigned.

Several authors (1, 2) have posited that precise criteria should be established for psychiatric inpatient placement because standards for hospitalization are seldom explicit. Such criteria for admission would logically include severity of illness as well as usefulness of programs unique to the inpatient setting for the patient. Measurement of the severity of illness, however,

is complicated by the tendency for patients to exaggerate or minimize their reports of symptoms (3). This creates a diagnostic dilemma for the clinician and an interpretation hazard for the psychometrist. The extent to which this distortion is systematic lends itself to examination focusing particularly on the way it blurs discrimination between groups and interferes with appropriate placement.

In this investigation we examined the placement process of a large psychiatric institute with both inpatient and outpatient facilities. The purpose of the study was to show whether placement was justified by differences in standardized tests of personal and social functioning. Additionally, we examined the hypothesis that distortions in symptom reporting do occur and that this distortion is a function of response bias. We have defined response bias as the tendency to minimize or exaggerate the presence of symptoms; therefore, we analyzed the influence of response bias in interpreting the differences.

METHOD

Setting

The Texas Research Institute of Mental Sciences is the research and training arm of the Texas Department of Mental Health and Mental Retardation. In order to satisfy its mission, the Texas Research Institute has established a broad-based service component that includes an inpatient unit and one of the largest outpatient departments in Texas. A patient's entry into the adult service involves evaluation at each stage of a three-tiered system that graduates from a paraprofessional level through the front-line therapist to the doctoral-level supervisor. Medical responsibility for each case is assigned to the psychiatrist attached to the unit.

The initial contact for services is made with a caseworker in the Information and Referral Service. The paraprofessional staff member at this critical juncture is familiar with criteria regarding appropriateness for admission to each of the adult service units. Following this contact, an appointment is made for the patient to be interviewed by a primary therapist from the adult outpatient unit. A psychiatrist reviews the information obtained by the primary therapist and, if appropriate, also interviews the patient. The decision as to placement of the patient is then made.

This course of events is followed, even for people who obviously require hospitalization, to comply with the state mental health commissioner's rule regarding outpatient evaluation for least restrictive setting. The rule was made in response to an increasing social conscience within the state department of mental health and was influenced by court precedents. It contains minimum guidelines for hospitalization that are used by the outpatient personnel in the placement decision process. These guidelines are as follows:

1. Both mental impairment or a reasonable presumption of mental impairment and social impairment may justify admission.
2. Evidence of the following *must be* documented: a) ideational or behavioral indications of dangerousness to self or others, b) need for planned psychiatric, medical, social, or psychological evaluation or treatment; special drug therapy; treatment requiring hospitalization or continuous skilled observation.
3. Evidence of the following *may be* present: a) impairment of social, familial, or occupational functioning of sufficient severity to require the structure or safety of inpatient services, b) failure of outpatient or extended care management, c) inability to meet one's own basic life and health needs, d) behavior "intolerable" to patient, family, or community, e) need to interrupt deleterious psychosocial interactions.

If a crisis exists, the entire review sequence is condensed into a single day. A request for emergency treatment is not, of itself, a sufficient criterion for crisis intervention. The working definition of "crisis" is based on the imminence of a bad experience for the patient or others. If harm may come within a 72-hour period, a crisis exists and immediate steps are taken. A primary therapist conducts a complete interview to answer the questions remaining from the briefer screening examination. The supervising psychiatrist then talks with the patient and conjointly with the therapist determines the appropriate disposition. On the average, a third of the crisis patients are hospitalized, a third receive intensive outpatient crisis intervention, and a third are referred to another clinic.

Subjects

The subjects for this study were 71 inpatient and 90 outpatient new admissions to the Texas Research Institute of Mental Sciences. Subjects were entered into the study sequentially. All patients who were able to respond to questions were asked to participate. In neither group was participation presented as part of a treatment program, and it was stated on the consent form that participation was voluntary. All of the subjects signed the consent form after the nature of the study had been explained.

The subjects displayed the spectrum of psychiatric problems seen at our facility, from severe psychoses to phobias and marital discord. Treatment is provided in the outpatient clinics according to the nature of the problems presented. The principal objective of the inpatient service is to stabilize the patient and return him or her to appropriate outpatient care as soon as possible. The length of stay is usually 20 to 25 days.

Instruments

The instruments in the study from which data were selected were the Denver Community Mental Health Questionnaire (4), a measure of effective level of personal and social functioning; the Mini-Mult (5), an ab-

TABLE 1
Unadjusted Means and Means Adjusted for L Scale for MMPI and Taylor Manifest Anxiety Scale Scores of 71 Inpatients and 90 Outpatients

Scale	Unadjusted Mean Score			Adjusted Mean Scores		
	Inpatients	Outpatients	Significance[a]	Inpatients	Outpatients	Significance[a]
MMPI						
Hypochondriasis	19.2	16.7	p<.02	19.4	16.5	p<.01
Depression	30.7	28.4	p<.06	31.1	28.0	p<.01
Hysteria	27.2	26.3	n.s.	27.5	26.0	n.s.
Personality disorder	29.6	28.1	p<.06	29.8	27.9	p<.01
Paranoia	14.8	14.1	n.s.	15.0	13.9	n.s.
Psychasthenia	35.2	33.7	n.s.	35.6	33.4	n.s.
Schizophrenia	38.2	33.6	p<.01	38.7	33.2	p<.001
Hypomania	21.8	19.7	p<.001	21.9	19.6	p<.0005
Taylor Manifest Anxiety Scale	27.0	25.8	n.s.	27.8	25.0	p<.09

[a]By two-tailed t test.

breviated version of the MMPI; and the Taylor Manifest Anxiety Scale (6), a measure of proneness to express anxiety.

Statistical Methods

A two-tailed t test was run to measure differences between inpatients and outpatients on each of the scales. To determine whether these differences would remain once response bias was controlled for, an analysis of covariance was performed using the lie (L) scale of the MMPI. The L scale items represent minor faults or foibles to which most people would readily admit and was designed as a correction factor for bias in socially desirable response sets. Burish and Houston (7) reported favorable construct validity for the L scale as a measure of defensive denial. "Defensive denial" is defined here as the unwillingness to admit minor faults or foibles. Following the analysis of covariance, the t tests were computed on the adjusted means and their values were compared with the unadjusted t values.

RESULTS

The first phase of this investigation was the determination of whether social support system differences were evident in inpatients compared with outpatients. On the basis of the hypothesis suggested by Mendel and Rapport (8) that social support resources are critical factors influencing the decision to hospitalize, inpatients were expected to have less access to these resources than did outpatients.

A t test analysis of social functioning compared the inpatient group with the outpatients on the scales of the Denver questionnaire. Differences that achieved or approached significance were on 1) the public system dependency scale (p<.001), which showed that inpatients required more support from social agencies, 2) the productivity scale, a measure of employment and training (p<.07), which showed that inpatients were less active, 3) the interpersonal isolation from family scale (p<.06), which showed that outpatients had less family contact, and 4) the drug use scale (p<.001), which showed that inpatients abused hard and soft drugs more than outpatients did.

A t test analysis was also performed using the scales of the MMPI and the Taylor anxiety scale. Table 1 displays the results of this analysis. As can be seen, those scales with significant or near-significant differences were schizophrenia, hypomania, personality disorder, hypochondriasis, and depression. In addition, the L scale of the MMPI showed a significant difference. In each of these scales the inpatients rated a higher pathology score.

In order to control for possible contamination by response bias, analysis of covariance was performed on each of the MMPI scales and the Taylor scale using the MMPI L scale as the covariate. As can be seen in table 1, in each case the introduction of the L scale increased the difference between the inpatient and outpatient means and materially reduced the probability level.

A similar analysis of covariance was performed on the Denver questionnaire social functioning scales. In this case, no consistent or significant differences were observed following the introduction of the L scale into the equation.

CONCLUSIONS

The process of review for least restrictive setting described above appears to be a valid mechanism for accurate placement in an inpatient setting or an outpatient setting according to psychopathological and sociodemographic differences. Standardized testing suggests real differences in social support systems for inpatients compared with outpatients. Inpatients appear to be in the peculiar situation of being alienated from the greater society and yet dependent on it. Our results show that inpatients, in contrast to outpatients, require more support from public agencies and from their families, are less likely to be employed or in-

volved in vocational training, and are more likely to abuse drugs. In addition, given placement status, these social functioning scales do not appear to be affected by response bias. This probably reflects the inpatients' casual acknowledgment of the use of and need for a protective environment.

At first blush, the differences in pathology measured by the MMPI and the Taylor scale did not show the magnitude of significance expected when inpatients are compared with outpatients. This is consistent with the results of Dohrenwend and Crandell (3), which showed that outpatients scored worse than inpatients in terms of symptom disclosure. After correcting for response bias in our study, however, a shift in magnitude was revealed. The mean difference between inpatients and outpatients was increased in each of the eight MMPI scales as well as the Taylor anxiety scale. These differences became significant at less than the .01 level in five of the nine scales. The shift in magnitude indicates the effects of response bias on test data. We can hypothesize several different explanations for this tendency:

1. Inpatients feel the vulnerability of being sick and wish to please or to respond as if they were healthier than they are so they will be discharged as soon as possible.
2. The disturbed inpatient does not wish to admit or cannot correctly perceive the presence or extent of his or her illness.
3. Inpatients obtain benefit from being placed in a protective environment. Inpatients may respond to test data more like outpatients because they feel safe and comfortable and because their disorder appears less unusual given their new surroundings.

Whatever the cause, it is clear that response bias plays an important role in the power of tests of pathology to discriminate between inpatients and outpatients. We do not support testing for psychopathology as an antecedent to placement without correcting for response bias when interpreting results. Standardized testing for the need for social support systems, however, does not appear to be influenced by response bias. Preplacement evaluation for these needs may be useful for case management by revealing the availability of external resources.

Our results demonstrate that standardized testing before inpatient or outpatient placement may be an unnecessary intrusion into patient and therapist time. When precise criteria are described, a sequence of evaluation by progressively higher trained clinicians succeeds in accurately placing patients in the setting most appropriate to their needs.

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CLINICAL AND RESEARCH REPORTS

This section contains 1) new research findings, including preliminary data from pilot studies, either clinical or laboratory; 2) worthwhile replication studies; 3) case reports that describe a truly new syndrome or cast new light on established ones; and 4) case reports that indicate a new therapeutic procedure of potential value or call attention to adverse effects of drugs or previously unreported complications of therapeutic interventions. Program descriptions and literature reviews cannot be printed in this section. Criteria for format are listed in "Information for Contributors" in each issue; papers that do not adhere to these criteria will be returned to the author.

Psychological Manifestations of Nonbacterial Thrombotic Endocarditis

BY THOMAS B. MACKENZIE, M.D., AND MICHAEL K. POPKIN, M.D.

It is well established that bacterial endocarditis may be heralded by an organic mental disorder (1). However, the psychological presentations of nonbacterial thrombotic endocarditis, also known as marantic endocarditis and degenerative verrucal endocarditis, appear to be unreported in the psychiatric literature. This omission may be explained by the fact that nonbacterial thrombotic endocarditis has been thought to be an incidental autopsy finding (2). Recently, however, it has been recognized as a significant cause of morbidity or death (3). Nonbacterial thrombotic endocarditis may be defined as the presence of fibrin-platelet vegetations on one or more uninflamed, nonulcerated valve leaflets (4). It is generally considered to be a hypocoaguable state (5).

We have recently encountered two patients who had an organic mental disorder that was an early feature of nonbacterial thrombotic endocarditis. These patients illustrate the psychopathology and clinical settings that should alert the psychiatrist to the possibility that the disorder might exist. The following case reports support the addition of nonbacterial thrombotic endocarditis to the psychiatrist's differential diagnosis of etiological factors in organic mental disorders.

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Case Reports

Case 1. Mr. A was a 74-year-old farmer who was admitted for evaluation of abdominal pain and a 13.6 kg weight loss. Several days before his admission he had noted the onset of left-sided pain and weakness. In the hospital a well-differentiated adenocarcinoma was discovered after a biopsy was performed on a right pleural mass. The site of the primary carcinoma was unknown, although an abdominal computerized tomography suggested fullness in the area of the pancreas. Findings from a cardiac examination were normal. With regard to his neurological deficits, a head computerized tomography was normal and an EEG showed irregular slowing bilaterally. Psychiatric consultation was sought 6 weeks after admission because Mr. A withdrew, had minimal verbalization, and refused to eat, which suggested a depressive disorder. During the psychiatric examination, a severe organic mental disorder manifested by lethargy, inattention, dysmnesia, and dysarthria was evident. His level of consciousness continued to diminish, and the patient died 50 days after admission. An autopsy showed disseminated adenocarcinoma of the pancreas, nonbacterial thrombotic endocarditis on the mitral valve, and cerebral infarcts of varying ages.

Case 2. Ms. B, a 45-year-old housewife with a 1-year history of disseminated ovarian carcinoma previously treated with melphalan and corynebacterium parvum, was admitted for surgical restaging of her tumor. At the time of admission she was alert and understood the reasons for her admission. While in occupational therapy with other oncology patients several hours later, she became amnesic for recent events and was temporally disoriented. Findings from neurologic and cardiac examinations were otherwise normal. The patient seemed unconcerned about her deficits and when informed of the reason for her hospitalization, she asked whether surgery would be postponed until she improved.

Her physicians suspected a dissociative reaction and requested a psychiatric consultation. During the interview Ms. B seemed embarrassed and stated that her "memory had been scared away." A dense anterograde amnesia and a patchy, inconsistent retrograde amnesia were evident. An EEG with nasopharyngeal leads showed mild diffuse slowing; head computerized tomography was normal; and a two-dimensional echocardiogram suggested mitral valve thickening and prolapse. Five days after the onset of amnesia, she became lethargic and nonresponsive to verbal stimuli and developed a right hemiparesis. Ms. B died 11 days after admission. An autopsy showed disseminated ovarian carcinoma, nonbacterial thrombotic endocarditis on the mitral valve, and cerebral infarcts of varying ages.

Discussion

Psychiatric consultation was sought because both patients were thought to be experiencing psychological symptoms reactive to their malignancies. However, the patients were suffering from organic mental disorders. Mr. A had a delirium that steadily progressed over the course of 1 month. In contrast, Ms. B manifested the abrupt onset of an amnestic syndrome. Autopsies showed that both patients had nonbacterial thrombotic endocarditis and secondary cerebral embolization; cerebral metastases were not present. The appearance of focal findings in Mr. A, e.g., hemiparesis, and the abrupt onset in Ms. B favor an embolic rather than a paraneoplastic etiology.

Nonbacterial thrombotic endocarditis is not a rare occurrence. An estimated 10% of cerebral emboli arise from the disorder (6), and the incidence at autopsy may be as high as 1.6% (7). Between 30% and 50% of patients with nonbacterial thrombotic endocarditis show evidence of cerebral embolization, usually within the distribution of the middle cerebral artery (6). The incidence of microemboli may be much higher.

Nonbacterial thrombotic endocarditis is usually found in persons more than 40 years old who are afflicted with a chronic illness (4). As a result, cachexia is seen in more than half of these patients (7). Eighty percent of the cases of nonbacterial thrombotic endocarditis are associated with known or occult carcinoma, most often of the pancreas, colon, and lung (4). In a majority of cases the malignancy is disseminated; however, in a few patients the tumor is operable at the time nonbacterial thrombotic endocarditis becomes clinically manifest. When associated with a malignancy, nonbacterial thrombotic endocarditis is accompanied by thrombophlebitis in 30% of the cases (8). Chronic heart disease of rheumatic or arteriosclerotic origin is the most common associated condition in patients who do not have cancer (9).

It is impossible at present to determine whether specific psychopathology is characteristic of nonbacterial thrombotic endocarditis because case reports of the disorder have been authored by nonpsychiatric physicians; consequently, details of the mental status are unreported. According to one study (10), 30% of the resultant neurological syndromes are without focal neurological signs. Although the onset of symptoms is usually abrupt, the evolution is subacute and unabating when microemboli are present. A clinical picture of delirium is typical. However, as our second case report shows, nonbacterial thrombotic endocarditis may also be present with selective cognitive impairment. The prognosis for nonbacterial thrombotic endocarditis is usually poor; however, if the tumor has not metastasized, surgical treatment may prevent further morbidity.

In the presence of a progressive or apoplectiform onset of an organic mental disorder, what factors should cause the psychiatrist to suspect nonbacterial thrombotic endocarditis? First is the existence of a malignancy in a person more than 40 years old. A stepwise progression of symptoms suggesting embolic etiology should raise the question of the presence of the disorder. Evidence of thrombophlebitis should heighten clinical suspicion. In a small percentage of cases, the appearance of or change in a cardiac murmur may suggest the existence of vegetations. Evidence of peripheral embolization increases the likelihood of nonbacterial thrombotic endocarditis. Once it is considered, echocardiography can be confirmatory. Abnormal coagulation indices, suggestive of a hypercoagulable state, also support the diagnosis. In our patients head computerized tomography did not detect cerebral embolization secondary to the nonbacterial thrombotic endocarditis. However, in both instances the EEG was diffusely abnormal, confirming the diagnosis of an organic mental disorder.

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A Preliminary Report on the Prevalence of Domestic Violence Among Psychiatric Inpatients

BY ROBIN D. POST, PH.D., ALLAN B. WILLETT, M.D., RONALD D. FRANKS, M.D., ROBERT M. HOUSE, M.D., SUSAN M. BACK, PH.D., AND MICHAEL P. WEISSBERG, M.D.

Researchers have gathered data suggesting that domestic violence has become a serious problem in our society (1). At the Yale-New Haven Hospital, Rounsaville and Weissman (2) found that battered women constituted a substantial minority of admissions to the emergency room, psychiatric, and surgical services. These authors believed that the nature and extent of the physical abuse experienced by these women might have remained undetected had staff members not been specifically instructed to ask questions about domestic violence. Hilberman and Munson (3) found that half of the women who were referred for psychiatric evaluation by the medical staff of a rural health clinic reported being victims of domestic violence. Stark and associates (4) criticized physicians and mental health workers for failing to recognize evidence of spouse abuse among their patients and failing to intervene appropriately.

As part of a research project on the adult inpatient units of Colorado Psychiatric Hospital, we designed a structured interview to elicit information about patients' histories of physical aggression. The project simultaneously served teaching, research, and clinical purposes. Our research objectives were to determine the prevalence of domestic violence in a population of psychiatric inpatients and to discover how the personality characteristics and life experiences of victims and of perpetrators of domestic violence differed from each other and from patients without a history of domestic violence. Our clinical objective was to ensure that staff routinely question all patients about domestic violence. Our training objective was to help therapists and other hospital staff to recognize patients with problems of domestic violence and provide effective treatment.

Method

All patients admitted to the two adult inpatient units of Colorado Psychiatric Hospital between August and mid December 1979 were asked to give informed consent and participate in the interview except those patients who remained too disorganized to provide a reliable history or too agitated to handle a one-hour interview. Four research interviewers gave the structured standardized interview, which included questions about incidents of spouse battering and other acts of violence. The interviewers focused on the nature of the violence, the patient's role as victim or perpetrator, the injuries sustained, and the medical and legal consequences of each incident. We will report on spouse battering only.

We defined battering as "significant physical harm inflicted by a partner including slapping, kicking, punching, biting, or attacking with a weapon." We devised 3 categories for the first 60 patient interviews: 1) batterers, 2) victims of battering, and 3) patients who reported no history of spouse battering. A clinical psychologist and a psychiatrist, each having more than 7 years of clinical experience, classified the patients. These classifications yielded initial reliability levels of 85% agreement for batterers and 90% agreement for victims. Those cases where ratings differed were resolved by discussion between raters.

Results

The preliminary results of the 60 interviews (38 women, 22 men) indicated that 29 of the patients (48%) had a history of battering within an intimate relationship. Fifty percent of the female patients (N=19) revealed that they had been battered, while 21% of the female patients (N=8) reported that they had battered their partners. In contrast, 3 male patients (14%) reported being battered, while 6 male patients (27%) had battered their partners. Six women and 1 man (12%) stated they had been both victims and perpetrators of spouse abuse. Of these 7 patients, 1 was clearly a batterer and was beaten by her partner only in self-defense, 1 engaged in battering to protect himself, and 4 alternated as victims or perpetrators in different battering episodes. In the case of the seventh patient, mutual provocation seemed to occur regularly and the partners battered each other. The average age of both the battered patients and the nonbattered patients was 33.7 years, compared with the batterers' average age of 39.0 years. These averages did not differ statistically. Both groups of patients averaged 12½ years of school attendance.

Seventeen of the battered patients (77%) had experienced several abusive episodes. Six victims (27%)

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called the police and 8 victims (36%) sought medical attention for their injuries at least once. The victims reported a variety of injuries; the most common were facial injuries, black eyes, bloody noses, split lips, and contusions.

Few of the batterers reported that they had inflicted injuries; only 2 batterers (14%) stated that their victims had sought medical attention. While the batterers may have engaged in milder abuse than that experienced by the battered patients, the data may also reflect a tendency on the part of the batterers to minimize the damage they inflicted. Six of the batterers (43%) reported several abusive episodes. Six of the batterers (43%) also stated that the police had been called.

Discussion

Based on these patient interviews, spouse battering emerged as a major problem in our inpatient psychiatric population. Nearly half of the patients interviewed had been involved in violent episodes with partners and exactly half of the women said they had been battered by partners. As other researchers (5, 6) have previously reported, women appear to have been battered far more frequently than men.

In an earlier study using retrospective data from patient charts ("A Comparison of Battered and Non-Battered Female Psychiatric Patients," to be presented at the American Psychological Association annual meeting, Sept. 1–5, 1980) Back and associates found that therapists had noted a history of spouse abuse for 25% of the female patients sampled. At that time therapists had not been specifically instructed to ask patients about domestic violence. When our interviewers asked direct questions about domestic violence the estimated prevalence of battering among women doubled. This finding supports Rounsaville and Weissman's contention (2) that patients do not readily volunteer information about the violence they have experienced. Our technique of asking patients direct questions about violence in their lives proved highly effective. We were impressed, as were Rounsaville and Weissman (2), with the patients' willingness to talk about problems of spouse abuse. The reported prevalence of batterers in our sample may be an underestimate because of the exclusion of patients who were significantly agitated and/or disorganized. Taintor (7) has suggested that an increased risk of assaultive acts is associated with increasing severity of psychiatric symptoms. It seems that avoidance of knowledge about spouse abuse in psychiatric screening has been more a function of the interviewer's failure to ask about it than the patient's reluctance to talk about such problems.

This research experience has led us to establish two priorities for training in our institution. One priority is to teach therapists to talk effectively with patients about violent experiences. The other is to incorporate questions about domestic violence into psychiatric screening interviews to ensure that patients have the opportunity to reveal their concerns about violence in their lives.

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Correlation of Cerebrospinal Fluid Lactate with Age

BY JEROME YESAVAGE, M.D., AND PHILIP A. BERGER, M.D.

Some investigators have suggested that part of the intellectual decline that accompanies aging is due to impaired metabolism of glucose, which in turn results in reduced production of energy-rich compounds that support neuronal function (1, 2). Cerebrospinal fluid lactate levels may provide a measure of cerebral glucose metabolism.

Lactate is mainly derived from glycolysis and removed by oxidative respiration. Increased CSF lactate concentration, therefore, may be due to increased glycolysis, decreased respiration, or both. Lactate crosses the blood-brain barrier slowly, and although it has been shown in animals that lactate concentrations in brain correlate closely with those in CSF, neither brain nor CSF lactate concentrations correlate with sagittal blood lactate (3). Furthermore, it has been shown that lactate concentrations in lumbar and cisternal CSF are correlated even when absolute values are abnormal (4).

Elevated CSF lactate levels are a nonspecific marker of cerebral pathology that may also reflect impaired glucolytic metabolism. Abnormal CSF lactate concentrations have been found in a number of neurological diseases; cerebrovascular accident, meningitis, encephalitis, and intracranial hemorrhage have been associated with CSF levels of lactate two to three times normal concentrations (5). In addition, studies of patients with Alzheimer's disease, a common presenile dementia, have linked elevated lactate concentrations (1.72 mEq/liter) and increased lactate to pyruvate ratios with decreased neurotransmitter turnover and impaired intellectual function (6). However, one other study (5) of a population of patients with a wide range of disease showed that CSF lactate correlated with CSF glucose and that both levels varied with age up to age 50, at which time both levels plateaued. Unfortunately, this study of 177 samples of CSF glucose and lactate included 38 samples with abnormally high values for CSF glucose. Production of lactate can be stimulated in the setting of normal oxidative metabolism by glucose infusions, resulting in high serum and CSF levels of glucose. To our knowledge there have been no studies of CSF lactate in a wide age range of patients with normal fasting blood sugars and without neurological or medical disease. Therefore, the following study was undertaken to examine the CSF lactate levels in normal patients.

Method

The population studied included 41 volunteers with a wide age range (mean age, 45.9; range, 22–70) who were engaged in research on cognitive function and in studies designed to provide normative data for spinal fluid studies. All volunteers were men functioning in the community in similar socioeconomic settings. All could perform detailed cognitive testing involving complex instructions, and none had acute or chronic serious medical or psychiatric illnesses. Unfortunately, because the volunteers were involved in different protocols, no uniform cognitive testing was applied to all of them, except for a basic mental status examination that documented orientation and the ability to do serial subtractions and to remember three objects over 5 minutes. Nonetheless, the volunteers passed the same physical examination and laboratory, ECG, and X-ray studies without evidence of serious defects.

Lumbar punctures were performed between 8:00 and 9:00 a.m. while the volunteers were in the lateral decubitus position and before they arose from bed; they had fasted for at least 12 hours.

Lactate concentrations were measured within 24 hours using a modification of the method developed by Marbach and Weil, which employs the oxidation of lactate to pyruvate (7).

Results

Data were analyzed using Spearman rank-order correlations corrected for ties. The volunteers' ages together with CSF lactate concentrations are presented in table 1. We obtained a correlation of .333 between lactate levels and age, which is significant at the .05 level using a two-tailed test. An examination for a correlation between CSF lactate and CSF glucose concentration was performed. The correlation was negative and nonsignificant ($r=-.152$). Glucose levels fell into a narrow range between 60 and 70 mg/100 ml.

Discussion

These results differ from results found in patients with neurological or medical diseases in that no corre-

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TABLE 1
Comparison of Age and CSF Lactate Levels in 41 Volunteers

CSF Lactate Levels (mEq/liter)	Number of Volunteers				
	Ages 20–29 years (N=11)	Ages 30–39 years (N=4)	Ages 40–49 years (N=4)	Ages 50–59 years (N=14)	Ages 60+ years (N=8)
1.9–2.0				1	2
1.7–1.8		1		3	1
1.5–1.6	4	1		4	3
1.3–1.4	4	2	1	5	2
1.1–1.2	2		3	1	
1.0	1				

lation with CSF glucose was found. This may be partially explained by the narrow range of CSF glucose values and the inclusion of patients with high CSF glucose levels in other studies. Such high glucose concentrations may increase lactate concentrations by increased metabolism of glucose to lactate by the Embden-Meyerhof pathway (4). None of our patients had elevated CSF glucose levels; hence, it is less likely that this effect would be seen.

Future studies of CSF lactate should also control for respiratory function. Although our volunteers were without overt pulmonary disease, arterial PO_2 does decrease with age and is associated with increased CSF lactate production (3). However, subjects with moderate overt pulmonary disease have been found to have normal cerebral circulatory function (8). Nonetheless, whether increased CSF lactate concentrations are related to minimal pulmonary changes associated with age or are due to metabolic disease of the brain, such changes noted in an overtly normal population are potentially important.

Interestingly, not all of our aged volunteers had high CSF lactate concentrations. There were 8 individuals over age 50 with lactate concentrations equal to or less than the mean for those under 50. Nevertheless, the correlation of lactate concentrations with age is consistent with the findings in the group with Alzheimer's disease and in the groups with neurological diseases. Further study with sophisticated cognitive testing should now be done to determine if such changes in CSF lactate concentration are associated with subtle intellectual decline due to degenerative processes that have not yet become clinically obvious.

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Training in Brief Therapy: A Survey of Psychiatric Residency Programs

BY JOHN F. CLARKIN, PH.D., ALLEN FRANCES, M.D., ZEBULON TAINTOR, M.D., AND MARIE WARBURG, M.D.

Many factors have caused the recent growth of clinical practice and research in brief psychotherapy. The mental health system is increasingly under pressure to limit costs, justify the utilization of services, and provide prompt and readily accessible care (1). Studies of the outcome of brief therapy have revealed promising and surprisingly enduring results (2). The field is also of inherent interest to clinicians, researchers, and teachers. However, the extent and manner in which brief therapy is taught have not yet been investigated.

This paper reports the findings of a survey of psychiatric residency programs conducted in 1978 by the American Association of Directors of Residency Training. The 152 respondents represented 75% of all active, approved residency training programs in psychiatry in the United States. Thirty-two percent were university affiliated and 30% were university based. A majority of programs (58%) had an eclectic theoretical orientation, 25% were psychoanalytic, and 16% were distributed across such categories as biological, humanistic, and community.

Findings

Eighty-eight percent of the programs offered a training experience in brief psychotherapy, and 78% required it. Only 22% provided their brief psychotherapy instruction as part of a block rotation specifically geared to training in brief therapy. Fifty-two percent had one service that was specifically organized to teach and deliver brief psychotherapy. Sixty-three percent of the training directors predicted that their current emphasis on brief psychotherapy would continue for the next 5 years, while 36% expected growth and only 1% believed that there would be less emphasis on brief psychotherapy.

The training programs that provided formal training in brief psychotherapy varied in their teaching methods. Supervised clinical experience was most prominent (74%), with moderate emphasis on seminars (64%) and supervised reading (60%), and little emphasis on research (10%). The supervised clinical experience usually involved more than 5 patients (60%) or from 3 to 5 patients (29%). The patients spanned a wide range including adults (97%), adolescents (93%), children (76%), families (89%), and couples (88%). The number of sessions for the brief psychotherapy usually ranged from 6 to 12 (71%), with a small percentage consisting of 12 to 30 sessions (19%) or less than 6 sessions (10%). The majority of the programs (57%) had from 2 to 3 brief therapy supervisors. Twenty-three percent had 4 or more supervisors. The supervisors often used video tapes (69%) as teaching instruments and 74% had a follow-up of the patients after the brief psychotherapy.

We inquired about instruction in several different types of brief psychotherapy. The most frequently taught models were: problem solving and anxiety suppressing (79%), crisis intervention (69%), focal or dynamic (58%), and brief family therapy (60%). Only 37% of the institutions taught brief behavior therapy. When asked which model their program taught best, 38% of the training directors selected focal therapy, 23% selected problem solving, 21% crisis intervention, 10% brief behavior therapy, and only 6% brief family therapy.

Discussion

Investigating and documenting the current practices of residency training programs in teaching brief therapy may be helpful in several ways. Data from the survey and from polling training directors about their plans will help to resolve the controversy over what should constitute the essential core of a psychiatric residency. The survey method tabulates data on national practices of training programs and allows each program to determine how it compares with the empirically derived norms. Trends in training can also be determined by comparing the results of surveys performed at regular intervals. Data that document training methods can help to correct misconceptions about program content and can correct the impression that psychiatrists do not learn how to conduct treatment during their residencies.

There are, of course, several limitations to the survey method. The quality of the data depends on the accuracy and thoroughness of the individual responses to the inquiry. It may be difficult to capture in questionnaire format a realistic reflection of the quality, spirit, and effectiveness of the training reported. Survey data describing one portion of a program can be misinterpreted if not seen in the context of the total program.

Our survey suggested that a large majority of residency directors consider training in brief therapy an

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essential part of their programs. A majority assume that the current emphasis will continue for at least the next 5 years, while a considerable number predict growing emphasis in the future. We suspect that brief psychotherapy training is expanding, although there are no previous data for comparison. Such growth would reflect increased recognition of its efficacy, the need to adapt long-term psychotherapy to forms that allow for treatment of more patients, and problems of reimbursement. The amount of training in brief psychotherapy already provided indicates the recognition it has achieved, even though this modality is not now specifically included in the *Essentials of Accredited Residencies* (3). The core curriculum requires experience in "individual psychotherapy (including some experience in long-term therapy) . . . crisis intervention . . ." but does not mention brief psychotherapy.

There is an interesting disparity in the current teaching of the various models of brief therapy. Although problem-solving and crisis models are most frequently taught, the training directors more often believe that the focal dynamic model is the one they teach best, perhaps because focal therapy has the best established theory, widest literature, and most definite technical guidelines. Problem-solving brief therapy and crisis interventions are less tied to theory, place greater emphasis on flexibility and resourcefulness, and may be inherently more difficult to teach. As brief therapy matures, the different models will probably become more clearly differentiated from each other, and their instruction will become more systematic, comprehensive, specific, and comparative.

The current organizational practice in the majority of programs is to mix brief therapy training with other training experiences rather than to concentrate it in one block of time. However, half of the programs we surveyed had a specific service to teach and perform evaluations and brief treatment. As more data become available, it should be possible to assess the advantages and disadvantages of using block rotations and specialized services in training residents in brief psychotherapy.

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Cerebrospinal Fluid Cortisol Levels in Depression and Schizophrenia

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A number of studies suggest that some patients with major depressive illness have a relatively specific abnormality in glucocorticosteroid hemeostasis. Urinary excretion of both 17-hydroxycorticosteroid and free cortisol is moderately elevated in depressed patients in comparison to controls. Plasma cortisol levels are also frequently elevated in depression, especially in the late afternoon and evening, when these patients fail to show the expected circadian decline in cortisol (1). When compared with schizophrenic patients and controls, depressed patients show an early escape from suppression of plasma cortisol following dexamethasone administration (2). The corticosteroid defect appears to be state-dependent, with return to normal responses following symptom remission; however, the altered response may characterize a biologically and clinically distinct subtype of depressive illness (3). The results of one study (2), which are in apparent agreement with urinary and plasma data, showed that depressed patients had elevated levels of cortisol in lumbar CSF (11.4 ng/ml) compared with schizophrenic patients (3.9 ng/ml). In our preliminary study reported here, we did not observe group differences in CSF cortisol levels among patients with depression, schizophrenia, and personality disorder.

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Method

To compare the cortisol levels in the CSF of patients with depression, schizophrenia, and personality dis-

orders, lumbar punctures were performed in conjunction with amine metabolite studies on 28 moderately to severely ill hospitalized patients who were diagnosed according to *DSM-III* research criteria. The mean age for the 3 men and 5 women patients selected with endogenous major depressive illness (2 unipolar and 6 bipolar) was 38 years; the mean duration of illness since their first hospitalization was 6.9 years; and the average previous psychiatric hospitalizations per patient was 2.4. The 14 schizophrenic patients, 7 men and 7 women, included 3 patients with schizoaffective illness. The mean age for this group was 26 years, the previous psychiatric hospitalizations per patient was 2.8, with a mean duration of illness of 6.3 years; and their mean score at the time of admission on the Phillips Scale for prognosis of their schizophrenia was 16.3.

A comparison group consisted of 6 men patients (mean age, 23 years) with severe personality disorder but free of major affective illness or alcoholism; they were hospitalized at the National Naval Medical Center, Bethesda, Md. All patients gave informed consent, had been medication free for 2 weeks before the study, and followed a research diet with restricted monoamines. Lumbar punctures were performed at 8:00 a.m. following 9 hours of bed rest and an overnight fast. One-half-milliliter samples of CSF were frozen at −20°C until assayed by radioimmunoassay (4).

Results

The CSF cortisol concentrations (mean±SEM) were not different for patients with depression (16±3 ng/ml, N=8), schizophrenia (20±2 ng/ml, N=14), or personality disorder (21±1 ng/ml, N=6) (F=1.3, n.s., analysis of variance). CSF cortisol values were not related to global nurses' ratings of depression or anxiety in the depressed patients or to psychosis ratings in the schizophrenic patients. Cortisol values tended to be higher in the 3 schizoaffective patients (26±4 ng/ml) than in the 11 other schizophrenic patients (18±2 ng/ml, n.s.). Cortisol values by radioimmunoassay in our patients were somewhat higher than previously published values for normal controls using fluorimetric (5) or competitive protein-binding methods (6).

Discussion

Our observation of similar CSF cortisol levels in depressed patients and neurological controls is consistent with a previous report (7). Values for depressed patients were lower than for controls in a smaller study (8). The relative increase in CSF cortisol in depressed patients reported by Carroll (2) may have been influenced by the administration of 2 mg of dexamethasone 3 days before the lumbar puncture because dexamethasone has a relatively long biological half-life of 36–72 hours (9). Thus, the differences in CSF cortisol levels in that report may in part reflect a prolonged manifestation of the differential response to dexamethasone observed in depressed and schizophrenic patients. Patient characteristics, such as chronicity or severity of illness, could also contribute to differing results. Differences in CSF cortisol concentrations between drug-free depressed patients and other groups might be most apparent in lumbar puncture samples taken in the evening, when the differences in plasma cortisol are most marked. Our observation of similar CSF cortisol levels in schizophrenic patients and controls is in agreement with previous data (5). The tendency toward higher CSF cortisol values in 3 schizoaffective patients suggests the importance of carefully discriminating such patients from other diagnostic groups in future studies of this system. Lerner and coworkers, in work now in preparation, have found similar CSF cortisol levels in depressed patients, schizophrenic patients, and healthy volunteers.

Demonstration of differing levels of cortisol in the CNS of psychiatric patients would be of interest in view of the possibility that brain corticosteroid receptors, in areas such as hippocampus or parietal cortex (10), could be involved in the regulation of cognitive and emotional behavior. Because free cortisol appears to cross the blood-CSF barrier relatively freely (5), future CSF studies should include simultaneous plasma cortisol measurement. The data in this preliminary sample warrant a more extensive study to compare the CSF cortisol levels in depressed and schizophrenic patients with those of a control group of healthy volunteers.

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Differentiating Mania from Schizophrenia by the TRH Test

BY IRL EXTEIN, M.D., A.L.C. POTTASH, M.D., MARK S. GOLD, M.D., AND DAVID M. MARTIN, M.A.

Differentiating mania from schizophrenic psychosis has important implications for treatment and has aroused much controversy in recent years (1). Recognition of the specific efficacy of lithium and neuroleptics in the acute and prophylactic treatment of bipolar affective disorder (2) and schizophrenia (3) respectively has resulted in appropriately increased emphasis on distinguishing these two disorders accurately. Despite an impressive body of genetic, biological, clinical, and pharmacological response data supporting the concept of bipolar affective disorder and schizophrenia as distinct entities (1), differentiating mania from schizophrenia in the acute clinical situation can be difficult in many cases. Patients with classic bipolar disorder can in the manic state develop paranoia and Schneiderian first-rank symptoms that are thought to be diagnostic of schizophrenia (4). Similarly, patients with schizophrenic disorder can in the acutely psychotic state manifest hyperactivity, irritability, and grandiosity suggestive of a manic state.

We report here that a biological marker may help distinguish mania from schizophrenia. The magnitude of the release of thyroid-stimulating hormone (TSH) from the pituitary after infusion of thyrotropin-releasing hormone (TRH) can help distinguish unipolar from bipolar depression (5). We report here that manic patients showed a decreased TSH response to TRH compared with psychotic schizophrenic patients. The TSH response to TRH of patients with schizophrenic psychosis was not distinguishable from that of the control patients by the TRH test.

Method

We included in this study five consecutively admitted patients who met Research Diagnostic Criteria (RDC) (6) for mania, five consecutively admitted patients who met RDC for schizophrenia, and a control group of five consecutively admitted patients with the primary diagnosis of personality disorder. The schizophrenic patients were diagnosed as undifferentiated subtype by RDC, and all were actively psychotic. Age and sex distributions for each group of patients were similar. Patients with clinical evidence of thyroid disease were excluded from the study. T_4, T_3-uptake, and TSH levels were determined before the TRH infusion; they were within normal limits for all patients. All patients were free of medications, except flurazepam, for at least 1 week before the TRH infusion. Medication histories of the patients revealed that in the 2 months before the TRH infusion, one of the manic patients, one of the schizophrenic patients, and none of the control patients received neuroleptics. During this time none of the patients received lithium or antidepressants.

An indwelling venous catheter was placed in patients who were at bedrest at 8:30 a.m. after an overnight fast. At 9:00 a.m. 500 μg of synthetic TRH was then administered over 30 seconds through the catheter. Before and 15, 30, 60, and 90 minutes after TRH administration, small samples of blood were obtained for measurement of serum TSH in duplicate by radioimmunoassay (5). The maximum TSH response, or delta TSH, was determined for each patient by subtracting the baseline TSH level from the peak TSH level after TRH administration.

Results

There were no significant differences between patient groups in baseline T_4, T_3-uptake, or TSH levels. The mean serum TSH concentrations for each group before and 15, 30, 60, and 90 minutes after TRH infusion are given in table 1. Within each patient group the mean TSH level at 30 minutes was significantly higher ($p<.01$) than the mean baseline TSH level. There were no delayed peaks. TSH values peaked at 15 or 30 minutes for all patients, except for one schizophrenic patient who peaked at 60 minutes. The TSH response in our control patients was in the same range as reported for normal subjects (7). There were no significant differences between the mean TSH levels for control and schizophrenic patients at any of the time points. The mean TSH levels in manic patients 15, 30, 60, and 90 minutes after TRH infusion were significantly decreased ($p<.05$) compared with control patients at the same time points; at 30 minutes they were significantly decreased ($p<.05$) compared to schizophrenic patients. The mean delta TSH levels in the manic patients was significantly lower than the mean delta TSH levels in the control group and in the schizophrenic patients (see table 1). The mean delta TSH levels in control and schizophrenic patients were not significantly different.

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TABLE 1
Serum TSH Levels in Control, Schizophrenic, and Manic Subjects After Infusion of 500 μg of TRH

	Serum TSH Levels (μIU/ml)					
	Control Group (N=5)		Schizophrenic Group (N=5)		Manic Group (N=5)	
Minutes After Infusion	Mean	SE	Mean	SE	Mean	SE
0	2.9	0.4	2.5	0.7	2.3	0.1
15	11.4	1.1	10.1	1.9	6.3	0.9[a]
30	12.9	1.6	11.6	1.4	6.8	1.4[a, b]
60	11.3	1.5	9.4	1.6	5.5	0.8[a]
90	10.3	1.5	6.9	1.2	4.2	0.7[a]
Δ[c]	10.0	1.3	9.3	0.7	4.8	1.1[d]

[a]Significantly different from control patients ($p<.05$, two-tailed t test).
[b]Significantly different from schizophrenic patients ($p<.05$, two-tailed t test).
[c]Determined for each patient by subtracting baseline from peak TSH level after TRH infusion.
[d]Significantly different from control and schizophrenic patients ($p<.02$ and $p<.01$ respectively, two-tailed t test).

Discussion

The results demonstrate a decreased TSH response to TRH in manic patients but not in schizophrenic patients, which is not explainable by differences in baseline thyroid function. This finding is consistent with previous reports of nonsignificant decreases in TSH response to TRH in mania (7). This finding may have practical application in diagnosis and treatment planning, as well as contribute to the understanding of the biological substrate of the two disorders.

The finding demonstrates agreement between clinical diagnosis using reliable and strict diagnostic criteria (RDC) and the magnitude of the TSH response to TRH. This agreement suggests that whenever mania is difficult to distinguish clinically from schizophrenia, the results of the TRH test may help solidify the physician's clinical impression and help determine the choice of treatment. Further research is needed to determine whether the results of the TRH test predict outcome or response to lithium versus neuroleptics in manic, schizoaffective, and schizophrenic states. There has been much interest during recent years in the response to lithium of subgroups of schizophrenic patients (1). Of particular interest may be the pharmacological response of patients whose TRH test results are not consistent with clinical diagnosis, e.g., lithium response in schizophrenic patients with a blunted TSH response.

The release of TSH from the anterior pituitary is mediated by the hypothalamic tripeptide TRH (8). Norepinephrine and dopamine stimulate and serotonin inhibits TRH release (9). Our findings are consistent with the hypothesis that mania is characterized by increased central noradrenergic and possibly dopaminergic transmission. This would lead to increased TRH release and subsequent homeostatic adjustment of the pituitary TRH receptors to become less sensitive or "down-regulate." The TSH response to TRH has been shown to be decreased in unipolar depression (5, 10). An understanding of why unipolar depression and mania manifest similar TRH test abnormalities requires further study. Because there is some evidence that TRH itself has antidepressant effects (10), it is possible that alteration in TRH release is not just an index of changes in brain monoamines but is etiologically related to affective illness.

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Catatonia in a Patient with Subdural Hematomas

BY SCOTT W. WOODS, M.D.

When Kraepelin (1) classified catatonia as a subset of dementia praecox, he emphasized that catatonic behavior occurred in many other clinical settings. Nevertheless, reports of catatonia in association with nonschizophrenic illnesses have been overlooked until recently. Gelenberg (2) defined catatonia as a syndrome and referred to catatonic patients who were diagnosed as having affective disorders, neurotic and situational disturbances, and approximately 40 organic conditions. I will describe a case in which transient catatonic signs developed in a patient with bilateral chronic subdural hematomas and presumed Wernicke's encephalopathy.

Case Report

We admitted Mr. A, a 58-year-old unemployed, alcoholic man, to our psychiatric inpatient service for evaluation of his bradykinetic state with organic and catatonic characteristics.

Six days before he was admitted, Mr. A came to the emergency room complaining of a fever and cough that had lasted 3 days and an inability to dress himself during the preceding week. His physical examination was normal, and the emergency room physician diagnosed a viral syndrome. Mr. A refused to leave the hospital after dismissal and was arrested for loitering.

In jail Mr. A sat in one position for long periods, spoke only when spoken to, and was incontinent of urine. The police returned Mr. A to the emergency room because he seemed unable to take care of himself. He was disoriented and remained slow in speech and action. When examining Mr. A, a neurologic consultant noted waxy posturing. Mr. A's limited lateral gaze improved after the emergency room physician had administered thiamine. Mr. A's gait was wide-based and short-stepped. He reported a similar episode that had been diagnosed as "hallucinations," but denied current hallucinations. Review of Mr. A's records indicated that alcohol-related psychoses with hallucinations had precipitated two previous psychiatric hospitalizations. No mention was made of prior catatonia. Mr. A had no family history of psychosis or alcoholism.

His medical history included alcoholism, adult-onset diabetes mellitus, diverticulitis, and hemorrhoids. He took no medicines, was allergic to aspirin, smoked one pack of cigarettes per day, and said he had not drunk any alcoholic beverages for at least 6 days. He had sustained a head laceration with possible loss of consciousness 2 months before admission. A retired Air Force master sergeant, he had been unable to work for the last 5 years, apparently because of alcoholism. He had been sleeping in laundromats for several weeks before coming to the hospital.

I first examined Mr. A two days after his admission. He had deteriorated markedly and was completely incontinent of urine and unable to walk. A mental status exam revealed that Mr. A was alert but oriented only to person. He sat quite still in a wheelchair. He could not repeat any of three words he was asked to remember after a 5-minute time lapse. When pressed for the words, he would touch my knee and proclaim, "You just heard them." He was mute for several periods of 3–5 minutes and engaged in no spontaneous speech. Mr. A answered questions slowly in short single sentences, sometimes appropriately. Some of his replies consisted of changed unintelligible words. I documented no delusions or hallucinations. Mr. A's affect was flat, his face mask-like, and his motor behavior markedly bradykinetic. He did not move the left side of his body spontaneously.

Mr. A's vital signs were normal. He had several well-healed scars on his scalp and a benign systolic murmur. His visual fields were intact to confrontation, although acuity could not be tested. His pupils were equal in size and briskly reactive. Mr. A exhibited complete loss of upward, downward, and intortive voluntary gaze and partial loss of lateral gaze despite full lateral conjugate eye deviation on doll's eyes maneuver. He would not open his mouth. I noted severe generalized muscular rigidity and some spasticity but no catalepsy. Stereognosis and graphesthesia were decreased on his left side. Although I could not test Mr. A's coordination, his deep tendon reflexes were brisk and symmetrical, except for absent ankle jerks. His plantar reflexes were extensor bilaterally. Mr. A stood with difficulty, leaned backward, and appeared not to know how to walk. His snout, root, suck, grasp, palmomental, and jaw jerk reflexes were present.

Laboratory test results revealed a mild macrocytic anemia but were otherwise unremarkable. However, CT scan disclosed subdural hematomas in Mr. A's right parietotemporal region and in his left occipital region. Mr. A underwent evacuation of the right hematoma with gradual resolution of his bradykinesia, incontinence, left-right asymmetries, ophthalmoplegia, hypertonicity, left Babinski sign, and most of his frontal release signs. He still walked with short steps on a wide base. He was oriented to name and time but thought he was in another California city and confabulated on occasion. He spoke at length in a tangential fashion, with normal affect. From the 8th to the 10th postoperative day I observed that Mr. A again demonstrated cataleptic behavior.

Discussion

To my knowledge, this is the first report of subdural hematoma associated with catatonic signs occurring in

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an individual with relatively unclouded level of consciousness. The minimal criteria for recognizing the catatonic syndrome have not been adequately determined (3); however, Mr. A's catalepsy, mutism, and stupor seem sufficient (4). In one catatonic patient a subdural hematoma was accompanied by a depressed skull fracture and a semicomatose state (5).

Allen and associates (6) discovered subdural hematomas in 8% of 3,100 consecutive autopsies of state mental hospital patients. Formerly, invasive procedures such as cerebral angiography or exploratory craniotomy may have dissuaded psychiatrists from pursuing the possibility of the presence of subdural hematoma. With the development of brain scanning and of computerized tomography, subdural hematomas may become a more commonly recognized cause of catatonia.

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Desipramine: Cardiovascular Effects and Plasma Levels

BY MATTHEW V. RUDORFER, M.D., AND ROBERT C. YOUNG, M.D.

The tricyclic antidepressants can produce symptoms of cardiovascular toxicity such as disturbance of cardiac rate and rhythm, prolonged conduction, decreased myocardial function, and hypotension. These effects have been demonstrated most clearly in cases of tricyclic overdose in humans and in animal studies.

Desipramine has been subjected to little direct clinical study (1) except for an occasional report of overdose (2) or as the major demethylated metabolite of imipramine. The purpose of this study is to quantitate the changes in several cardiovascular parameters by examining a group of clinic patients without heart disease who were being treated with desipramine and to investigate the association between these changes and steady-state plasma levels.

Method

We selected 14 outpatients aged 22 to 41 years old (mean, 30 years), who were free of medical illness and had not been treated with psychotropic medications for 4 weeks. Informed consent was given after the procedures involved in this study were explained. A pretreatment ECG was obtained, together with measures of blood pressure and pulse taken while the patient was sitting and immediately after the patient stood. Desipramine was taken in a single bedtime dose beginning with 50 mg. The dose was increased by 50-mg increments every 3 days until the patient was receiving 150 mg/day of desipramine h.s. Compliance was monitored by pill count. Repeat ECG, pulse, and blood pressure parameters were obtained after 21 days. On days 20 and 21 venous blood samples were collected 10–14 hours after the bedtime dose using heparinized evacuated tubes. Venoject (Kimble-Terumo) tubes were used because they yield reliable determinations that are not suppressed compared with those determinations made from all-glass collection systems (3).

Plasma desipramine levels were determined by gas chromatography–mass spectrometry using the method of Biggs and associates (4), with trifluoracetylimidazole as the derivatizing reagent. Samples were run in duplicate, and paired determinations agreed within 5%. Plasma levels on the 2 days were averaged; the coefficients of variation were within 14%.

Data were analyzed using Student's t test and Pearson's product-moment correlation coefficient. All probabilities are two-tailed.

Results

The mean (±SE) steady-state desipramine level was 201±34 ng/ml; the highest value was 407 ng/ml. The therapeutic range for plasma desipramine levels, although not well studied, is believed to be 100–300 ng/ml (5). Before treatment no ECG abnormalities were noted.

Desipramine treatment resulted in statistically significant increases in PR and QRS intervals, as noted in table 1, but not in the corrected QT (QTc) interval.

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TABLE 1
Effect of Desipramine on ECG Findings[a] and Blood Pressure Measurements[a] of 14 Physically Healthy Patients

Measurement	Before Drug Treatment	During Drug Treatment	Change	Significance[b]
ECG rate (beats/minute)	79	94	+15	p<.01
PR interval (milliseconds)	146	158	+12	p<.05
QRS interval (milliseconds)	69	76	+7	p<.05
QTc interval (milliseconds)	402	413	+11	n.s.
Systolic blood pressure, sitting (mm Hg)	120	109	−11	p<.02
Systolic blood pressure, standing (mm Hg)	112	103	−9	p<.02
Systolic positional difference	8	6	−2	n.s.
Diastolic blood pressure, sitting (mm Hg)	80	76	−4	n.s.
Diastolic blood pressure, standing (mm Hg)	79	75	−4	n.s.
Diastolic positional difference	1	1	0	n.s.

[a]Values are expressed as means.
[b]Paired t tests were used.

The ECG interval durations generally remained within normal limits, however. One PR interval was as long as 200 milliseconds, but none exceeded this length. One QRS interval exceeded 100 milliseconds by 13 milliseconds; this was associated with a plasma level of 360 ng/ml. Changes in the PR interval were significantly correlated with pretreatment values ($r=-.60$; $p<.05$); changes in the QRS and QTc intervals were not significantly correlated ($r=.40$ and $-.10$, respectively).

There was a significant correlation between plasma desipramine levels and changes in PR interval ($r=.53$; $p<.05$). Although the correlation between plasma desipramine levels and changes in QRS interval ($r=.49$) was not significant, the 7 patients with QRS increases of greater than 5 milliseconds had significantly higher plasma levels (281 ng/ml±36 ng/ml) than those with smaller increases (122 ng/ml±39 ng/ml; $p<.02$). Plasma desipramine levels were not correlated with changes in QTc interval ($r=-.02$).

Desipramine treatment produced statistically significant increases in the ECG rate up to 115 beats/minute. The increase in ECG rate during treatment was correlated with the pretreatment rate ($r=-.53$; $p<.05$). The resting radial pulse of patients increased in both sitting (mean, 76–90; $p<.01$) and standing (mean, 87–104; $p<.01$) positions. The patients did not complain of palpitation. Plasma desipramine levels were not correlated with changes in the ECG rate ($r=.15$).

Desipramine treatment was associated with consistent but small decreases in systolic blood pressure in both the sitting and standing positions. These decreases were correlated with pretreatment values ($r=.84$ and .93, respectively; $p<.001$). Before treatment there was a small drop in systolic blood pressure when patients rose from a sitting position to a standing position ($p<.05$) and during treatment ($p<.02$); the magnitude of this difference did not change significantly. Changes in systolic positional difference were correlated with pretreatment differences ($r=.69$; $p<.01$). Diastolic blood pressure was not significantly altered either by posture or by drug treatment. Changes in diastolic blood pressure during treatment were significantly correlated with pretreatment values sitting ($r=.77$; $p<.01$) but not with pretreatment values standing ($r=.41$). Changes in diastolic positional difference were significantly correlated with pretreatment differences ($r=.88$; $p<.001$). Standing also produced an increase in pulse rate both before and during treatment ($p<.001$); the degree of this increase did not change significantly. The subjects did not have subjective or objective complications of hypotension.

Plasma desipramine levels were significantly correlated with changes in systolic positional difference during treatment ($r=.64$; $p<.05$); these levels were not significantly correlated with changes in systolic blood pressure sitting or standing ($r=.35$ and 0, respectively), with changes in diastolic blood pressure sitting or standing ($r=-.05$ and .21, respectively), or with changes in diastolic positional difference ($r=.27$).

Discussion

These young, physically healthy subjects who were treated with desipramine had consistent prolongation of intracardiac conduction. This finding contrasts with the apparent lack of such an effect detected in similar subjects at therapeutic plasma levels of amitriptyline (6) and nortriptyline (7); to our knowledge these drugs reportedly produce only tachycardia in such subjects. In further contrast, the therapeutic total tricyclic levels of patients being treated with imipramine, the tertiary amine of desipramine, have been reported to produce not only tachycardia but greater degrees of conduction delay (8, 9) and significant postural blood pressure changes (10); however, these patients were older and had preexisting heart disease.

More data comparing the cardiovascular toxicities of individual tricyclics at therapeutic plasma levels in older and in young healthy populations are needed.

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Cardiovascular Effects of Nortriptyline in Geriatric Patients

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Concern about the cardiovascular side effects of antidepressant medication may often deter clinicians from prescribing these drugs appropriately for depressed patients with a history of ECG evidence of cardiac disease. Epidemiological studies have suggested that amitriptyline may be associated with sudden death in patients with cardiac disease (1, 2). Vohra and associates (3) and Burrows and associates (4) investigated the effects of antidepressants on intracardiac conduction in clinical and experimental studies and concluded that nortriptyline may be particularly cardiotoxic and that it may have significantly greater effects on several ECG parameters than doxepin. Zeigler and associates (5) reported the development of premature ventricular contractions in a 65-year-old man treated with nortriptyline. However, Freyschuss and associates (6) reported that nortriptyline had very little effect on inducing orthostatic hypotension. Because many geriatric patients suffering from clinical depression will also have evidence of preexisting cardiac disease, psychiatrists may refrain from using antidepressant medication in these patients or may prescribe doses that are too low to obtain maximum therapeutic efficacy. To assess more accurately the clinical importance of cardiac side effects of antidepressant medication in the elderly, we report here the preliminary results from a controlled study we conducted on the effects of standard doses of the tricyclic antidepressant nortriptyline in geriatric and nongeriatric depressed patients.

Method

We selected as our study group 12 geriatric depressed patients who were being treated in the inpatient services of the Texas Research Institute of Mental Sciences or the Texas Kerrville State Hospital. The patients had a mean age of 67.8 years (range, 60–78 years). Three younger depressed patients were also selected; they had a mean age of 25.8 years. After a 1- to 3-week drug-free washout period, each patient was started on a fixed dose of nortriptyline, usually 150 mg/day t.i.d. To determine the effects of nortriptyline on the patient's cardiovascular system a standard ECG recording was made once or twice a week during the patient's drug-free period and then 2 to 4 weeks after the patient started treatment with nortriptyline. Our ECG results were based on an average of 2.6 ECG recordings per patient in the predrug period and 3.9 recordings during regular treatment with nortriptyline. A research nurse using a standard procedure assessed the patient's orthostatic hypotension two or three times a week in the morning before the first dose was administered. Blood pressure was checked twice while the patient was resting in bed and checked twice again within a minute after the patient stood up. Plasma levels of nortriptyline were determined by drawing plasma samples twice a week between 8:00 a.m. and 8:30 a.m., 11 to 12 hours after the previous night's dose had been taken. The assays of nortriptyline were done by a gas liquid chromatographic procedure with a

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TABLE 1
Effects of Nortriptyline on ECG[a] Findings in 15 Patients With and Without Preexisting Cardiovascular Disease

Patient	Age (years)	Steady-State Plasma Level (ng/ml)	ECG Findings				
			Before Drug Treatment	Change After Drug Treatment			
				Description	QTc[b] (sec)	PR[c] (sec)	HR[d] (beat/min)
1	64	140–180	Sinus arrhythmia, single and bigeminal ectopic beats (supraventricular premature contractions and premature ventricular contractions)	Sinus rhythm; decrease in ectopic beats	−.02	.03	−1
		187–250[e]		T-wave inversions; 1° heart block	−.05	.02	−1
2	61	144	Supraventricular premature contractions	None	−.05	.02	−4
3	74	145	Right atrial hypertrophy; P. pulmonale; old interior wall infarct pattern	None	.03	.00	15
4	21	155	Wolff-Parkinson-White syndrome	None	.00	−.02	16
5	79	90	Left and right bundle branch block; left ventricular hypertrophy; inferior wall ischemic changes	Slight increase in left bundle branch block only	.04	.01	−1
6	70	133	Normal; old inferior wall infarct	None	−.01	−.01	3
7	63	164	Normal	Normal	.02	−.01	3
8	67	124	Normal	Normal	.02	.02	14
9	64	138	Normal	Normal	.01	.02	−2
10	60	133	Normal	Normal	−.02	.03	6
11	67	133	Normal	Normal	.01	.02	13
12	78	181	Normal	Normal	.00	.04	−4
13	59		Normal	Normal	.00	.00	−1
14	22	129	Normal; bradycardia	Normal	.02	.00	25
15	32	128	Normal	Normal	.01	.02	−33

[a]Standard ECGs used; patient 1 was studied a second time with 24-hour Holter-monitor ECGs.
[b]Change in QTc=steady-state QTc minus predrug QTc; steady state QTc=Q-T interval divided by the square root of the R-R interval.
[c]Change in PR=steady-state heart rate minus predrug heart rate.
[d]Change in HR=steady-state heart rate minus predrug heart rate.
[e]Toxic range.

nitrogen detector; chlorimipramine was used as the internal standard.

Results

Most geriatric patients achieved plasma levels of nortriptyline (mean steady-state plasma levels=90–160 ng/ml) within or slightly above the previously established therapeutic window for nortriptyline (see table 1). At these plasma levels nortriptyline had no clinically significant effects on the ECGs of geriatric patients. The mean (±SD) changes in standard ECGs when the patients were receiving nortriptyline as compared with their ECGs during the predrug washout period were heart rate=3.5±6.6 beats/minute; QT_c=6±29 milliseconds; PR=11±10 milliseconds; QRS=10±11 milliseconds. Although some of the geriatric patients had statistically significant increases in heart rate and QT_c, no patient's mean heart rate increased to levels above 100 beats/minute, i.e., none experienced clinically significant tachycardia, and the maximum increase in mean QT_c was 32 milliseconds. Even when patients who had evidence of clinically significant preexisting cardiac disease were treated with standard doses of nortriptyline (150 mg/day) and had plasma levels up to 180 ng/ml (patients 1 to 6 in table 1), nortriptyline produced no evidence of increased cardiovascular toxicity.

The effects of nortriptyline on decreasing intracardiac conduction were evident in patient 1. She was studied a second time with 24-hour Holter-monitor ECG recordings made weekly during 3 weeks of a drug-free washout period and subsequently during several weeks of treatment with 100 mg/day of nortriptyline. When her plasma nortriptyline levels were 140–180 ng/ml, her ectopic beats decreased from a mean of 42 ectopic beats per 24-hour period during the 3-week baseline period to a mean of 15 ectopic beats per 24-hour period during her third week of treatment with nortriptyline. When she had previously been treated with a higher dose of nortriptyline, 150–200 mg/day, she had plasma levels of 187–250 ng/ml; standard ECGs showed that she had developed signs of first-degree heart block. This subsided when the nor-

triptyline dose was reduced. These quinidine-like effects of nortriptyline are similar to those previously reported (7, 8) for another antidepressant, imipramine.

The degree of orthostatic hypotension produced by nortriptyline did not differ in 5 geriatric and 3 younger patients in whom we made standardized assessments. Orthostatic hypotension results, therefore, were combined across age groups. Nortriptyline did increase the drop in the systolic component or orthostatic hypotension but not in the diastolic component. During the washout period the mean drop in systolic pressure when the patient changed from a lying position to a standing one was 10.3 mm Hg; during the regular treatment with nortriptyline the mean drop was 24.6 mm Hg. Therefore, the effect attributable to nortriptyline was 14.3 mm Hg. This is similar to the reported effect (7, 9) of imipramine on the systolic component of orthostatic hypotension (mean=−15.2 mm Hg). The mean drop in diastolic pressure from lying to standing during the washout period was 1.9 mm Hg, and during treatment with nortriptyline it was 3.8 mm Hg, resulting in a drug effect of 1.9 mm Hg. This is smaller than the drug effect on diastolic orthostatic hypotension reported for imipramine (mean=−12 mm Hg) (7, 9).

Discussion

The cardiovascular effects of nortriptyline in these geriatric patients appear to be different in important respects from previous clinical reports (1, 6). A standard dose of nortriptyline, 150 mg/day, produced blood levels within or slightly above the therapeutic window in most of the geriatric patients. We found nortriptyline at this dose to be safe in regard to cardiovascular toxicity, even in patients with stable, preexisting cardiovascular disease.

It is important to look at the dose, blood levels, and clinical condition of the patient when interpreting the differences between our results and those of other investigators. Vohra and associates (3) and Burrows and associates (4) reported clinically significant effects of nortriptyline on the patients' ECGs. The mean nortriptyline level of their patients, however, was 196 ng/ml, which is well above the therapeutic window; a few of their patients had plasma levels of 300 ng/ml or 400 ng/ml. In the study by Zeigler and associates (5) the patient who developed premature ventricular contractions had a nortriptyline level of 176 ng/ml. In contrast, most of the patients in our study had nortriptyline plasma levels between 90 and 160 ng/ml. The patient who developed first-degree heart block had nortriptyline levels above 187 ng/ml. These comparisons emphasize the importance of considering the patient's blood levels of the antidepressant when making clinically relevant interpretations of the comparative cardiac toxicity of antidepressant drugs.

Although other researchers (1, 2) reported a significantly higher number of sudden deaths due to cardiac conditions in patients treated with amitriptyline, many of the amitriptyline patients they studied were admitted to a hospital with acute conditions. On the other hand, all of our patients with preexisting cardiac disease had relatively stable, nonacute cardiac conditions.

Orthostatic hypotension may be a clinically more important side effect of antidepressants, particularly in geriatric patients. More frequently than other cardiovascular side effects, orthostatic hypotension may occur at antidepressant blood levels necessary for adequate clinical response. Contrary to the results of Freyschuss and associates (6), who reported no decrease in orthostatic blood pressure due to nortriptyline, our results indicate a significant effect of nortriptyline on systolic orthostatic hypotension. This effect is quantitatively similar to that reported by Glassman and associates (9) for imipramine. Although nortriptyline produced less orthostatic hypotension in the diastolic component than imipramine, the clinical significance of the systolic versus diastolic component of orthostatic hypotension is unclear.

Other preliminary data for our study (10) indicate that at these standard doses nortriptyline is an effective antidepressant in geriatric depressed patients. We believe, therefore, that standard doses of nortriptyline, e.g., 100–150 mg/day, may be used safely and effectively in geriatric patients, even for those with stable, preexisting cardiac conditions. Plasma-level monitoring, however, may be more mandatory in geriatric patients in order to quickly identify those patients who may be subject to greater risk of cardiac side effects. These conclusions pertain to the specific tricyclic antidepressant we investigated, nortriptyline, and may not be generalizable to other antidepressant drugs.

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Iatrogenic Opiate Addiction: Successful Detoxification with Clonidine

BY DENNIS S. CHARNEY, M.D., AND HERBERT D. KLEBER, M.D.

Recently researchers have documented the effectiveness of the α_2-adrenergic agonist, clonidine, in eliminating objective signs and subjective symptoms of heroin and methadone withdrawal (1–3). These preliminary findings suggest that clonidine enables a faster, less severe detoxification from opiate drugs than gradual methadone reduction. If these observations are confirmed, clonidine will be potentially useful not only in detoxifying heroin and methadone addicts but also in treating patients with iatrogenic addictions resulting from chronic pain syndromes. The following is, to our knowledge, the first report of the use of clonidine to detoxify an iatrogenic opiate addiction secondary to chronic pain.

Case Report

Ms. A, a 51-year-old woman, had a 28-year history of chronic interstitial cystitis. For the past 15 years, Ms. A had been addicted to an oxycodone combination (Percodan), which she had begun to take because of chronic bladder pain. During the last 10 years she had been taking at least 40 tablets per week. Her urologists felt that Ms. A's complaints were excessive and her constant use of narcotics unwarranted. Despite these reservations, her physicians constantly supplied Ms. A with oxycodone prescriptions. During Ms. A's 15-year use of oxycodone both as an inpatient and an outpatient, several attempts to taper the oxycodone failed. Six years before we admitted Ms. A, she had attended once a week psychotherapy sessions for 6 months. Her therapist had described her as a chronically depressed woman with passive-dependent personality features.

In September 1979 Ms. A was referred to our Drug Dependence Unit by her urologist, who was no longer willing to supply oxycodone prescriptions for Ms. A's chronic complaints of bladder pain. In addition to oxycodone, Ms. A was receiving propanolol, 40 mg t.i.d., and hydrochlorothiazide, 50 mg/day, for essential hypertension and chlordiazepoxide hydrochloride, 10 mg t.i.d., for chronic anxiety. We could not definitively ascertain the etiology of Ms. A's pain, but it was clear that psychological issues contributed to it and that Ms. A had become tolerant to the analgesic effects of oxycodone. Considering this tolerance and Ms. A's history of unsuccessful detoxification attempts, we recommended oxycodone detoxification with clonidine.

At admission Ms. A weighed 86.4 kg and her standing blood pressure was 120/80. On the first day of hospitalization Ms. A received her regular doses of oxycodone (1 tablet, 4.5 mg, every 4 hours). However, we decreased her propanolol dose to 40 mg b.i.d. and discontinued her diuretic because of the anticipated synergistic interaction with clonidine in lowering blood pressure. We maintained Ms. A's chlordiazepoxide hydrochloride dose of 10 mg t.i.d. throughout her hospitalization to avoid a benzodiazepine withdrawal syndrome. On day 2 we discontinued oxycodone, and gave a single 40-mg dose of propanolol, and administered clonidine, 0.1 mg at 8 a.m. and 3 p.m., and 0.2 mg at 10 p.m. We continued to administer clonidine until Ms. A was discharged on day 10 (see table 1).

Throughout her hospitalization Ms. A's withdrawal signs and symptoms were assessed by a nurse's abstinence rating scale (2) three times daily. We checked Ms. A's blood pressure, lying and standing, and her pulse immediately before and 1 hour after administering clonidine, except for the first 2 dosages, when we took her blood pressure each hour for 3 hours after administering the clonidine.

Based on the abstinence scale and clinical observation, Ms. A exhibited few signs or symptoms of withdrawal. We observed only mild anorexia (days 3–5) and diarrhea (day 4). On day 2 Ms. A complained of a headache. Since the oxycodone combination contained caffeine, we thought Ms. A's headache might be a symptom of caffeine withdrawal and advised her to increase her coffee intake. This stopped the headaches and they did not recur. Ms. A had a significant fall in standing blood pressure, from 120/80 to 90/60, at the higher doses of clonidine, which are associated with mild postural dizziness and lightheadedness. Ms. A did not complain of pain during hospitalization. At discharge she was asymptomatic, her blood pressure had returned to 120/80, and the only medication she was receiving was a diuretic.

At a 6-month follow-up Ms. A remained free of narcotics and was attending biweekly supportive psychotherapy sessions at the Drug Dependence Unit. Her urologist remarked that he had never seen her so alert and energetic.

Discussion

Although somewhat milder, the abstinence syndrome associated with oxycodone resembles that of

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TABLE 1
Clonidine Dosage Schedule to Detoxify Patient with an Iatrogenic Opiate Addiction

Hospital Day[a]	Clonidine (mg)			
	8 a.m.	3 p.m.	10 p.m.	Total
2	.1	.1	.2	.4
3	.3	.3	.4	1.0
4	.3	.3	.3	.9
5	.3	.3	.3	.9
6	.3	.3	.3	.9
7	.2	.2	.2	.6
8	.1	.1	.1	.3
9	.1	0	0	.1

[a]On hospital day 1, the patient received oxycodone, 1 tablet every 4 hours; on hospital day 10, the patient was discharged.

morphine and lasts approximately 7 days (4). In Ms. A clonidine almost completely blocked the emergence of the syndrome, presumably by reducing norepinephrine turnover in the central nervous system (5).

The effective clonidine dose in Ms. A (10 μg/kg per day) resembles doses used in previous studies with heroin and methadone addicts in which doses up to 17 μg/kg per day were used (1–3). We used the three-times-daily dosage schedule because a single dose of clonidine can eliminate the opiate abstinence syndrome for 240–360 minutes (1). Two factors determined the necessary duration of clonidine administration for smooth detoxification: 1) the half-life of the addicting narcotic, which is related to the length and intensity of the untreated abstinence syndrome, and 2) the need for a clonidine taper. Abrupt discontinuation of clonidine causes a withdrawal syndrome (6), but its effects are not severe and patients can withdraw from clonidine with relative ease. Five days of relatively high doses of clonidine and a 3-day taper provided a safe, effective detoxification for Ms. A.

These findings may have several important therapeutic implications. Prescription drug addiction is a common problem for patients complaining of chronic pain (7). Clonidine can be used as a rapid nonnarcotic means of helping these patients to withdraw from narcotic analgesics. Clonidine in conjunction with psychotherapy may enable patients who do not suffer from discernible physical illness but who have psychological difficulties to detoxify and remain drug free. For patients with painful physical disease, periodic clonidine detoxifications may provide more effective analgesia by temporarily reversing the tolerance developed over long-term narcotic use. These patients may not complain of pain during the detoxification because clonidine has been shown to have analgesic properties similar to the effects of morphine in laboratory animals (8, 9).

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IN MEMORIAM

Hugh Thompson Carmichael 1898–1980

I met Hugh Carmichael when he spoke to our freshman medical class at the University of Illinois in Chicago; he was then and to the end of his days one whose presence lent a dignity to his surroundings. I next met Hugh Carmichael during my training at the university department of psychiatry. He became my mentor, analyst, colleague, and friend. Those were the halcyon days for many of us at Illinois when, under the direction of our department head Melvin Sabshin, Carmichael was appointed director of undergraduate education in psychiatry. Many of us, infected by his enthusiasm and abiding quest for excellence, became involved with the problems of curriculum design, experimental teaching formats, and evaluation mechanisms. His style of leadership was particularly supportive of new directions; he welcomed and was an instant advocate of creativity in teaching and learning, which he would energetically advance if it fulfilled his standards of excellence (Hugh did not too gladly abide foolishness).

When he came to work for APA after retirement from the University of Illinois, Carmichael continued his interest in education, now with an emphasis on the self-assessment of the members of the American Psychiatric Association, which would hopefully lead to self-improvement and a lifetime of learning. Once again he was able to infect a number of willing workers with the abiding notions of the Carmichael litany, which emphasized excellence as a way of life. Carmichael's emphasis (1–4) on the continuation of the education of psychiatric physicians was one of the important early influences on the continuing education movement.

Carmichael had many other "selves"; in his lifetime he was a general practitioner, a neuroendocrinologist, a neurologist and neuropathologist, a research psychiatrist, and a training and supervising psychoanalyst (Chicago Institute for Psychoanalysis). He held important positions in many major bodies in psychiatry and psychoanalysis, including the vice-presidencies of the American Psychiatric Association and the American Board of Psychiatry and Neurology. From his origin in Peterborough, Ont., Canada, at the turn of the century, his educational and professional travels took him to the Mayo Clinic, Albany Medical College, Worcester State Hospital, and the University of Chicago hospitals and medical school, among other places. His scientific and professional activities in these years ranged from psychophysiologic research to neuropathological investigations.

When he came to Chicago in the 1930s, Hugh Carmichael worked with Grinker at the University of Chicago and began his psychoanalytic training with Terese Benedek, who became his revered mentor and analyst. It was under her guidance that he wrote his first analytic paper, "A Psychoanalytic Study of a Case of Eunuchoidism" (5). For 25 years he was a training and supervising analyst at the Chicago Institute for Psychoanalysis. When he transferred to the University of Illinois in the 1950s, it was to help develop the department of psychiatry at Illinois with Francis Gerty. With his characteristic vigor he here influenced many young psychiatrists to become involved in psychoanalytic psychiatry. It was also at Illinois that he performed the first psychotherapy that was filmed.

A description of Hugh Carmichael would never be complete without telling of his generosities as friend and as physician. Perhaps one of his most important "teaching" activities was to take us into his home, to demonstrate through his interactions with his ever-gracious wife Carolyn and his family how to "do business," as he always said, in a family setting. The atmosphere was always filled with merriment and, more importantly, the sense of acceptance by our leader.

These considerations lead to the final summarization of Hugh Carmichael as the complete teacher—one who through his example of gentility and empathy, his capacity to function as a nurturer of our autonomy, and his capacity to lend content to our ideals helped develop in all those who came under his wings the impetus to achieve. He was, I believe, a fortunate man; he had achieved the ability to stand apart from his students and be filled with joy at their accomplishments. What man could wish for more?

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HYMAN L. MUSLIN, M.D.

LETTERS TO THE EDITOR

This section contains reactions to Journal *articles, statements of opinion, brief research findings or ideas, short case descriptions, requests for information, etc. Letters should be submitted in duplicate to the Editor, who makes all decisions regarding publication. Letters must be typed double spaced throughout and should not contain more than 500 words and 5 pertinent references. Criticisms of published articles will automatically be sent to the author(s) for response. Letters will be edited for clarity and conformance with* Journal *style. We regret that we cannot inform writers of the disposition of letters or return those which are not printed.*

Defining Drug-Induced Supersensitivity

SIR: In "Neuroleptic-Induced Supersensitivity Psychosis: Clinical and Pharmacologic Characteristics" (January 1980 issue), Guy Chouinard, M.D., M.Sc., and Barry D. Jones, M.D., proposed that patients whose psychosis becomes progressively resistant to neuroleptic therapy and show end-of-dose symptom exacerbation should be considered dopaminergically supersensitive. They compared this supersensitivity with that seen in patients with tardive dyskinesia, relating the former to mesolimbic dopamine receptors and the latter to the striatum. While the description of such patients may be accurate, the use of the term "supersensitive" to describe these symptoms is unwarranted from the data given and may be entirely misleading in terms of neuropharmacologic implications.

Drug-induced supersensitivity refers to an enhanced behavioral or biochemical response to an agent (1). The development of a new symptom complex after drug exposure, as seen with tardive dyskinesia, or the development of symptomatology that is more marked after drug exposure than before therapy are examples of drug-induced supersensitivity. The cases presented by Drs. Chouinard and Jones, however, do not fit these criteria.

The patients described were psychotic when neuroleptic therapy was initiated and became psychotic again as their next dose of depot neuroleptic approached. Eventually their psychoses were no longer controlled by the former doses of medication, so that higher neuroleptic doses were required. When a higher drug dose is needed to achieve the same behavioral effect, we refer to the phenomenon as "tolerance." There is no indication from the report that the psychosis itself progressed, only that the same symptoms tended to recur at doses that had formerly controlled them.

Before maintaining that the patients showed supersensitivity, the authors must demonstrate a clear progression of the psychotic symptom complex itself, perhaps in the form of new hallucinations in patients who never hallucinated before or a significant deterioration in thought disorder compared with premedication psychiatric evaluation. Drs. Chouinard and Jones provided no data to indicate that this progress occurred, so the assertion that these patients showed dopaminergic supersensitivity was premature and unjustified.

I do not dispute the possibility of mesolimbic supersensitivity. Laboratory data on receptor site changes in this system after chronic neuroleptic exposure suggest that, at least in experimental animals, the phenomenon may exist. Rather, I would emphasize the precise pharmacologic restrictions of supersensitivity and stress the importance of an appropriate experimental design to identify it.

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CHRISTOPHER G. GOETZ, M.D.
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Drs. Chouinard and Jones Reply

SIR: Dr. Goetz appears to have overlooked an important characteristic of the phenomenon we described—the increased frequency of the reemergence of psychotic symptoms in patients treated chronically with neuroleptics. The courses of our patients' illnesses were different from those one would normally expect in schizophrenia. In our patients relapse occurred immediately and invariably with drug withdrawal or dosage reduction and their symptoms emerged as drug blood levels declined. Before "supersensitivity" developed, the relapse rate averaged once per year. Not only did the frequency of relapse increase markedly, but this kind of relapse differed from that of the true schizophrenic relapse because it could be controlled immediately by increasing the drug dosage. For this reason we chose the term "supersensitivity" to characterize the emergent psychosis. If we were to experimentally demonstrate the emergence of psychotic symptoms not previously present or exacerbation of symptoms by the long-term administration of neuroleptics, we would have to administer neuroleptic drugs to nonpsychotic subjects or deliberately allow exacerbation of symptomatology without appropriate dosage adjustment. Both of these approaches would be unjustifiable from an ethical point of view. We intended our article to draw attention to signs of what we consider to be supersensitivity to the drug's antipsychotic action developing in patients on long-term neuroleptic treatment.

When we speak of "tolerance," we mean "behavioral tolerance" and not "metabolic tolerance" as Dr. Goetz seems to imply. Behavioral tolerance to chronic treatment consists of a return of behavior to pretreatment baselines after significant initial changes, and is related to a neurochemical adjustment at a subcellular level. Behavioral tolerance to neurolep-

tics has been clearly shown to occur in animals (1). In humans behavioral tolerance to sedative action and parkinsonian side effects of neuroleptics is well established. However, tolerance to the therapeutic effect of neuroleptics has not been demonstrated. We first observed this tolerance in a double-blind study examining the therapeutic effect of long-acting injectable neuroleptics (2). Furthermore, metabolic tolerance was ruled out in our patients since their prolactin levels continued to increase with increased doses of neuroleptics.

We drew the analogy between the supersensitivity psychosis and tardive dyskinesia because both are related to long-term neuroleptic administration, follow a similar progressive course, and respond in the same manner to dosage variations. A recent study demonstrated an increase in dopamine binding sites in the mesolimbic regions of postmortem brains of schizophrenic patients that correlated with neuroleptic treatment (3). This finding supports the theory that supersensitivity in the mesolimbic region causes the increased frequency of reemergence of psychotic symptoms.

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GUY CHOUINARD, M.D., M.Sc.
BARRY D. JONES, M.D.
Montreal, Que., Canada

A Political Implication of Drug-Induced Supersensitivity

SIR: "Neuroleptic-Induced Supersensitivity Psychosis: Clinical and Pharmacologic Characteristics," by Guy Chouinard, M.D., M.Sc., and Barry D. Jones, M.D. (January 1980 issue) will heighten psychiatrists' concern about the adverse effects of these powerful drugs. I would like to point out another unhappy implication of these findings. The abuse of psychiatry for political purposes, e.g., silencing dissenters by removing them to mental hospitals for "treatment" has been well documented (1, 2) and has drawn official condemnation from the World Psychiatric Association. This practice may include the forcible administration of neuroleptics over long periods of time (2). The possibility arises that a psychotic state based on supersensitivity could be induced in a previously nonpsychotic "patient" held in such circumstances. Although this might occur in only a minority of cases, the chances would be increased by severe environmental stress. Subsequent examination by an impartial team investigating alleged abuse might then reveal the presence of psychosis (which did not exist before "treatment") and the investigators might incorrectly conclude that the hospitalization and treatment had been clinically justified.

Cases of alleged political abuse of psychiatry involving involuntary administration of neuroleptics should be investigated as quickly as possible, before the "treatment" itself could cloud the clinical picture. American psychiatrists should continue to confront this important issue in the forum of the World Psychiatric Association and to press for the early investigation of all cases.

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ROGER K. PITMAN, M.D.
Memphis, Tenn.

In Defense of the JCAH

SIR: I would like to comment on Dr. Daniel B. Schuster's letter to the Editor, "The Burden of Documentation" (May 1980 issue).

The Joint Commission on Accreditation of Hospitals has always enjoyed and promoted a favorable working relationship with the medical community. In the past several years we have made a conscious effort to strengthen that relationship. We believe one of the outcomes has been significant involvement in all aspects of the survey and accreditation programs by members of APA.

Standards of the Joint Commission consistently stress medical responsibility for patient care. Concurrently, they have attempted to appropriately acknowledge the role and responsibilities of nonmedical professionals in the field of mental health. We appreciate Dr. Schuster's concern with the increase in need for documentation. This issue is among many that are receiving close attention as the proposed revisions to the *Consolidated Standards*. They are currently being subjected to extensive review by the mental health field. As in the past, comments from APA members will make an important contribution to the final revision of these *Standards*.

The role of the Joint Commission is to develop quality assessment and quality assurance mechanisms that will assist facilities in creating an appropriate setting for the delivery of optimal, achievable patient care. We seek to accomplish this task through a thoughtful process of education, consultation, and evaluation in cooperation with the field we serve.

JOHN E. AFFELDT, M.D.
President, Joint Commission on Accreditation of Hospitals
Chicago, Ill.

Assessing Mistakes in Psychotherapy

SIR: In "Common Mistakes in Psychotherapy" (December 1979 issue) Peter Buckley, M.B., Ch.B., and associates addressed an area important to all of us involved in the teaching and practice of psychotherapy. However, the authors omitted information essential to the proper evaluation of their contribution. This presumably unintentional omission could reflect a tendency to overgeneralize their findings.

The term "mistake" implies there is a correct or appropriate type of therapy to be administered. What is termed correct or appropriate psychotherapy in any given case of psychotherapy supervision depends on at least three factors: the type or diagnosis of the patient, the type of psychotherapy

being conducted, and the theoretical orientation of the supervisor. I teach psychotherapy on a general inpatient service where we treat severely disturbed patients. Buckley and associates listed "wanting to be liked by the patient" as the most common mistake in psychotherapy. However, on our inpatient service "wanting to be liked by the patient" more frequently indicates that the therapist is building an emotionally positive, trusting, working alliance, the primary requirement for psychotherapy with our patient population. A supervisor of the existential school of psychotherapy who stresses sharing the patient's experience would be more likely to view the "stereotyped 'analytic psychotherapist' stance" as a mistake than would a psychoanalytic supervisor.

Buckley and associates should have noted how certain supervisors commented on certain types of therapy for certain types of patients. If, as seems likely, the information was gathered largely from cases of moderately to well-functioning patients in insight-oriented therapy supervised primarily by psychoanalytically oriented staff members, the authors should so inform the reader. We need to know to what degree the findings are generalizable and not assume that they are so.

The title of the article did not mention that the therapists concerned were residents. Although the body of the article does clearly state the training status of the therapists, it also refers to the possible general applicability of the findings. Again, such general applicability should be demonstrated, not assumed. The title should accurately describe the contents for those readers who will only skim the article. Perhaps "Common Mistakes in Psychoanalytically Oriented Insight Psychotherapy by Psychiatric Residents" would have been better.

I appreciate Dr. Buckley and associates' paper and it is because I agree with the need to better evaluate psychotherapy that I write my comments.

BRUCE W. SCOTTON, M.D.
San Francisco, Calif.

Dr. Buckley and Associates Reply

SIR: Dr. Scotton is incorrect in assuming that our report of common mistakes was based on an a priori theoretical view of correct or appropriate psychotherapy. We based our 56-item questionnaire on elements in psychotherapy that had previously been related to negative effects by researchers (1) as well as on our own clinical experience and not on a particular theoretical ideology.

We also feel that there is some confusion on Dr. Scotton's part between the establishment of a working alliance and the wish on the part of the therapist to be liked by the patient. We do not think that these are equivalent. If the therapist's needs are unconsciously paramount in the treatment, it can hardly be therapeutic for the patient even if he or she likes the therapist. The therapist who wishes to be liked by a severely disturbed patient may ignore aspects of the patient's communication that are discrepant with that wish and hence obviate an effective treatment. We also feel that "assuming a stereotyped analytic psychotherapy stance regardless of the actual treatment situation" is a mistake, no matter what the theoretical orientation of the therapist or the supervisor might be. We believe such rigidity is inimical to effective therapy.

Our group of supervisors were primarily of a classical psychoanalytic persuasion, but the group did include practitioners trained in existential and interpersonal schools of psychoanalysis. We are not convinced that the theoretical beliefs of supervisors are as critically important as Dr. Scotton would suggest, since a number of studies have shown that theoretical distinctions between therapists blur with extensive experience and that experienced therapists, despite differing theoretical orientations, are more alike in technique than inexperienced therapists (2, 3).

We agree with Dr. Scotton that our title was too broad and that we should have indicated that the psychotherapy was being conducted by psychiatric residents.

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PETER BUCKLEY, M.D., CH.B.
TOKSOZ B. KARASU, M.D.
EDWARD CHARLES
Bronx, N.Y.

On New Trends in Psychotherapy

SIR: "Recent Trends in Psychotherapy" (April 1980 issue) by Judd Marmor, M.D., is a lucid, comprehensive psychosocial analysis of the factors involved in the explosion of new psychotherapies.

Dr. Marmor wrote, "It is a romantic notion that inner experiences alone can change and transform external reality . . . . We are responsible for much that happens to us, but the illusion of total responsibility can be destructive to a vulnerable personality."

Dr. Marmor also indicated that the "early rich hopes of the psychoanalytic revolution have not been fulfilled. . . . Its results have been relatively disappointing."

Dedicated psychoanalysts have attempted to modify Freud's classical approach by "increasing flexibility, modifying the frequency of visits, allowing patients to sit up during therapy, interviewing significant others, controlling transference regression, modifying noxious environmental factors where possible, and functioning as participant observers." It seems that the more psychoanalysis changed, the more it resembled Adolf Meyer's psychobiology.

All these changes in psychoanalytic technique were necessary and helpful. But is the final result of these modifications still Freud's "psychoanalysis"? Freud would have differed. He was cognizant of the analyst "who attempted to emphasize some single one of the findings or views of psychoanalysis at the expense of all the rest." Of one such modifier (Jung) Freud wrote, "He has changed the hilt, and inserted a new blade into it, and because the same trademark is engraved on it, we are required to regard the instrument as the former one" (1).

Dr. Marmor's paper is a scientific and humanistic tour de force. He is to be congratulated for presenting his clinical astuteness and holistic philosophy in the best possible format.

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JACOB H. CONN, M.D.
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The Voting Mental Patient

SIR: In light of the upcoming national elections, I thought it appropriate to consider the voting status of psychiatric patients. How many of the psychiatric patients who are hospitalized exercise their voting rights? How many patients have the right to vote taken away from them by legal means (commitment or otherwise)? Do states differ in dealing with aspects of the voting processes of patients? As elections approach, how many hospital directors provide literature from various political parties on the candidates or arrange for transportation to and from registration and the polls?

If the 1960s were the decade of voluntary admissions and back to the community movement, the 1980s should be the decade of civil liberation and the voting mental patient. All patients should be given the opportunity and encouragement to exercise their rights. Perhaps legislators would not be so slow to appropriate funds for hospitals and mental services if we made them aware that hospitalized patients are an active part of their constituencies. Political rallies at hospitals could have tremendous therapeutic effects, bringing mental patients back into the mainstream of American life. In certain local situations with close results at the polls, the patients of the mental hospitals, viewed as a deciding force, could become the targets of vigorous campaigning, forcing candidates into promises of action on their behalf.

VICTOR BERNAL Y DEL RIO, M.D.
San Juan, Puerto Rico

Identifying Pseudoseizures with Anhydrous Ammonia

SIR: Anhydrous ammonia has long been used in general medicine as a respiratory stimulant. Three recent cases suggested to us that it may have a value in psychiatry as well. However, we were unable to find any reports in the literature of its use. We request the assistance of *Journal* readers in locating references for the use of ammonia.

Case 1. Ms. A, a 26-year-old woman, was hospitalized for treatment of marital problems and secondary depression. After a stressful therapy session she fell in the hall. She exhibited jerking movements of the extremities, not typical of clonic movements. Her eyelids fluttered. She would not respond to verbal or painful stimuli. Her pulse and blood pressure were normal. One ampoule of anhydrous ammonia was administered. After one breath Ms. A opened her eyes and began talking. She complained of lightheadedness but remained alert thereafter.

Case 2. Ms. B, a 17-year-old woman who met *DSM-III* criteria for paranoid schizophrenia, had been taking haloperidol and benztropine mesylate intermittently. She was brought to the emergency room where she was found to have decreased spontaneous movements, cogwheel rigidity, and conjugate deviation of her eyes to the left. The most likely diagnosis seemed to be acute dystonia due to phenothiazines. However, Ms. B's medical history was vague enough to suggest a functional symptom, so one ampoule of ammonia was tried. After one breath she immediately turned her head and gazed at the physician. After a second breath Ms. B stood up and said she felt fine. She exhibited no signs of dystonic posturing during a 40-minute period of observation. Neurological reexamination revealed no abnormalities, and cogwheel rigidity had completely disappeared. Ms. B's deep tendon reflexes were brisk but symmetrical (3−). During another 40-minute period of observation no dystonic posturing recurred.

Case 3. Alfie, an 11-year-old boy, was admitted to a pediatric neurology service for evaluation of "spells" consisting of sudden onset of shaking, gasping, and crawling on the floor with eyes closed. He would not respond to verbal or painful stimuli during the spells, which usually began during tantrums. When a thorough workup, including EEG, CT scan, SMA-20, IVP, GTT, and 24-hour Holter EEG, yielded normal results, the neurologist believed his initial impression of pseudoseizures was confirmed. Treatment with saline injections had no effect on Alfie. During one spell an ampoule of anhydrous ammonia was administered. Alfie took one breath and his spell was immediately aborted. About 12 hours later another spell occurred and a second ampoule of ammonia was administered. After one breath Alfie stood up and said, "I don't need that; just give me the shot." No further spells occurred during the next 48 hours.

Under usual circumstances the patients we described would have received intramuscular injections for their symptoms. In cases such as these anhydrous ammonia may aid in the identification of pseudoseizures and other functional symptoms and render intramuscular treatments unnecessary. We would be particularly interested to hear from readers who have had experience with this management technique or who may know of relevant publications.

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Corrections

On page 641 of the book reviews section in the May 1980 issue the title of the book by Henry Kellerman, Ph.D., was listed incorrectly. The correct title is *Group Psychotherapy and Personality: Intersecting Structures*.

On page 651 of the May 1980 issue, in the list of those who successfully completed the examination of the American Board of Psychiatry and Neurology, the name Jong Jin, Marvin Young should have been listed as Jin, Marvin Young Jong.

The staff regrets these errors.

BOOK REVIEWS

Crisis Admission Units and Emergency Psychiatric Services. Public Health in Europe 11, *by J.E. Cooper. Copenhagen, Denmark, World Health Organization (Albany, N.Y., WHO Publications Centre USA, distributor), 1979, 118 pp., $6.60 plus $1.25 for postage and handling (paper).*

This book contains a comprehensive description of 15 emergency and crisis services in 8 European countries. These services range from psychiatric facilities without a distinct or separate emergency ward to sophisticated crisis units with overnight beds. When applicable, the author also describes the service delivery area, the physical arrangements and organization of the facility, the aim of the unit, the personnel, the patients, the record-keeping system, and related research and training. Emphasis in the discussion is on staffing models and issues. The author describes the organization of these units along a continuum from "the conventional medical," which maintains a vertical hierarchy, to the "unconventional social," which is based on a horizontal structure and involves teaming and sharing of clinical responsibility. Particularly informative are the sections on staff satisfaction, which discuss many of the issues involved in interdisciplinary teaming such as the blurring of professional roles.

Although the volume begins with a somewhat sketchy summary of crisis theory and practice, the focus in the book is on an organizational description of services and staffing rather than a discussion of various crisis intervention psychotherapy approaches. This manual fills a gap in the literature on emergency service delivery by pulling together materials that are usually widely scattered or generally unavailable. The format of the book makes the information easily retrievable. The appendixes, which contain a bibliography describing emergency service delivery and a comprehensive directory of the names and addresses of the directors of the European programs, provide additional useful information.

I highly recommend this manual as an excellent resource for anyone currently involved with organizational issues in an emergency setting and for those thinking of developing a crisis service.

ELLEN L. BASSUK, M.D.
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Parents and Children in Autism, *by Marian K. DeMyer. Washington, D.C., V.H. Winston & Sons (New York, N.Y., Halsted Press, John Wiley & Sons, distributor), 1979, 262 pp., $17.95.*

Dr. DeMyer is a well-known and respected authority in infantile autism. This volume is the product of her many years as Director of the Clinical Research Center for Early Childhood Schizophrenia, Indiana University School of Medicine. Data are presented on long-term studies of 155 families with autistic children; they are compared with matched families with normal children and matched families with nonautistic retarded children.

The book is not primarily a research report, although the author is familiar with the major research trends in the field and presents brief summaries of her own studies. Rather, as she puts it,

> This book traces the clinical course of autism from birth to adulthood and parental reactions and behavior from the mother's pregnancy through life with an "adult child." Empirical data are supplemented by case histories to illustrate some of the most typical features of the disorder. I have tried to show the many facets of normal and autistic development by using the eyes and the words of parents whose observations are too often underemphasized in psychiatric writings.

Dr. DeMyer succeeds admirably in her purposes. Her style is informal, even conversational, and refreshingly free of professional jargon. Her clinical expertise and sensitivity to her child patients and their parents are evident throughout. The individual chapters take up the problems and symptoms at successive age periods and in specific areas, such as speech and communication, feeding and eating, elimination and toilet training, sleeping, social behavior, emotional expression, discipline, and intelligence. There are careful tabulations of the incidence of these problems, the nature and intensity of parental concerns, and the approaches used by parents in handling the child's symptoms and difficulties. Wise, empirical advice is offered throughout. Many brief and pertinent clinical vignettes illustrate the issues discussed.

Although the author tries to maintain an optimistic approach, she does not hide the reality of the bleak prognosis for most cases of autism and the extraordinary stresses imposed on parents and siblings who live with and care for an autistic child. In line with other recent research findings, Dr. DeMyer's reports of her own comparisons of the personality characteristics of autistic and control parents show no significant differences between the two groups. However, she emphasizes that the parents of autistic children face enormous difficulties and pressures in rearing these children and that "such multiple stresses, continuing over months or years, had effects on the parents as individuals and marriage partners and eventually on the other children in the family" (p. 150). Of special interest are the chapters detailing the new problems and stresses the autistic child faces as he or she goes through adolescence and enters adulthood. In some cases, however (a small minority), there is significant and even dramatic improvement during these older age periods.

This volume can be highly useful to all professionals re-

sponsible for the care and treatment of autistic children. It can also be recommended to sophisticated parents of such children.

ALEXANDER THOMAS, M.D.
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Nim, *by Herbert S. Terrace. New York, N.Y., Alfred A. Knopf, 1979, 291 pp., $15.00.*

For the person who is unfamiliar with the problems involved, the hours spent, and the endless devotion necessary to try to teach a chimpanzee to communicate, this is an excellent book. The author and his colleagues spent more than four years in the task and another year analyzing their findings. Theirs was a thorough and courageous effort that only an informal account can capture.

The aim of the author's project was to demonstrate that a chimpanzee can create a sentence by communicating in sign language. Such aims carry their own set of methodological and conceptual burdens, and the author is well aware of them. For example, is the concept of "sentence" well enough defined to establish criteria for judging a chimpanzee's signs? Is it appropriate to compare chimpanzee signing to human utterances or does one have a choice? If one is using human teachers to teach sign language can he ever be certain that subtle communications are not predetermining a chimpanzee's signing sequences that others are taking as indications of sentence formations? A variety of attempts have been made to confront and, at times, avoid these problems through such means as token and computer communication, but in the attempt described in *Nim*, as in all previous attempts to determine if chimpanzees can create sentences, the findings remain ambiguous.

That the author was never fully satisfied that he achieved his aim makes the book no less interesting. His accounts of his frustrations, joys, and sorrows make *Nim* worthwhile reading.

MICHAEL T. MCGUIRE, M.D.
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Women: Psychology's Puzzle, *by Joanna Bunker Rohrbaugh. New York, N.Y., Basic Books, 1979, 491 pp., $15.00.*

The author of this book is a feminist and a careful and thorough scholar. This lengthy, closely printed text contains quotations from approximately 50 sources ranging from articles published in *Ms.* magazine to Freud's original papers, as well as many footnotes. The 22-page bibliography should, itself, be of great value to anyone who undertakes teaching "women's" studies as well as to the much larger group who speak on subjects of interest to women.

This generally excellent but pedantically written book covers all of the usual material in considerable depth. Psychiatrists will find the section on theories of female personality of particular interest. Dr. Rohrbaugh discusses women in psychoanalytic theory. She reviews cognitive-developmental and social learning theory in general and as they relate to women in our society. In "Woman's Place in Society" Dr. Rohrbaugh goes beyond the usual presentations regarding the dilemma of women, marriage, motherhood, and the single state (including lesbianism) to include working women and minorities.

In her chapter on "Third World Women" Dr. Rohrbaugh empathetically discusses the situation of black women in the United States and the concepts of black matriarchy and black macho. She concludes this chapter with a strong opinion:

> It is time for social scientists to examine the black experience from the viewpoint of blacks, themselves. It is time to stop imposing white, middle-class values on millions of Americans who do not necessarily share them. And, it is time, some would add, for black women to act on their own behalf. (p. 264)

This emphasis on the need for social scientists, physicians, and mental health workers, especially researchers in all of these fields, to consider "women's own opinions and experiences" is evident throughout the book. Sexuality, birth control, pregnancy, rape, the battered woman, and women's activities in sports are all carefully covered with a strong feminist focus.

The psychiatrist, especially the psychiatrist in private practice, will find Dr. Rohrbaugh's section on women and mental health of great interest. She discusses hysteria as superfemininity and poses questions as to why men with social charm, the ability to manipulate other people, and a lack of guilt for their actions are considered psychopaths instead of hysterics. She also writes about several other "female" mental illnesses, including depression, anorexia nervosa, and phobias.

Most interesting is Dr. Rohrbaugh's presentation of feminist therapy—both individual therapy and consciousness-raising groups. To quote her: "One of the central tenets of feminist therapy is that the therapeutic relationship should not involve power. A therapist should neither be a patriarch nor dominate or intimidate a client" (p. 445). Of consciousness-raising groups, in particular, Dr. Rohrbaugh states, "I would say that CR is not group therapy, nor is it appropriate for everyone, but it is a positive alternative to traditional psychotherapy for the millions of women who are seeking personal growth" (p. 461).

Dr. Rohrbaugh, a well-trained psychologist who is currently in private practice in Boston, teaches courses on the psychology of women and women and mental health at Harvard. She has produced a scholarly and thoughtful book that cogently presents current feminist views. The book represents a sound reference for psychiatrists who seek to understand feminist thinking.

NANCY A. DURANT, M.D.
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Becoming Female: Perspectives on Development, *edited by Claire B. Kopp with Martha Kirkpatrick. New York, N.Y., Plenum Press, 1979, 461 pp., $25.00.*

If the research data reported in this book about becoming female and becoming male were tabulated, it seems to me that one would find an equal amount of information about the development of women and men. Thus the subtitle, *Perspectives on Development*, with the additional words female and male, aptly summarizes its contents. One of the greatest

strengths of the book is its fine review of some areas of research literature, particularly of the past 10 years, on issues of importance in male and female development.

The book is divided into four sections: Identity and the Immediate Environment, Characteristics, The Larger Environment, and Biological Considerations. There are 24 contributors representing 10 professional disciplines. There are 8 psychologists, 2 or 3 educators, sociologists, pediatricians, experts in women's studies, historians, and 1 expert each in public health, child psychiatry, anthropology, and genetics. The uniqueness in perspective of experts from such widely differing disciplines is reflected in each person's scholarly approach to the topics. In some instances, these differences in scientific methodology may pose problems for the reader, who must change to a different cognitive orientation for each author. On the other hand, the reader might find that these variations lend richness to the book.

An intriguing aspect of the book is the blend between some authors' professional expertise and their personal identification with the topic. This blend shapes the presentation. One example is the chapter "Growing Up with a Physical Handicap," which is a highly personal autobiography by Shirley Sargent, a historian, who developed the physically and psychologically painful dystonia musculorum deformans during childhood. Another example is Gloria Powell's chapter "Growing Up Black and Female," which crosses the border back and forth between a sociological, historical approach to the Afro-American woman as black nanny, black mammy, beast of burden, prostitute, and the wicked witch and a personal commentary about the intimate meaning of being black for the author. Dr. Powell demonstrates that the interaction between men and women can have a mutually destructive effect.

The book opens with a remarkably clear articulation of the development of identity by Phyllis Katz. She describes the varying psychological theories of sex role acquisition, including the effects of direct reinforcement, modeling processes, identification (anaclitic and defensive), and, finally, the cognitive developmental view in which the child recognizes himself or herself as girl or boy. Dr. Katz then analyzes the problems inherent in these theoretical constructs and offers an alternative theoretical model of three levels of sex roles. Such an analytic approach to a topic with a comprehensive review of the research literature is most evident in the articles by the psychologists.

I thought the chapter titled "Sex-Related Differences in Spacial Ability: A Developmental Psychological View" by Lauren Harris an exceptionally fine presentation about verbal-linguistic ability as well as visual-spacial ability. Dr. Harris examined these topics from viewpoints ranging from those in the social experiential literature to those in the biochemical genetic literature. The thoroughness with which he reviews the literature is indicated by the length of the chapter (48 pages) and the extensive bibliography.

The chapter "Effects of Observed Violence on Females: A Critical Review" is similar in its level of analysis of the problem, lengthy bibliography, and the fact that it poses questions rather than answers. Susan Franzblau points out that although the television female symbolizes altruism, she also signifies passivity and, often, victimization. Although the television male plays the violent perpetrator and plays him successfully, he also represents competence and power. In the real world, the primary victims of males are males, not females. The TV portrayal of female victimization is a distortion of the real world, adding even more confusion and ambivalence to the already murky picture that females have of their role responsibilities. The author concludes with a theory that the effect of television on females may be to activate pro-social responses that increase with age.

In addition to the aforementioned authors, an attitudinal approach that stresses the continuing need for the development of hypotheses and carefully designed research is demonstrated in the presentation of some individual authors' research projects. Examples include "The Role Music Plays in Adolescent Development" and "The Role of Laughter and Humor in Growing Up Female." The other articles serve an overview function and include the following topics: father-daughter and mother-daughter relationships, female delinquency, play of girls (and boys), women geniuses, cross-cultural perspectives, genetics, clothing, physical growth in adolescence, and nutrition.

A poignant, thought-provoking commentary about these current perspectives on human development are the brief excerpts, mainly by writers from the latter half of the nineteenth century, that are given at the beginning of each chapter. These Victorian quotations reminded me of probably the most famous Victorian story of all, about a girl named Alice who learns about a new world through following a male rabbit. Alice tries constantly to control her growth and the impact of her behavior on others. In her answer to the blue caterpillar's question, "Who are you?" Alice comments that she knew who she was when she got up in the morning but she can no longer understand herself since she is constantly changing. She pointedly remarks that the caterpillar will understand her once he has also had to undergo a metamorphosis. Finally, she questions whether he, even now, knows who he is. The continued popularity of *Alice in Wonderland* reflects the fact that each of us becomes puzzled and confused at varying times in our lives with the problems of controlling growth and development, our social interactions, and differentiating the real from the imaginary. This book offers a series of readings placing these problems of women and men in contemporary social context.

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Working with Dreams, *by Montague Ullman, M.D., and Nan Zimmerman. New York, N.Y., Delacorte Press/Eleanor Friede, 1979, 323 pp., $10.00.*

Working with Dreams is written for the lay reader who is interested in the interpretation of dreams and the use of dreams in daily life.

The book is essentially divided into two sections. The first is a remarkably clear, concise, and easily understood exposition of contemporary theories of the phenomenon of dreaming, its physiological correlates, and methods of interpretation.

In the first section Dr. Ullman presents a lucid and detailed comparison of the differing perspectives of Freud, of Jung, and of himself. He compares and contrasts these views in relationship to the theoretical aspects of dreaming and the clinical interpretation of dreams. Although he emphasizes his differences with Freud and his greater sympathy with Jung, an examination of the differences seems to reveal a continuing allegiance to Freud.

The second half of the book, aside from a chapter on precognitive and telepathic dreaming, is largely a description of the way Ullman and his coauthor, Nan Zimmerman, have organized dream study groups in which members present their dreams for "group analysis."

This method is indeed useful, as I know from my experience with the use of actual dreams of the patients of candidates in a psychoanalytic seminar. The clinician who offers a dream from a patient is often rewarded by a wealth of relevant associative material from his or her colleagues. Nevertheless, I have serious reservations about the function of the dream study groups that the authors describe. First of all I am not convinced that expertise is as unessential to useful dream understanding as they seem to suggest. The use of dreams in such groups as the "dream people" of Malaya described by Noone and Holman (1) is entirely different: ritual formulas are used in the interpretation of the dream, adding a measure of safety that Ullman's groups, with their more personal and more idiosyncratic interpretations, do not provide. Furthermore, there seems to be a degree of distress initiated in some individuals in Ullman and Zimmerman's groups, which, although passed over rather lightly, I found of concern. Finally, it is not at all clear, as the authors themselves suggest, whether it is the beneficial results of the communal dream analysis or of the more traditional group process that holds the group together. I feel too little attention is paid to the latter element, and I believe this in itself constitutes a danger.

I suppose that my basic concern with the book's second section centers about what Ullman and Zimmerman enthusiastically espouse, namely, a lay approach to the use of dreams to illuminate and guide our daily lives. I, too, believe all people should pay attention to their dreams as a wonderful and creative aspect of their experience. This, however, is different from a formal study group in which even the expert, which Dr. Ullman certainly is, insists on denying his experience and expertise and refuses a role as leader. The object of this work is said to be non-"clinical"; yet the process itself, as with so many other projects in which so-called "elitism" or "professionalism" is decried, exists in a borderland fraught with risk.

REFERENCE

1. Noone R, Holman D: In Search of Dream People. New York, William Morrow & Co, 1972

JAMES C. SKINNER, M.D.
Boston, Mass.

The Broken Connection, *by Robert Jay Lifton. New York, N.Y., Simon and Schuster, 1979, 466 pp., $15.95.*

Continuing in Freud's tradition of trying to explicate clinical data, Robert Lifton, eminent psychiatrist and author, essays a new psychology based on mankind's symbolizing of the life cycle. In so doing he hopes to obtain a new paradigm. Lifton seeks "general principles concerning death imagery and struggles for continuity." The book is divided into prologue, parts one, two, and three, epilogue, and appendix. It is heavily annotated and tightly written.

In the eight chapters of part one Lifton compares various concepts of death and immortality. He concludes that it would be better to speak of a "sense of continuity" than a "sense of immortality." He wishes to trade the concept of "instincts" for what he calls "the inchoate image." This latter term he defines as "man's innate plan as a cultural animal." Such a change would replace the concept of "castration anxiety" with a more inclusive term, "death anxiety." Death images begin at birth and continue to be experienced throughout the life cycle. Images of death equivalents include such things as images of separation, disintegration, and stasis. Each of these images of death has an equivalent counterpart associated with vitality. Thus connection is associated with separation, integrity with disintegration, and movement with stasis. Lifton ably reviews the traditional psychosexual stages of development and reinterprets the developmental tasks and experiences in terms of his new paradigm. With an Eriksonian flair, he continues this into adolescence and adulthood. His chapter on "Culture and Connection" is particularly lyrical. He expresses here his belief that death equivalents and life parameters have a biological origin in the inchoate imagery but are embedded in cultural symbolization. He demonstrates this by describing funerary customs among the traditional and more modern Japanese and compares these with modern American customs. Buddhist tradition is compared with the Judeo-Christian.

In the 10 chapters of part two of the book, Lifton applies his outlined system to concepts of fundamental emotions. He also extends this to the neuroses, schizophrenia, and the phenomenon of suicide. This was the most interesting part of the book to me, and I think it will be to any practicing psychotherapist. Chapter 11, titled "Guilt," is a masterpiece. Lifton observes,

> The pragmatic-secular-optimistic constellation characteristic of much of American life makes the experience of guilt virtually a sin. And this in turn contributes to the susceptibility of young Americans to movements demanding that they wallow in guilt. In any case, guilt has acquired a not entirely undeserved bad name.

Lifton compares the guilt of two survivors of extreme situations, a Viet Nam veteran who had been at My Lai but who did not fire his gun and a "zealot saint" of Hiroshima. Lifton is at his finest in reevaluating some of the classical Freudian cases, redefining schizophrenia, and reexamining the Buffalo Creek disaster and the Hiroshima atomic bombing. His own work with survivors of these latter two horrors permits him to speak with great authority. There are many useful data on survivor trauma for the practicing therapist. One chapter is devoted to Yukio Mishima, a serious contender for the Nobel Prize in literature who dramatically committed seppuku in 1975. Lifton uses him as a case history to illustrate his thesis. The attempt is convincing.

In the 5 chapters of part three Lifton examines "broader historical phenomena around the theme of death and continuity." He points out our sense of awareness that we have weaponry which could totally wipe out mankind and postulates a "psychic numbing." It is as if all people were beginning to act like victims. He makes a strong case by reviewing the impact of the continual threat of death on traditional historical victims. This worldwide psychic numbing has resulted in the "ultimate contemporary deformation—a condition we may call nuclearism." Modern man attempts to deal with his death anxiety regarding nuclear weapons by seeing salva-

tion as well as ultimate destruction in nuclear weapons. Ultimate salvation includes such things as the uses of nuclear power to solve the energy crisis and hopes for nuclear technology that will revolutionize modern medicine.

A four-part appendix concludes the book. Appendix B, "Death Imagery and Psychosomatic and Character Disorder," is of further interest to the clinician. Lifton extensively quotes a 1963 article by Elliot Luby, who sees Lifton's work as "very compatible with our paradigm." Appendix C, which includes historical notes on schizophrenia and death, is also thought-provoking.

This is a brilliantly written work of scholarship. Despite its being well written, I found the book difficult to read (death anxiety?). About halfway through the book I noticed that the color of its cover, by accident or by design, reminded me of the vestments and artifacts traditionally used in the requiem mass in the Roman Catholic Church.

In summary, *The Broken Connection* is a seminal work that will probably be referred to for some years to come. Whether or not it will establish a new paradigm will depend, I think, on Lifton's further working his thesis. I highly recommend the book.

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Annual Progress in Child Psychiatry and Child Development 1979, *edited by Stella Chess, M.D., and Alexander Thomas, M.D. New York, N.Y., Brunner/Mazel, 1979, 690 pp., $20.00.*

This is the eleventh year of the *Annual Progress in Child Psychiatry and Child Development*. It continues the labor of love undertaken by its editors over a decade ago with their devotion to children and their recruitment of others to their cause. Each volume has changed as the emphasis of the professional's interest in children has shifted from year to year. The editors have made a significant contribution during these years by permitting their selection of articles to reflect the changing interest of professionals as well as attempting to identify significant contributions to the ever-expanding scientific literature about children. Thus an examination of the current volume not only exposes the reader to a collection of 37 articles in 11 major areas but also serves as a signpost of where we are now, where the field seems to be going, and where some of the inadequacies are in our overall efforts to address ourselves to the problems of children.

The areas of interest include infancy studies, developmental issues, language cognition, temperament studies, family studies, the hyperactive child, mental retardation, clinical issues, delivery of services, and adolescence. There is a heavy investment in the presentation of developmental issues, reflecting our growing preoccupation with the nature of the child and the need for reexamination of concepts in the light of the inadequacies of our efforts to identify childhood disorders. The content of other articles appears to be related to the intensified interest in early childhood development by professionals from the disciplines of psychology and education. The transition in content of past and present volumes appears to be determined as much by this newly developing unity among professional disciplines as by shifting interests. A reading of the current volume compared with some of the earlier ones shows this change extending to language use as well as techniques of research.

A number of the articles warrant special mention, but by so doing I do not wish to take away from the general level of excellence among the others. The best of the articles appear to be those clearly identified as reviews of a particular subject. Important issues are raised and historical presentations of the subject are developed so that the reader can use this volume as an effective instrument for reference or review of such subjects as neonatal assessment procedures, concepts of human development, nonverbal communication, hyperactivity, childhood depression, anorexia nervosa (the bibliography is excellent), day care, and adolescent theory.

With the limitations of a book made up of the writings of many different authors with different styles, points of reference, and philosophies, the 1979 volume of *Annual Progress in Child Psychiatry and Child Development* is a contribution to all of us interested in children.

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New York, N.Y.

Theoretical Criminology, by George B. Vold, 2nd ed., *prepared by Thomas J. Bernard. New York, N.Y., Oxford University Press, 1979, 422 pp., $14.95.*

Preoccupation with the cause of crime has been the bête noir of sociologists, psychiatrists, physiologists, and penologists as well as judges, legislators, and the concerned citizen. The tremendous amount of thinking and writing on this intriguing subject for almost three centuries has not yet yielded a satisfactory, unitary theory; therefore, treatment of criminals has been equally unsatisfactory. Professor Vold, whose work has been posthumously updated and edited by sociologist Thomas Bernard and aided by additions necessitated by new researches since the original book was published in 1958, surveyed the chaotic scene objectively and deftly.

Professor Vold, who died in 1967, was held in high esteem by sociologists and criminologists. He summarized the unwieldy mass of information and theory on crime causation with simplicity and honesty, placing psychological, biological, and social theories in perspective. His book is a pleasure to read; its treatment of that cumbersome, complex group of behaviors, united only in being nominated by law as "crime," is a model of clarity. Vold offers no dogma, no final solutions, no priorities, only a measured exposition of theories that never seem to cover all facts surrounding man's perpetual wrongdoing.

The early chapters outline the various schools. The classical school (Beccaria), which advocated "equal punishment" for "equal crime," was based on the theory that a criminal could make a rational choice of conduct. The anthropological school (Lombroso, Goring, Hooton) held structural differences (dysplasias) responsible for crime. The psychological school believed that mental deficiency was basic for crime. The psychiatric theory held that personality distortions and "psychopathy" underlay criminal action. The more recent psychoanalytic, dynamic interpretation of crime by many authors postulated an "acting-out" of neurotic character distortions. In general, Vold held that biologic-psychologic explanations have limited usefulness.

The author examines the economic theories of crime cau-

sation, e.g., "poverty causes crime" and the more recent ecological theories positing that "invading and retreating cultures," clashing in a spreading city generate conflict that eventuates in crime, especially among young people. The work of the Chicago school of sociologists is accented; this school held that conditions of life in a city with changing ecology form criminal patterns. Following this, the author discusses the anomie tradition of Durkheim and Merton, who held that a breakdown in regulation allows and even encourages members of the society to achieve their goals outside the law, as in embezzlement, fraud, and cheating on one's income tax, the so-called white-collar crimes.

The discussion then moves to criminal action as "learned behavior" (Lindesmith, Sutherland), according to which the accent is on community influence rather than individual deviance or abnormality of personality. Learning by association, i.e., Sutherland's differential association theory, was, according to Vold, "an exciting episode in criminological thinking," which has yielded during the recent past to the interactionist (Mead) and group conflict (Vold) theories of crime. The former holds that what social situations mean to the individual is vital in explaining his or her behavior, and the latter insists that there is a "shifting . . . equilibrium of opposing group interests in society" and that these "endless interactions of moves and counter-moves" become the essential element that gives rise to crime. The law-abiding group, through the legal process, forms the law and defines crime, while other subgroups conflict with the majority position to attain their own satisfactions.

The group conflict hypothesis was Vold's own development—"normal antagonisms and conflicts of human groups"—represented in the struggle for political power and concretized in cultural institutions such as the law. However, the author makes it clear that the group conflict theory holds chiefly where criminal acts "flow from the collision of . . . in-groups" and out-groups. In other words, "The enactment and enforcement of laws . . . [is] the result of the conflict and compromise process as each group struggles to enhance its own position." A contemporary development of the conflict theory entails a discussion of Marxist philosophy of political organization, whether laws are formed to protect a privileged class ("criminal law is the tool of the politically dominant") or a consensus of an enlightened electorate, as the classical school insisted.

The essential problem that Vold and his editor faced in this work is whether a criminological theory can be evolved on the basis of social and/or cultural research, which tends to be "divided between meaningless specifics . . . and broad generalizations." In his summary Vold concludes that no one theory explains the wide diversity of misbehavior that is called crime until more exact definitions and measurements of social pressures on individuals can be devised.

For the psychiatrist who has struggled, as I have, with criminal action as psychologically determined, it is arresting and stimulating to consider the broader canvas of social theories of crime. The book is devoted to theory, whose importance is its foundation for treatment, which we must admit has been anything but successful over the decades. Professor Vold's book, with the additions by his followers, is a necessary tranquilizer for the forensic psychiatrist who is afloat in the squally sea of criminality.

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New Developments in the Use of the MMPI, *edited by James N. Butcher. Minneapolis, Minn., University of Minnesota Press, 1979, 390 pp., $27.50.*

This volume is a collection of papers by authors with special expertise in the use of the MMPI in the various areas discussed. Many of these papers were presented at a 1979 symposium on recent developments in the use of the MMPI in St. Petersburg, Fla. The 10 chapters cover a wide range of issues, starting with a very technical chapter by Mary Koss, who discusses MMPI item content, issues, and problems. This is followed by chapters on the clinical use of the MMPI, including one on the evaluation of personality changes with the aging process by Gynther, "Ethnicity and Personality," also by Gynther, "Use of the MMPI with Medical Patients" by David Osborne, "Use of the MMPI in Personnel Selection" by James Butcher, and "MMPI in the Evaluation of Treatment Effects" by Steven Hollon and Monica Mandell. There are two chapters on the use of the MMPI in the classification of criminal offenders, one by Edwin Megargee and one by Raymond Fowler. These discuss the use of a computerized interpretation of the MMPI in correctional decisions.

The last few chapters are primarily research oriented, one by James Butcher and Lee Anna Clark on recent trends in cross-cultural MMPI research, and a very interesting chapter by David Lachar and James Sharp on the "Use of Parents' MMPIs in the Research and Evaluation of Children." Finally, there is an excellent bibliography of MMPI research organized by such topics as affective disorders and related studies, organicity and intellectual functioning, and treatment outcome.

The generally high level of the material presented in this volume is, I believe, a tribute to Dr. Butcher's careful editing and his encyclopedic knowledge of the field. Although much of the material contained in the volume would be of interest primarily to individuals using the MMPI in research activities, there is important, clinically useful information in several of the chapters that will be valuable for clinicians working in the areas of corrections, in general medical settings where assessment of the personality problems of patients may be of importance, and for clinicians using psychotropic medications in treatment and in clinical trials.

Although this volume would not be recommended for inclusion in every psychiatrist's library, it certainly should be available for reference in mental health centers, in psychiatric hospitals, and in general hospitals with psychiatric units and active psychiatric-liaison services.

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Existential Foundations of Medicine and Psychology, *by Medard Boss; translated from the German by Stephen Conway and Anne Cleaves. New York, N.Y., Jason Aronson, 1979, 297 pp., $20.00.*

This is a difficult book to comprehend, in part because of the unfamiliarity of most American readers with existential philosophy and psychiatry. The paragraphs that follow are intended to present some of its principal contentions and to put them into the perspectives of more familiar schools.

A useful starting point is the existential description of pa-

thology. Every disorder, from a broken leg to schizophrenia, is described from the existential viewpoint in its impact on human freedom, by which is meant one's ability to relate fully to the world or, in Boss's language, to have access to the modes of being in the world. These modes or basic human characteristics include the ability to move through space and time (and not only measurable space and time, but the ability to imagine absent spaces and times), accept uncertainty and mortality, coexist in a shared world, and reach an affective attunement. Descriptions of pathology are descriptions of impingements on these modes, and the therapist's responsibility, whether as surgeon or psychiatrist, is to place himself or herself with the patient in the task of restoring human freedom. In other words, pathology is here described in terms of the *highest* human functions rather than the lowest. It is not that the "lowest" (for example, a metabolic defect) are not important; many, of course, are fatal. The point is that they cannot describe what is human.

The advantages, Boss argues, of retaining human reference points for descriptions of pathology are several. Human reference points direct attention to large-scale impingements on freedom that make reductionistic lesions secondary or symptomatic and their corrective measures ineffective in the life of the patient as a whole. Boss is not against setting fractured legs. What he means is that many fractured legs are symptoms of accidents, which in turn are cries for help of beleaguered individuals. The surgeon must place himself or herself with the patient in the world unless he or she is to be a mere technician who will all the more readily, Boss fears, become an agent of state medicine and lose the great role of healer.

Human reference points make possible a real locating of the patient and therefore of joining him or her in the task of liberation. Every reductionist view, whether the reduction to chemical events or psychic complexes, threatens not only our grasp of the patient's whole situation but also our ability to get with him in its correction. Here the classic illustration is the patient hurried through and out of the university clinic; a rare defect is discovered, but the patient never has the opportunity to voice his chief complaint. The first goal of the existential healer is to see the world from the patient's point of view. This may mean concentration on the broken leg (to which the painful member had reduced the patient's world), but attention cannot as a rule remain there long. Once the acute phase of the illness is over, the healer seeks to share the patient's now enlarged world. It may be there *is* no enlarged world; the concentration on the leg continues, as in some hypochondriases. Or the patient's world may open up to full human freedom and the healer is dismissed, but there may be specific impingements that need attention.

Boss's accounts of phobias are illustrative. Emphasis does not fall on intrapsychic conflicts or environmental conditioning (much as these may shape the choice of the specific phobia) but on the underlying attitudes, for example, narcissistic expectations that make any interruption, breakdown, or intrusion a feared attack on the patient's entitlement always to expect changelessness, safety, even immortality. This is not Boss's language. He writes of the agoraphobic person's inability to tolerate large open spaces because in fact it is from these that "unfamiliar and uncertain things" may enter. The point is the same: the agoraphobic person is accused of having a brittle character or world outlook. Psychoanalytic and behaviorist treatments may be effective, but Boss implies this is because both provide the brittle patient with an ally against uncertainty. The existential claim, which I have seen sustained enough not to dismiss it, is that the same result can be achieved more fully and directly by allying with the patient in his world view and then helping him gradually to modify it.

The existential view is curiously like the medical view. Both see the patient as impinged upon: in the medical view, by bacteria, viruses, cancers; in the existential view, by these and, as well, other people, social systems, etc.

The human reference indicates not only the substrate of disease and the possibility of being with the patient in his situation, it also provides goals of therapeutic work: the definition of full human freedom. The detail in which existentialists do this should be of special interest to psychiatrists who have had to construct treatment goals from disease concepts or the broad strokes of Freud's love and work.

In different words, the book claims there is an existential and not an exclusively physical science foundation to medicine and psychology. An exclusively physical science foundation, Boss argues, leaves us capable of seeing "only causal relationships among isolated objects." An existential foundation recognizes what Boss believes is the reality of the human state: that its occupants are not isolated objects, their relationships can never be fully quantified or causally connected, and that the goal of their being is a "non-objectifiable openness." At this point existential psychiatry, like all the schools, begins to move within the circle of its own words. Let me attempt a translation. In the human sense, I may be closer to my friend a thousand miles away than I am to the floor I walk on, unless perhaps it is the floor of my home. However, a true measurement or objectification of these psychic distances is probably not possible, although they are often the distances we care most about. My humanness consists not only in my capacity to walk that floor, but also to be close to my friend so far away.

These contentions are challenging and not readily dismissed, but it must be said that Boss seldom makes their acceptance easy. The tone of the book is polemical, at times cranky, and repetitious. Freud's metapsychology is as much an albatross for Boss as it is for many analysts; he cannot leave off picking at it, although the accusations he makes (reductionism, reification, nineteenth-century physicalistic analogies, etc.) are well-known to the literature of all the schools, including psychoanalysis itself. Moreover, Boss's presentation of existential work suffers from the same tone. He does not speak well even of the existentialists. Indeed, he cannot speak of many at all. (The American reader will miss any reference to May, Fromm, Rogers, or Kohut.) Most readers, I suspect, will deplore the "only I know" attitude so epidemic on the present sectarian scene.

I agree with Boss that much theorizing along natural science lines handicaps as much as it equips the practitioner, perhaps especially the psychotherapist. He agrees, too, that the gap between observer and observed favored by some perhaps outdated students of natural science at times provides an obstacle to both understanding and treating. On the other hand, and at other times, the gap between observer and observed is serviceable in that it allows for an objectivity toward the self of great value. Further, I believe it is possible to show that objective and quantitative notions, in other parts of this book disapproved of, are bootlegged in for the purpose of making an often useful way of thinking and working into a general psychology. I believe this last is the abiding sin of all the great schools.

Boss's approach, too, is reductionist in a way he does not appear to notice. The many subtle and useful differentiations of the other schools are reduced to a few general statements that do not allow us to appreciate the intellectual and therapeutic value of the differentiations. (I believe this is especially true of his discussions of transference.) Existential writings often have a flat, broad quality, for all their occasional rising to poetry and philosophy, that does not fit well the uneven terrain of actual clinical work. I write this appreciating that Boss's own clinical work probably does not imitate his conceptualizations. It is very hard to write accurately of clinical work.

Finally, one must acknowledge the many values of the existential viewpoint that this book underlines: existential work, more than any other, appreciates the importance of the *future*; we know, but often refuse to acknowledge, our flimsy structure of *causes* (different causes can produce the same psychopathological picture, and the same cause can produce very different pictures). Further, a heightened understanding of *dreams* springs from seeing them as translations of actual human situations, and the description and understanding of *affects* profits greatly from the phenomenological approach.

If reprinted, the book should be carefully proofread.

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Logotherapy in Action, *edited by Joseph B. Fabry, Reuven P. Bulka, and William S. Sahakian. New York, N.Y., Jason Aronson, 1979, 369 pp., $30.00.*

As the title promises, this book is about logotherapy. It is edited and written by followers of Viktor Frankl, who calls himself the "father of logotherapy." Despite the frequent attempts to clarify the nature of logotherapy, there is a peculiar failure to define this special approach to therapy in clinically meaningful terms. Although several contributors attempt to do so, I found myself less than satisfied with their definitions. Logotherapists seem partial to a definition that stresses the term "meaning": "Logotherapy, then, is therapy through meaning (logos)." "Logotherapy is a method to help persons whose life has become empty and meaningless to lead a fuller existence." In addition to defining therapy in these rather restricted terms, logotherapists appear to throw out psychopathology in any usual sense. For them, psychoneurotic or conflict-based illness, psychosis, and all varieties of psychiatrically defined illness are either unimportant or not within the domain of logotherapy. Their preference is to address what they perceive to be an epidemic of meaninglessness that pervades contemporary American culture (apparently more than it does European culture). The task of the logotherapist is to show the client the significant meaning in his or her life no matter how disadvantageous the life situation may be. Furthermore, there appears to be no category of human suffering (as opposed to psychiatric illness) that cannot be addressed through this form of therapy.

It would be easy to dismiss this book were it not for the fact that it is clearly the product of well-intentioned individuals with a variety of backgrounds in counseling psychology training (only Frankl and Gerz have medical training). Although the editors' contention that logotherapy is a widely applied form of psychotherapy is inaccurate with reference to psychiatric outpatient settings, it is undoubtedly being applied by counselors in a number of settings. Unfortunately, no data are provided to allow for a judgment about the extent of its use. From what I could ascertain from the writings of various contributors, it would not surprise me if some individuals with a variety of neurotic disorders were helped by the kind of interventions described in the text. Both of the major technical maneuvers of logotherapy, paradoxical intention and dereflection, are based on the assumption that the will to meaning in life allows individuals to exercise control of their condition by modification of their attitudes, even those toward neurotic symptoms. Gerz used paradoxical intention with a patient incapacitated by the fear of having a heart attack by repeatedly encouraging the patient to make himself have a heart attack. The patient responded by giving up his fear in three sessions. Dereflection was applied in one example to an impotent man who was told that the problem was not serious but he must absolutely restrain himself from intercourse and tell his spouse of the doctor's orders. Released from the demands of sexuality the patient became sexual and potent once again.

These techniques and examples are not totally unfamiliar to clinicians. Psychoanalytically oriented therapists are aware of the occasional need for manipulations, even in analysis. The difference here is that these very short-term interventions are characterized by a lack of attention to the past, the inner emotional life, and, most of all, to the transference. The philosophical core of logotherapy is in fact present in part in psychoanalysis; it is necessary for analysands to accept the fact that the exploration of their symptomatic manifestations, dreams, and affects will add new meaning to their lives. Furthermore, the capacity to tolerate painful affects related to past losses, disappointments, and mistakes is a requirement of a successful treatment.

At this point the similarities cease and logotherapy becomes a form of "wild analysis" in which the transference power of the therapist is used without any apparent awareness by therapist or patient. This schism between logotherapy and clinical psychiatry and psychoanalysis is worrisome to say the least. One can only hope that those individuals with serious emotional distress and needs manage to find their way to therapists whose training permits them to approach their problems in a way that identifies and lessens the influence of pathological trends in the personality.

Finally, it should be noted that the insistence by almost all of the book's contributors that the major "collective neurosis of our time is spiritual" is not nearly as innocent and naïve an assertion as it might appear. This form of externalization has had its appeal to pessimistic social critics, such as Lasch (1), in whose work it reached a peak of expression of the confusion of internal and external problems. Although the insistence that psychiatric problems and suffering result from an attitudinal defect caused by "moral decay" in society may have important repercussions in the hands of a widely read social critic, the effect of such an attitude on a psychotherapist is directly destructive of that therapist's capacity for empathic understanding and for the amelioration of individual suffering through interpretation.

This volume illustrates the unfortunate effect of abandoning the medical model of evaluation and treatment of emotionally based disorders. I consider the counseling psychologists following this system of therapy to be both misled and superficially trained. This new volume will represent a definite disappointment to those who first encountered Frankl in

his moving book *From Death Camp to Existentialism* (2). From that early book it would have been impossible to predict that Frankl and his followers would derive a "supraordinate" theory of therapy that discards an entire corpus of theory and practice in psychoanalysis and psychiatry. That Frankl could find meaning in his experience within the concentration camps was and is moving. Retrospectively, however, his development of logotherapy leads me to wonder whether his assertion that survival was based on finding meaning in experience does not represent an inaccurate, narcissistically based assumption. Perhaps it is chance alone that determines survival in a randomly destructive environment. Psychiatric readers of this book will find little to reflect on that might enhance their clinical effectiveness or stimulate their intellectual processes. Unfortunately, with its few techniques, concepts, and ideas, it might appeal to some individuals whose training is nonmedical as a simplified system of therapy. Patients treated by logotherapy will have received an infusion of a philosophical stance that, while possibly helpful, will deprive them of insight into the nature of their suffering.

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Alcohol and Alcoholism, *by the Special Committee of the Royal College of Psychiatrists. New York, N.Y., Free Press (Macmillan Publishing Co.), 1979, 155 pp., $10.95.*

The Special Committee of the Royal College of Psychiatrists is chaired by Dr. Griffith Edwards, who is well known by alcohologists in the United States. The remainder of the committee is composed of distinguished members of the College.

This report was written in response to the awareness of how serious a problem alcoholism is becoming in the United Kingdom. Alcohol consumption per capita rose 87% from 1950 to 1976, and "guilt for offenses of drunkenness" per population doubled. The report is directed to a broad audience, not just a professional audience, and undoubtedly it is hoped that some of those reached will be decision makers in government and health policy councils. Because this is the goal of the report, it is not intended to be a scientific or technical monograph. There are no references to supportive literature, but a bibliography is provided for further reading. Anyone familiar with the field will not find new information. It is not a book on how to do it. However, it is broadly educational for those not so familiar, and that is the purpose of the report. Because it is about the drinking problems of England and Wales, it is an excellent review for anyone interested in cross-cultural studies of alcohol use and misuse.

"Alcoholism," as used in this report, embraces not only the dependence syndrome but also alcohol-induced impairment. I was therefore surprised to learn that there are "only" 300,000 to 500,000 people so inflicted in England and Wales, which have a population one-fourth that of the United States, where we estimate a prevalence of more than 10,000,000 when this more inclusive definition is used.

Although they make passing reference to "Dr. Anstie's limit" (that of a nineteenth-century British physician) of three ounces of whiskey or half a bottle of table wine or two pints of beer a day to be safe of ill affects of alcohol, the committee members seem less conservative. They warn that daily consumption of half a bottle of spirits or eight pints of beer creates "a very considerable risk of acquiring dependence, and this statement should not be taken as meaning that lower levels are necessarily safe." They estimate that two bottles of wine daily may put a pregnant woman into the danger zone for having a child with the fetal alcohol syndrome. They say that four pints of beer daily or "four doubles of spirits" or a bottle of wine "constitute reasonable guidelines for the upper limit of drinking." Although convincing data are lacking on this subject, a report like this intended for the public might have been wiser to have stayed with "Dr. Anstie's limits."

The general statements about treatment efficacy suggest that about 30% of alcoholics will be abstinent a year after intervention, 30% will be less impaired but still drinking, and 40% will be unchanged. This closely parallels the "one-third rule" usually found in follow-up studies in the United States. The committee has the same treatment controversies we do: inpatient versus outpatient, intensive versus minimal treatment, generalist versus specialist, abstinence versus "return to normal drinking," psychotherapy versus behavioral therapy, and so forth.

The recommendations made are sound—primarily that public awareness be greatly increased so that social expectations about alcohol consumption will change. The committee believes that a drop (or at least no further rise) in per capita consumption is an important preventive strategy, something being debated in the United States. They oppose relaxation of license controls and advocate better coordination among government agencies that have some involvement with alcohol and health. They suggest that liquor advertising should be studied and perhaps curtailed, that industry and business should reduce pressures on employees that encourage drinking, and that health professionals should be better educated about alcoholism.

This interesting report is well thought out and clearly written. It is to be hoped it has a beneficial impact on the citizens of England and Wales.

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Drug and Alcohol Abuse: A Clinical Guide to Diagnosis and Treatment, *by Marc A. Schuckit, M.D. New York, N.Y., Plenum Medical Book Co., 1979, 193 pp., $16.95.*

It must take a great deal of courage to write yet another book on the diagnosis and treatment of drug and alcohol abuse. In the past two decades we have seen innumerable texts on the subject written from every possible perspective. Some have been good, some bad, and many have been mediocre. The current volume tends toward the good end of the spectrum.

Dr. Schuckit has produced a concise, well-organized, and readable volume. It admirably fulfills his two-fold goal of presenting a volume that can serve as a broad overview of

the common drugs of abuse (including alcohol) as well as providing rapidly accessible data on emergency treatment procedures. Superfluous material is virtually nonexistent. One gets a lot of meat and very little fat. Appropriately selected references can guide the motivated student toward greater depth of knowledge in any area of interest.

The author should also be commended for including problems of alcohol abuse along with other substance abuse. The current tendency to treat alcohol abuse as though it were an entirely separate disease or illness from that of other drug abuse is more political than scientific. A single handbook that comfortably treats the spectrum of drugs of abuse is valuable.

Each chapter treats a major category or class of drugs with abuse potential. Within each chapter sections on pharmacology, epidemiology, and diagnosis are followed by a discussion of emergency problems and treatment characteristic of that class. Special chapters on multi-drug use, over-the-counter drugs, and rehabilitation add breadth and depth to the book. Of special interest to the practicing physician is the well-written chapter on "Emergency Problems: A Rapid Overview," which establishes a logical framework for rapid clinical assessment of drug intoxication when the nature of the drug taken is unknown.

I have a few carping criticisms. The author's definitions of drugs of abuse and of drug abuse are unsophisticated. Part of the problem, of course, lies in the fact that it is not just the pharmacology of the drug that makes it a drug of abuse but, rather, human behavior. The social and psychosocial patterns of use produce abuse. The permutations and combinations of possible behavior patterns and pharmacological interreactions are so numerous as to virtually negate any possibility of clear, concise, and meaningful general definitions.

It is disturbing that the author made no attempt to tie his work into the definitional framework of either *DSM-III* or *ICD-9-CM*. The addition of one or another of these classification systems (they are for all intents and purposes virtually identical) would have added a useful dimension to an otherwise excellent volume.

In summary, this book should appeal to a wide audience. Physicians and others involved in the treatment of abusers—especially in emergency or crises settings—will find it an eminently practical tool.

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Handbook of Child and Adolescent Psychiatric Emergencies, *by Gordon K. Farley, M.D., Lloyd O. Eckhardt, M.D., and Frederick B. Hebert, M.D. Garden City, N.Y., Medical Examination Publishing Co. (Excerpta Medica), 1979, 185 pp., $12.00 (paper).*

This handbook is designed to provide mental health workers with help when confronting a child psychiatric emergency. The first three sections of the book are extremely brief, exhorting the clinician to see the opportunities and pitfalls involved when a child is seen under emergency conditions. In addition, they provide a comprehensive outline of a child psychiatric assessment.

The fourth section constitutes almost the entire body of the book. It is titled "Specific Child Psychiatric Emergencies." Since only a few situations in child psychiatry are truly emergencies, this section is in fact an abbreviated textbook of child, adolescent, and (in some cases) adult psychiatry. It discusses acute drug abuse, adolescent schizophrenia, anorexia nervosa, child abuse, and childhood psychoses. By summarizing many classical articles in one place, this section was useful to me as a succinct review of a large body of material, highlighting and refreshing my memory on many details in the assessment and treatment of numerous psychopathological conditions. In that context, the absence of new ideas or of inspired writing seemed less bothersome.

As a handbook of child psychiatric emergencies, the text does not distinguish true emergency situations from those which are less so. The alphabetic arrangement of material contributes to this. It might have been useful to organize the true emergency situations into one brief chapter.

There is a spotty quality to this book: some of its sections are exceedingly well done, including those on runaway children and school phobias, while others, including that on adolescent schizophrenia, represent a simple abridging of sections of the *Comprehensive Textbook of Psychiatry* (1). While this does not detract from the book's useful nature as a refresher, it does highlight the fact that the text could have done more.

After the long section on specific disorders of childhood, the section on psychopharmacology seems repetitious. It could be useful to a clinician who wants to look up a dosage or a particular pharmacologic treatment for child psychiatric conditions. However, I found it the least useful part of the book; it would have been better if it had been more tightly integrated into the previous section.

In conclusion, the *Handbook of Child and Adolescent Psychiatric Emergencies* represents a useful review of, in some cases, overview of child and adolescent psychiatry, with a particular emphasis on more or less acute situations and the emergency situations that sometimes arise from them. The book refreshed my memory, organized a vast body of material into a useful summarized whole, selecting only those pieces of work the authors felt were most pertinent. On the whole, I think the book is a succinct and useful addition to existing literature in this field.

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The Psychobiology of the Depressive Disorders: Implications for the Effects of Stress, *edited by Richard A. Depue. New York, N.Y., Academic Press (Harcourt Brace Jovanovich), 1979, 437 pp., $29.50.*

This is a delightful volume that reviews the recent advances in genetic and biological aspects of affective disorders and recent research in life events and points toward areas where knowledge from these disciplines interface. The major thesis involves the concept of vulnerability to disease, a concept that focuses on environmental-biological interactions in the genesis of psychiatric disorders.

The volume has five major sections. The conceptual over-

view of stress and disease presents the major thesis. The second section consists of four chapters, three dealing with classification of depression and one presenting an overview of issues in measurement of stressful life events. The review of unipolar-bipolar distinctions is excellent; it proposes a thoughtful classification based on this dichotomy. A chapter on the endogenous-neurotic distinction is quite comprehensive and well referenced and discusses the effects of antidepressant medication and ECT. The relation of anxiety to depressive states has an excellent review of this topic, and the chapter on measurement of stressful life events presents a format for issues that are elaborated on in subsequent chapters.

The third section discusses the interaction of stress and biological variables in depression. The five chapters in this section concern norepinephrine metabolism in animals and in depressed patients. Neuroendocrine studies in depression, with emphasis on cortisol, are reviewed with regard to diagnosis, catecholamine metabolism, and stress. The chapter on genetic influences on catecholamine metabolism is concerned for the most part with animal studies, although the family studies of catecholamine metabolizing enzymes are covered. The relationships of neurophysiology to stress and depression are discussed.

The fourth section reviews psychosocial aspects of stress and depression. The six chapters in this section discuss studies of life events and depression with emphasis on the studies done in New Haven and London. Psychological theories of depression (reinforcement, self-esteem, and learned helplessness) are discussed in separate chapters. This section concludes with a chapter reviewing cognitive and behavioral factors in the regulation of stress.

The fifth section consists of two chapters that present models for integrating stress-biology interactions. Strategies for future research in depression, including studying children of depressive parents, are discussed.

Although this is a multiauthored volume, the editor has created a good sense of continuity from chapter to chapter and from section to section. The chapters are usually reviews and overviews of topical areas, and particular research studies are often highlighted. Each chapter is well referenced, although for the most part the references are only through 1977.

One problem with any recent book on depression is how to integrate previous studies with the new *DSM-III* nomenclature. *DSM-III* is not addressed in this volume. However, the topics covered are basic to our understanding of psychopathology and provide a useful background as clinicians become familiar with *DSM-III*. This book has considerable information and should be of value to both researchers in depression and clinicians who wish to review recent advances in depression research.

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Malpractice: A Guide for Mental Health Professionals, *by Ronald Jay Cohen. New York, N.Y., Free Press (Macmillan Publishing Co.), 1979, 320 pp., $13.95.*

This is an ambitious book by a legal amateur (the author is a psychologist). Although I am an adherent to the maxim that the law is too important to be left entirely to lawyers, I would observe that there are limits to the strictly legal contributions that nonlawyers can make in the law-behavioral sciences dichotomy. A glaring example of the legal naiveté of the author is the statement on page 48, "Whereas criminal cases must be tried by a jury, civil cases can be tried by a jury or judge." Criminal cases of course are regularly tried by a judge when the jury is waived.

There are repeated disclaimers that the book is authoritative, such as, "None of the case material should be construed as constituting a definitive statement of the law." One hundred eight pages are devoted to "vignettes" of illustrative cases. The author indicates that some of these cases are "sketchy." In many, in my opinion, they are worse than that—"simplistic," "distorted," and "irrelevant" are adjectives that occurred to me as I reviewed them. I do not understand, for example, what *In Re Gault* has to do with malpractice.

The author is to be applauded for the enormous and difficult undertaking this book must have entailed. The subject is thorny and complex and does not lend itself to easy generalizations. Quite the opposite: case law in this area is diverse, inconsistent, and often quite idiosyncratic.

I do not mean to convey the message that this book is without *any* merit. The mental health professional who is experienced in the workings of the legal system will learn from this book, indeed will learn a great deal. However, the book should be read with a healthy application of skepticism, given its lack of legal authority, which the author himself acknowledges. If you should get caught up in a malpractice action, the book will help you to understand the complexity of the area, but it is no substitute (nor is it intended to be) for either your insurance carrier or yourself getting the best malpractice lawyer you can find.

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Three Millennia of Chinese Psychiatry, *by John J. Kao, M.D. New York, N.Y., Institute for Advanced Research in Asian Science and Medicine, 1979, 179 pp., $10.00 (paper).*

The title of this monograph, which was first written as an M.D. thesis at Yale School of Medicine, appears to be misleading in that there is very little that we would find at all familiar until the beginnings of "missionary psychiatry" in the late 1800s. The meat of this book is an account of the author's observations on a trip to the People's Republic of China in 1973, which has been set in historical context.

Although I found the philosophical ideas of the initial section rather difficult to follow, once Dr. Kao starts describing the early beginnings of modern psychiatry, he becomes a lucid, thoughtful, and careful analyst of trends and events in the development of psychiatric knowledge and practice in China. The descriptions of the neglect and cruelty expressed toward the mentally ill are reminiscent of conditions in the United States and Europe before the advent of Dorothea Dix, Pinel, and others who made attempts to develop humane treatment methods. The historical material is plentifully documented through citation of sources. The account of the John Kerr Refuge for the Insane in Canton, of which I had not previously heard, was compelling and fascinating, a monograph within a monograph and worthy of separate publication.

In spite of these early efforts, only a minuscule portion of the total mental health problem in China was attacked until "after the liberation" in 1949, when the new government decided to devote resources to the study and development of psychiatry. As Dr. Kao points out, psychiatric services were part of a plan to provide general medical care in China, where almost none had previously existed, and prodigious efforts were made to train staff and obtain supplies. By edict, modern and traditional forms of medical care were combined and the best taken from each. An important underpinning of the new psychiatric therapy was political—the concept of the illness as the enemy and the enlisting of the efforts of the patient and his peers in defeating it. The cure thus lay within the individual.

Psychiatry and its patients also changed as ancient scourges were overcome and as a social system was developed that met the basic needs of people within a traditional Chinese framework. For example, mental disease due to syphilis disappeared as prostitution and venereal disease were practically eliminated; senile dementia responded to changes that gave the elderly useful roles and continuing relationships with their families.

Unfortunately, Dr. Kao's account does not cover the past seven years in China since his visit, nor does it attempt to review the more modern literature. For example, the thoughts of Chairman Mao are no longer regarded as therapeutic, and "the little red book" no longer accompanies the patient to the group therapy session. Since the Cultural Revolution, which was ending about the time of Dr. Kao's visit, there has been a substantial increase in the valuing of "progress" as defined in Western terms. Intellectuals are no longer expected to submerge their interests and blend with the peasants, with the consequent loss of scientific contributions and energy that might be more productively spent in the arena for which one was trained. When Dr. Kao wrote he was well aware that changes were continuing and inevitable, and one hopes that he will have an opportunity to share updated information.

All in all, this is an impressive piece of work, with much interesting detail, thoughtfully analyzed and documented. It would be of particular interest to those who wish to know more about the broader implications of the medical and psychiatric social-community care system that has evolved in China.

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The Later Papers of Sir Aubrey Lewis, LL.D., D.Sc., M.D. *New York, N.Y., Oxford University Press, 1979, 245 pp., $37.50.*

This book contains 24 papers written by Dr. Aubrey Lewis between the time of his retirement in 1966 and his death in 1975. The author's predominant orientation is historical and descriptive. He traces the historical development of the definitions and classifications of several mental syndromes from the time of the early Greeks to the present. For example, he lucidly traces the historical development of the concepts of such mental syndromes as anxiety, hysteria, paranoia, phobia, and the psychopathic personality. In each of these instances he reviews the varying use of the terms representing these concepts over time in each of several languages. From these data, using his own observations, he attempts through logical arguments to establish a preferred meaning for each term within the general classification of mental disorders. His arguments are cogent and stimulate one to think about the obvious nosological problems in psychiatry, such as the fact that different psychiatrists include differing patients in the same diagnostic category.

Dr. Lewis' paper on hysterias and his paper on phobias are both very succinct historical critiques of the development of these diagnostic syndromes. In three other papers he 1) traces the historical development of the concept of the endogenous-exogenous dichotomy, 2) shows the cultural relativity of the attitude that drug dependence is damaging, and 3) discusses the reciprocal influence of culture on mental disease and of mental disease (and psychiatry) on culture. Some of the 24 papers are concisely written reviews of books by various authors.

In his various papers Sir Lewis poignantly criticizes and puts into his own perspective many of the different psychiatric approaches. In separate papers he critically evaluates the behavioral, the biochemical, and the psychoanalytic points of view. Although at times he fails to state Freud's views correctly, at other times his criticisms of psychoanalysis are apt. In reviewing current drug research he puts it into perspective by comparing and contrasting it with earlier work on general paresis and ECT.

Although Dr. Lewis tends to critically evaluate the ideas and work of others, rather than put forth new ideas or observations of his own, his clear arguments and the breadth of his historical perspective are enjoyable and informative.

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La Jolla, Calif.

Des Fous dans la Ville? Gheel (Belgique) et Sa Thérapie Séculaire, *by Eugeen Roosens; translated by Maddy Buysee, Jacques Dumont, and Eugeen Roosens. Paris, France, Presses Universitaires de France, 1979, 207 pp., no price listed (paper).*

Virtually no psychiatrist is unaffected by the baffling circumstances of Gheel, the Belgian town that for centuries has provided family care for the mentally ill. Many reports of visits to Gheel are available in the literature from the early nineteenth century, when psychiatry acquired the status of an independent branch of medicine. However, these reports have been anecdotal and have tended to raise rather than to answer questions. Therefore, a monograph on Gheel by a Belgian sociologist in a language accessible to many is certainly welcome. A reading of this work clarifies many aspects of the family care provided for the mentally ill at Gheel, but it still leaves many areas obscure.

First of all, the title of the book is slightly misleading. The reference to Gheel's "centuries-old therapy" may lead readers to believe that this is a historical study. "Centuries-old therapy" is used here in the sense of traditional therapy as opposed to what is used currently. Only a few passages scattered here and there refer to the history of Gheel and the bibliography contains almost no historical references. The tradition of Gheel can be traced as far back as 1250, when the mentally ill were brought to Gheel imploring to be healed at the shrine of St. Dymphna, the Irish princess who, attempting to escape from the incestuous advances of her father, ran

away to Gheel, where her father eventually found and killed her. Although the religious emphasis decreased during the French revolution, the number of mentally ill in Gheel increased up until World War II: there were 200 patients around 1800, 400 in 1820, 900 in 1850 (following the closing of the mental hospital of Brussels), 2,000 in 1900, and 3,736 in 1938.

There are now 1,363 patients at Gheel; they are placed in 1,007 foster families (of which 356 have 2 patients, always of the same sex). Two-thirds of the patients are male, one-third are female; 45% are 55 years old and older; one-third are diagnosed as schizophrenic and tend to be withdrawn, and two-thirds are diagnosed as oligophrenic; they are placed in 10 different districts, each staffed by nurses who are always available and who work for the "Etablissement" (the Center for Family Care). This program has been run by the state since the second half of the nineteenth century, and the religious influence is minimal (even the annual procession for St. Dymphna was discontinued 15 years ago). Roughly half of the families who care for patients are rural and half are urban; they are all lower-class families. Their motivation to accept patients is economic rather than charitable. Although in the past there were many instances of exploitation of mental patients by families, this is apparently no longer the case.

The author of this book, a professor of sociology at the Catholic University of Louvain, focuses his study on the life of patients outside the context of the foster family. Toward the end of the book he reports that most of the patients in family care tend to be submissive the first three months, rather rebellious the next three months, and then to develop a more cooperative attitude. Thus the patient's "sick" image is eventually replaced by a "healthy" image.

Most of the chapters of the book deal with the result of a questionnaire distributed to the foster families and with direct observation by some of the author's students. The researchers found that most of the patients are free to walk outside on their own and to receive relatives, although 52% have no visitors at all. Most of the patients are indistinguishable from the other citizens when they are in public. This is in contrast to the past, when patients had to wear uniforms. Criminal behavior and sexual acting out among the patients are virtually nonexistent. The patients can attend movies for children on Sundays, and they spend long hours at the "cafe," which is similar to but not entirely like an American bar; a good part of the leisure time of the villagers evolves around the cafe. The patients are not served liquor there, at least not officially, and their behavior is tolerated through a condescending attitude and a network of community supervision. On the whole, life in Gheel—a town of 30,000 inhabitants—is not very eventful, in spite of the presence of the mental patients.

Should a patient become unmanageable, he or she is immediately taken care of by the nurse of the Etablissement. In fact, the worst fear of the patients is to be returned to the hospital. This happens only in rare instances (i.e., 22 cases in 1976), which is truly remarkable in view of the severe psychotic symptoms of many of the patients (several instances of these are reported in the book) and in view of the limited use of psychopharmacological agents. The book's findings indicate that most of the foster families are skeptical about medications and tend to view the disorder of their foster patients as organic; they do not consider dynamic and interpersonal factors. These families have a clear-cut perception of normal and abnormal behavior (which explains why the patients are never accepted as members of religious congregations) and emphasize the healthy aspects of the patients' personality—in fact, their boarders are never called "patients"; they are called "pensionnaires."

The reader may have many questions, such as, Is the system of care at Gheel unique because of the tradition? If not, could it be repeated somewhere else? How much of its success is due to the social stability of the population—both the foster families and the patients—which is at variance with the instability of the nuclear family of the highly industrialized nations, notably the United States? How does this system compare with similar ones recently instituted in developing countries, notably Africa? How do the results compare with other systems of family care elsewhere?

Much more research is needed to obtain even a partial answer to these and to many other questions, but it is refreshing to see that the obscurity that surrounded the Gheel system has been somewhat clarified. It is notable that this has been accomplished through the Gheel Family Care Research Project sponsored by Leo Srole, an American sociologist, with the support of Columbia University. A historical study of the Gheel system would be even more difficult to accomplish. The present volume may at least encourage others to continue to study this unique system, which anticipated by centuries today's concern with placing mental patients back in the community.

GEORGE MORA, M.D.
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Psychiatric Aspects of Minimal Brain Dysfunction in Adults, *edited by Leopold Bellak, M.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1979, 202 pp., $19.50.*

This is the edited volume of a conference on adult minimal brain dysfunction that was held in the spring of 1978. The conference participants are outstanding clinicians and researchers in this area. Specifically, the 18 contributors present a series of major papers that review the areas of definition, diagnostic aspects, treatment and management (including follow-up studies), family dimensions, and relationships to allied disorders (e.g., sociopathy and schizophrenia).

The book is arranged into four sections. The first section is a splendid overview of the concept of adult minimal brain dysfunction by the chairperson of the conference and editor of this volume (Dr. Bellak). This is followed by an outstanding review of the nomenclaturial and clinical-diagnostic issues involved by Dr. Paul Wender. The second section, titled Follow-Up and Family Studies, explores the clinical manifestations of this syndrome with particular reference to long-term studies and family trends, including an excellent follow-up study by Dr. Barry Borland of the social-behavioral adaptation of adults who had been viewed as hyperkinetic boys. The general discussion of this section by Dr. Jonathan Cole sets a high standard for the similar discussions that follow. The general discussion focuses on correlations to allied disorders and unanswered questions. All in all, it provides a wealth of clinical reflections that make the book clinically relevant.

The third section of the book, Neuropsychiatric Aspects, reviews ego functions in adults with minimal brain dysfunction, possible relationships to epilepsy and/or episodic ag-

gressive behaviors, sensory motor disturbances, and clinical correlates of cerebral dysrhythmias in individuals who had adult minimal brain disorder who later develop such disorders as schizophrenia. The last section, Therapeutic Aspects, includes two superb presentations of a kind rarely seen in a book of this nature. First, there is a refreshing review by Dr. Stanley Greenspan of psychotherapeutic potentials for individuals with this disorder. This is in contrast to the usual focus on psychoactive medication in treatment and management. In particular, this presentation takes a hard look at the developmental, intrapersonal, interpersonal-family dynamics, and "at risk" neurophysiological dimensions and correlates these in a meaningful approach to management. Similarly, Dr. L. Eugene Arnold's contribution, titled "Philosophy and Strategy of Medicating Adults with Minimal Brain Dysfunction," provides a number of excellent visual illustrations that clearly elaborate the key symptoms of adult minimal brain disorder as well as outline equally clear treatment guidelines for managing the syndrome. Particular reference is made to therapeutic-management considerations; the emphasis is on the fact that medication should not be abused, misused, or used as a crutch or "cure-all" for this complex disorder.

This is the only high-quality extended discussion of adult minimal brain disorder available in the psychiatric literature. It is presented in excellent depth, incorporates a wide-based approach to current knowledge and viewpoints, and, because of the consistent excellence of the formal-informal general discussions that accompany each major section of the book, there is a coherent wholeness to this volume that is rarely seen in edited endeavors.

In summary, this is an excellent volume for all those who are interested in specific developmental issues in psychiatry, child psychiatry, interrelationships of early childhood disorders to later adult maladaptive behaviors, and the perplexing issues of etiological determinants to the adult character disorders and major psychoses. The format of presentation is a model of clarity. I recommend it highly.

FRANK J. MENOLASCINO, M.D.
Omaha, Neb.

Brain and Mind. Ciba Foundation Symposium 69. *Amsterdam, the Netherlands, Excerpta Medica, 1979, 413 pp., $51.25.*

For those who are beguiled by the elusive problem of dualism, the title of this volume seems like an invitation to a feast. The student interested in the subject often becomes impatient with a priori philosophical speculation. There seems to be no way to unravel the mind/body distinction through finer verbal shadings. Many of us who come at the problem as psychological or social scientists see mind (as opposed to body or brain) as composed of a series of intervening variables, that is, as promisory notes to be paid off in the study of brain chemistry, physiology, and even, ultimately, physics. For others of us, myself included, psychological understanding is simply a separate framework, *complementary* to the "more objective" study of physiology, chemistry, and physics. The study of social sciences is a different way of viewing the same phenomena viewed by the hard scientist. An analogy might be drawn to a painting. The pigment chemist can describe the ingredients on the canvas: the amount of titanium, lead, cobalt, etc., present. Yet the painting, aesthetically, is a phenomenon unto itself, to be perceived artistically. Its meaning, in the framework separate from the objectively established one of the chemist, is mostly within the viewer, but it would not exist without its physics, the chemistry of physical structure.

Complementarity becomes a possible alternative to either/or approaches to important human experience. This complementarity justifies a dualism while accepting holism (the experience of the painting could not exist without its physical elements), yet it integrates both.

What is nice about this volume is that the physiologist attends to the philosophical problem as well as "doing his own thing." In that particular regard this book gives us nearly a full measure. A range of subjects is challenged, and a refreshing assortment of insights and facts on some new work can be gained. There is even a subject index at the end of this book, for which the editors are to be congratulated; very often published symposia omit that kindness.

The general format of the book is to present two papers on a particular subject followed by commentary by a third person and a general discussion. The subjects covered are the evolutionary aspects of the brain, language, perception and memory, consciousness, experimental surgery and clinical neurology, pain and the senses, psychosis and abnormal behavior, pain and mood, and triunism. Throughout there is a serious attempt at clarity (for which I, reluctantly, give the scientists higher grades than the philosophers) as well as an attempt to meet the challenge of the antiquity and profundity of the problem. I especially liked the essay titled "Language: Perspectives from Another Modality" by Ursula Bellugi and Edward Klema, an essay on American sign language. (As a small example of a bit of interesting information: right-handed native deaf signers have better processing of signs in the left hemisphere than the right, although spatial-temporal function is usually assumed to be more a right-brain function than a left-brain function.)

Another essay of interest to me is the one by Creutzfeldt on "Neurophysiological Mechanisms and Consciousness." Creutzfeldt introduces the term "reflective loop" to express the way in which the brain symbolically presents the world to itself and then reflects on that particular representation. This is guessed to be the basis of the dualistic experience of ourselves and the world and the basis of our phenomenology.

Other portions of this volume will interest both specialists and those with a broader interest in the subject. The book does not resolve the mind/body dilemma totally; it does, however, present both facts and ideas that make the ambiguity more tolerable. The people at Ciba are to be congratulated for this book.

THEODORE NADELSON, M.D.
Boston, Mass.

Work and the Family System: A Naturalistic Study of Working-Class and Lower-Middle-Class Families, *by Chaya S. Piotrkowski. New York, N.Y., Free Press (Macmillan Publishing Co.), 1979, 331 pp., $14.95.*

Thirty members of 13 working-class and lower-middle-class families from one midwest and one far west state participated in this study. Two of the families were observed at

work and at home for about 18 hours each. The other families averaged 3.5 hours of interviews.

Although this was a study of work and the family system, most interviews seemed dyadic—interviewer and single interviewee. I wonder what a family systems approach to the interviewing, involving the whole family, would reveal.

There is ample case history documentation of work as alienating to the family and family life, yet no other system is proposed. To work and to love may still have to be compartmentalized and separate.

The main impact this book had on me was to emphasize the psychosocial factors involving work and the family. This underlines the changing medical model of disease as exemplified in the work of George Engel's biopsychosocial model (1). In addition to symptoms, signs, and laboratory findings, this model carefully assesses the role of stress, family, work, the treating physicians, the health care system, and other variables in the person and his illness.

There are few surprises in this book, and I suspect that it will not be of much interest to clinicians. It may interest family researchers.

REFERENCE

1 Engel GL: The need for a new medical model: a challenge for biomedicine. Science 196:129–136, 1977

ROSS V. SPECK, M.D.
Philadelphia, Pa.

Child Psychiatry in Asean Countries: A Book of Readings, *compiled by William M. Bolman, M.D., and Thomas W. Maretzki, Ph.D., for the ASEAN Child Psychiatry Forum. Quezon City Philippines, New Day Publishers (Detroit, Mich., Cellar Book Shop, distributor), 1979, 180 pp., $8.25 (paper).*

This volume is a compilation of papers selected from those presented at a workshop on child and adolescent psychiatry held in Djakarta, Indonesia, and at the World Psychiatric Conference in Honolulu, Hawaii, in 1977. The book is divided into three parts in which 18 authors present their views on the Status of Child Psychiatry in Each Asean Country, Cross-Cultural Issues in Child Psychiatry in Asean Settings, and General Issues for Cross-Cultural Psychiatry.

The five chapters in part one are devoted to the status of child psychiatry in Indonesia, Malaysia, the Philippines, Singapore, and Thailand. Imagine what it would be like to be one of a handful of child psychiatrists for a population of more than 135,000,000 people, of whom approximately 40% are minors, representing 300 ethnic subgroups, 250 different languages, the major religions of Islam, Christianity, and Hinduism in addition to indigenous belief systems, and spread over 5,500 km of islands and ocean! Dr. Yan Prasetyo briefly describes such a practice in Indonesia.

Part two of the text is a potpourri of diverse papers on everything from child rearing in Java to the reculturation shock of returning professionals. Underlying these chapters are issues surrounding the imposition of Western concepts of treatment, pathology, and child development on the native population. This is specifically salient when Dr. Quintos informs us, for example, of the Filipino transgenerational advice, "Sleep with your children so that they will grow close to you." This, coupled with the extended family kinship patterns, makes one look at child developmental issues in a less ethnocentric manner.

Part three deals with the more general impact of culture on the practice of child psychiatry. Dr. Bolman's essay on urbanization debunks a commonly held belief that urbanization is dangerous to people in terms of their mental health as they shift from peasant-rural life to modern industrial life. He believes that what is dangerous is a type of experience people encounter in the transition from village to city, and he feels that urbanization in developing nations must provide "social structures or institutions that will be able to offer support and a sense of belonging and cultural continuity." He then goes on to provide several recommendations for the development of child and family mental health services. One proposal, for a preventively oriented integrated community service, is a "community information center that would serve as the psychological and social equivalent to the biomedical services of the public health center." Dr. Bolman also suggests "the creation of a network of community family life centers." These centers would be primarily day-care services for children that would allow the children to make the transition from the extended family network of rural life to the increased social responsibility and independence necessary for urbanized nuclear family living.

In sum, aside from some irritating misspellings, typographical errors, and the absence of an index, this collection of readings offers the practitioner a sobering mirror to his or her own professional ethnocentricities and may be a useful resource book in confronting the increasingly complex issue of providing child psychiatric services for the Indo-Chinese refugees coming to the United States, who are in many ways similarly dislocated in both cultural and urban life.

MICHAEL F. HEIMAN, M.D.
Cerritos, Calif.

Stopping Wife Abuse: A Guide to the Emotional, Psychological, and Legal Implications, *by Jennifer Baker Fleming. Garden City, N.Y., Anchor Books (Anchor Press/Doubleday), 1979, 516 pp., $8.95 (paper).*

"What this book is designed to do is to provide you with the insight, information, skills and knowledge you need if you are contemplating or are providing help to battered women," writes the author. As a psychiatrist who has a basic psychoanalytic understanding of the motivation for all human behavior but who has an eclectic approach to the resolution of the conflicts produced by the many factors involved in effecting that behavior, I think this book fulfills its goal primarily in providing valuable information about the legal systems, legislation, current research, and programs providing services to battered women. Interwoven throughout the book are statistics and information that help the reader realize the magnitude and scope of domestic violence, its historical roots, and the overwhelming task of attempting to prevent it. There is much good practical "how to" advice for friends and relatives, the legal and criminal justice system, mental health practitioners, children, and husbands in helping the "victim."

I think that by referring to the battered woman as the "victim" the author has done her a disservice and has compounded the anger of the assaulter. Negating the fact that a

woman *shares* in the responsibility for what happens to her and not helping her see that her reactions at a critical point in an angry exchange more often than not trigger the violent behavior contribute to the list of reasons why so many men are turned off by professional help.

Both men and women are influenced by the effects of their biological differences, parental conditioning, and societal stereotyping. Both are victimized by the intolerance bred by the lack of understanding of unconscious roots of human behavior. I agree that each may need some geographic and psychological private space following a violent outburst, but unless the participants can be helped to see that they must ultimately work together in finding the external factors, interpersonal forces, and intrapsychic forces contributing to the conflict, both will experience a sense of failure should the relationship not survive. The single most pressing goal of most people who seek professional help is to be able to establish and maintain a meaningful one-to-one relationship.

I believe that by not differentiating between counseling and psychotherapy, not understanding the levels of psychotherapy, ignoring the unconscious factors that motivate human behavior, and minimizing the medical role, the author removes the psychiatrist from the team needed to provide a full range of services. The psychiatrist could be a valuable member of that team.

Not only the author is responsible for contributing to the lack of insight and knowledge by negating the overwhelming unconscious forces that sabotage so many of the more superficial efforts, but so is the psychiatrist for not becoming more involved outside the "office." The lack of clarity as to what constitutes psychotherapy, the difference between emotional disturbance and mental illness, and the minimal role of the physician helper are the responsibility of the psychiatrist, who by his or her absence allows this oversight to be perpetuated to the detriment of those who desperately need help.

MARY ANN BARTUSIS, M.D.
Morrisville, Pa.

Currents in Alcoholism, Vol. 6: Treatment and Rehabilitation and Epidemiology, *edited by Marc Galanter, M.D. New York, N.Y., Grune & Stratton, 1979, 332 pp., $33.50.*

The thought occurred to me while reading this book that the great papers in the field of alcoholism have already been written and the great thoughts already expressed. However, people working in the area need a reason to get together. Few of the 26 papers in this volume have abstracts, and in too many cases the knowledge to be extracted from the abundance of charts, graphs, and tables is not worth the effort. There are six sections: 1) The Alcoholic in the General Hospital, 2) Ambulatory Treatment, 3) Outcome, 4) Alcoholism in Women, 5) Alcoholism in Adolescents, and 6) The Drinking Driver. Each section has an introductory paper that is itself interesting but does not take the place of an abstract.

Several of the papers concern the apparently ever-nagging problem of diagnosis and with the extent to which the criteria of the National Council on Alcoholism lead to false positives and negatives. A lot of the problem still seems related to the effort to find in the large motley of alcohol abuse a single disease entity.

In one section the point is made that admission to a general hospital very often provides the first opportunity to confront the alcoholic with his or her problem. Later, however, we are told that it is unlikely that many alcoholics would need inpatient care at all if outpatient facilities were adequate; this includes the idea that a great deal more withdrawal from alcohol could be done without using an expensive hospital bed for detoxification.

One paper draws attention to the similarity between religious conversion, cults, and AA, particularly with regard to the "social support network." While the social support network can indeed sustain sobriety, it can also sustain drinking or even sensible alcohol use. Another paper addresses the question of whether the professional and AA can coexist. The answer is yes, but the psychotherapist should leave AA alone, much as he or she leaves the patient's religious beliefs alone.

One study of patients allowed to choose to drink during hospital treatment revealed that on follow-up the abstinent patients did better but that the patients who were drinking moderately by no means underwent rapid, progressive deterioration. This raises again the question as to whether there is not a substantial group of alcohol abusers whom we could help more by offering treatment other than abstinence-oriented programs.

With regard to driving while intoxicated, one author estimated that only 1% of the people who have driven with a blood alcohol concentration of over .05 mg/100 ml have been arrested. Two studies of programs for people who drink and then drive failed to demonstrate that the programs were effective.

The section on women can be summed up quickly: women are drinking more. The percent of women who remain abstinent after treatment is not different from that of men. The daughters of alcoholic fathers drink more, and assertiveness training is a good adjunct to other therapy.

It could be that with AA groups in every small town, detoxification units in every county, halfway houses in every city, programs for alcoholics in every industry, and more strict prosecution of drunk driving we have gone as far as we can go. The only thing left is a major change in lifestyle.

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Books Received

This list acknowledges the receipt of recent books. Books of particular interest to the readers of this journal will be reviewed as space permits, and copies of the reviews will be sent to the publishers. Books cannot be returned to the publishers.

Ethnography: A Research Tool for Policymakers in the Drug and Alcohol Fields, *edited by Carl Akins and George Beschner. Rockville, Md., National Institute on Drug Abuse, 1980, 128 pp., no price listed (paper).*

AMA Drug Evaluations, 4th ed., *by the American Medical Association Department of Drugs, in cooperation with the American Society for Clinical Pharmacology and Therapeutics. Chicago, Ill., AMA, 1980, 1,470 pp., no price listed.*

A Psychiatric Glossary, 5th ed., *by the American Psychiatric Association. Washington, D.C., APA, 1980, 142 pp., $9.95; $5.95 (paper).*

The Social Animal, 3rd ed., *by Elliot Aronson. San Francisco, Calif., W.H. Freeman and Co., 1980, 365 pp., $16.00; $7.95 (paper) (comes with a study guide by Laura Valvatne).*

Special Needs and Services: Philosophy, Programs, and Practices for the Creation of Quality Service for Children. Child Care: A Comprehensive Guide, Vol. 4, *edited by Stevanne Auerbach, Ph.D., with James A. Rivaldo. New York, N.Y., Human Sciences Press, 1980, 251 pp., $19.95.*

Child Development in Normality and Psychopathology, *edited by Jules R. Bemporad, M.D. New York, N.Y., Brunner/Mazel, 1980, 531 pp., $25.00.*

Interviewing: A Guide for Health Professionals, 3rd ed., *by Lewis Bernstein, Ph.D., and Rosalyn S. Bernstein, M.A. New York, N.Y., Appleton-Century-Crofts, 1980, 188 pp., no price listed (paper).*

Surviving and Other Essays, *by Bruno Bettelheim. New York, N.Y., Vintage Books (Random House), 1980, 426 pp., $4.95 (paper).*

Assessing the Contributions of the Social Sciences to Health: American Association for the Advancement of Science Selected Symposium 26, *edited by M. Harvey Brenner, Anne Mooney, and Thomas J. Nagy. Boulder, Colo., Westview Press for the AAAS, 1980, 216 pp., $20.00.*

Biological Studies of Mental Processes, *edited by David Caplan. Gambridge, Mass., MIT Press, 1980, 322 pp., $24.95.*

Wolff's Headache and Other Head Pain, 4th ed., *edited by Donald J. Dalessio, M.D. New York, N.Y., Oxford University Press, 1980, 461 pp., $35.00.*

The Facts About "Drug Abuse," *by the Drug Abuse Council. New York, N.Y., Free Press (Macmillan Publishing Co.), 1980, 291 pp., $14.95.*

The Hospice Way of Death, *by Paul M. DuBois, Ph.D. New York, N.Y., Human Sciences Press, 1980, 207 pp., $16.95.*

Anticonvulsant Therapy: Pharmacologic Basis and Practice, 2nd ed., *by Mervyn J. Eadie, M.D., Ph.D., and John H. Tyrer, M.D. New York, N.Y., Churchill Livingstone, 1980, 330 pp., no price listed.*

National Health Insurance: Conflicting Goals and Policy Choices, *edited by Judith Feder, John Holahan, and Theodore Marmor. Washington, D.C., Urban Institute, 1980, 705 pp., $25.00; $12.50 (paper).*

Why? Children's Questions: What They Mean and How to Answer Them, *by Ruth Formanek and Anita Gurian. Boston, Mass., Houghton Mifflin Co., 1980, 213 pp., $8.95.*

Depression in Children and Adolescents, *edited by Alfred P. French, M.D., and Irving N. Berlin, M.D. New York, N.Y., Human Sciences Press, 1979, 290 pp., $19.95.*

Psychotherapeutic Interventions in Life-Threatening Illness: Advances in Psychosomatic Medicine, Vol. 10, *edited by Hellmuth Freyberger. Basel, Switzerland, S. Karger, 1980, 206 pp., $62.00.*

The Courage to Change: From Insight to Self-Innovation, *by Edrita Fried, Ph.D. New York, N.Y., Brunner/Mazel, 1980, 236 pp., no price listed.*

The Sociology of Mental Illness, *by Bernard J. Gallagher III. Englewood Cliffs, N.J., Prentice-Hall, 1980, 352 pp., $11.95 (paper).*

Alzheimer's Disease: Early Recognition of Potentially Reversible Deficits, *edited by A.I.M. Glen, M.B.Ch.B., D.P.M., and L.J. Whalley, M.D., M.B.B.S., D.P.M. New York, N.Y., Churchill Livingstone, 1979, 199 pp., no price listed.*

Drug Abuse Deaths in Nine Cities: A Survey Report. NIDA Research Monograph 29, *by Louis A. Gottschalk, M.D., Frederick L. McGuire, Ph.D., Jon F. Heiser, M.D., Eugene C. Dinovo, Ph.D., and Herman Birch, Ph.D. Washington, D.C., National Institute on Drug Abuse, 1980, 172 pp., $4.25 (paper).*

Social Problems in Cancer Control, *by Howard P. Greenwald. Cambridge, Mass., Ballinger Publishing Co. (Harper & Row), 1980, 281 pp., no price listed.*

The Body of Life, *by Thomas Hanna. New York, N.Y., Alfred A. Knopf, 1980, 203 pp., $9.95.*

Problems of Drug Dependence, 1979: Proceedings of the 41st Annual Scientific Meeting of the Committee on Problems of Drug Dependence, Inc. NIDA Research Monograph 27, *edited by Louis S. Harris, Ph.D. Rockville, Md., National Institute on Drug Abuse, 1979, 483 pp., $8.00 (paper).*

New Directions in Childhood Psychotherapy, Vol. 1: Developmental Considerations, *edited by Saul I. Harrison, M.D., and John T. McDermott, Jr., M.D. New York, N.Y., International Universities Press, 1980, 583 pp., $35.00.*

A Psychodynamic Approach to Adolescent Psychiatry: The Mount Sinai Experience, *edited by Don R. Heacock. New York, N.Y., Marcel Dekker, 1980, 347 pp., $46.50.*

An Introduction to Theories of Personality, *by B.R. Hergenhahn. Englewood Cliffs, N.J., Prentice-Hall, 1980, 374 pp., $15.95.*

Language and Language Disorders in Childhood, *edited by L.A. Hersov, M. Berger, and A.R. Nicol. New York, N.Y., Pergamon Press, 1980, 172 pp., $9.00 (paper).*

How to Help Your Child Have a Spiritual Life: A Parent's Guide to Inner Development, *by Annette Hollander, M.D. New York, N.Y., A & W Publishers, 1980, 260 pp., $12.95.*

Handbook of Lithium Therapy, *edited by F. Neil Johnson, Ph.D. Baltimore, Md., University Park Press, 1980, 442 pp., $39.50.*

Death and Dying: Views from Many Cultures. Perspectives on Death and Dying 1, *edited by Richard A. Kalish. Farmingdale, N.Y., Baywood Publishing Co., 1980, 153 pp., $17.95 (3 volume set) (paper).*

Caring Relationships: The Dying and the Bereaved. Perspectives on Death and Dying 2, *edited by Richard A. Kalish. Farmingdale, N.Y., Baywood Publishing Co., 1980, 154 pp., $17.95 (3 volume set) (paper).*

Death, Dying, Transcending. Perspectives on Death and Dying 3, *edited by Richard A. Kalish. Farmingdale, N.Y., Baywood Publishing Co., 1980, 154 pp., $17.95 (3 volume set) (paper).*

Psychology and Society: In Search of Symbiosis. Houston Symposium I, *edited by Richard A. Kasschau and Frank S. Kessel. New York, N.Y., Holt, Rinehart and Winston, 1980, 269 pp., $23.95.*

Anxiety and Emotions: Physiological Basis and Treatment, *by Desmond Kelly, M.D. Springfield, Ill., Charles C Thomas, 1980, 388 pp., $24.75.*

Psychology and National Health Insurance: A Sourcebook, *edited by Charles A. Kiesler, Nicholas A. Cummings, and Gary R. VandenBos. Washington, D.C., American Psychological Association, 1979, 639 pp., $19.50; $15.00 (paper).*

Homosexuality and the Law: Research on Homosexuality, Vol. 1, *edited by Donald C. Knutson, J.D. New York, N.Y., Haworth Press, 1980, 155 pp., $19.95; $9.95 (paper).*

Basic Psychiatric Concepts in Nursing, 4th ed., *by Joan J. Kyes, R.N., M.S.N., and Charles K. Hofling, M.D. Philadelphia, Pa., J.B. Lippincott Co., 1980, 716 pp., $15.75.*

The Age Factor: Love, Sex and Friendship in Age-Different Relationships, *by Jack LaPatra, Ph.D. New York, N.Y., M. Evans and Co., 1980, 214 pp., $9.95.*

Principles and Practice of Sex Therapy, *edited by Sandra R. Leiblum and Lawrence A. Pervin. New York, N.Y., Guilford Press, 1980, 394 pp., $22.50.*

Health Care of the Elderly: Strategies for Prevention and Intervention. Frontiers in Aging, Vol. I, *edited by Gari Lesnoff-Caravaglia, Ph.D. New York, N.Y., Human Sciences Press, 1980, 220 pp., $19.95; $9.95 (paper).*

Becoming Psychiatrists: The Professional Transformation of Self, *by Donald Light. New York, N.Y., W.W. Norton & Co., 1980, 418 pp., $18.95.*

The Manson Women: A "Family" Portrait, *by Clara Livsey, M.D. New York, N.Y., Richard Marek, 1980. 244 pp., $10.95.*

Homosexual Behavior: A Modern Reappraisal, *edited by Judd Marmor. New York, N.Y., Basic Books, 1980, 401 pp., $27.50.*

Readings in Medical Sociology, *edited by David Mechanic. New York, N.Y., Free Press (Macmillan Publishing Co.), 1980, 506 pp., $9.95 (paper).*

Love and Love Sickness: The Science of Sex, Gender Difference, and Pair-Bonding, *by John Money. Baltimore, Md., Johns Hopkins University Press, 1980, 242 pp., $16.95.*

Aids to Psychiatry, *by H.G. Morgan, M.A., M.D., D.P.M., in collaboration with M.H. Morgan, M.A., M.D. New York, N.Y., Churchill Livingstone, 1979, 152 pp., no price listed (paper).*

Teaching the Severely Mentally Retarded: Adaptive Skills Training, *by Allen A. Mori, Ph.D., and Lowell F. Masters, Ed.D. Germantown, Md., Aspen Systems Corp., 1980, 393 pp., $24.95.*

Sex, Motivation, and the Criminal Offender, *by Robert H. Morneau, Jr., Ph.D., and Robert R. Rockwell. Springfield, Ill., Charles C Thomas, 1980, 399 pp., $29.75.*

OFFICIAL ACTIONS

Highlights of the 133rd Annual Meeting

The 133rd annual meeting of the American Psychiatric Association, which focused on the theme "To Love and to Work," was held in San Francisco, Calif., May 3-9, 1980. The total registration was 11,428, including 5,858 members and Fellows, 1,530 nonmember professionals, 799 exhibitors, 190 members of the press, and 2,599 spouses and families of members.

The opening session, on Monday morning, May 5, was brought to order by Alan A. Stone, M.D., 108th President of the Association. The invocation was given by the Reverend Toshio Murakami, Director of the National Headquarters of the Buddhist Churches of America, San Francisco. Official greetings were extended by the Honorable Dianne Feinstein, Mayor of San Francisco.

Dr. Stone then introduced distinguished representatives of psychiatric and related organizations from the United States and other countries:

Dr. George Mahy, immediate Past President, Caribbean Psychiatric Association; Dr. Edwin Lipinski, President, Canadian Psychiatric Association; Dr. G.J. Sarwer-Foner, President, Canadian Psychoanalytic Association; Dr. Adib R. Mikhail, representing the Egyptian Psychiatric Association; Dr. Marcel Arditti, representing the French Medico-Psychological Society and General Secretary of the French Association of Psychiatry; Prof. D. Hanfried Helmchen, President, German Association for Psychiatry and Neurology; Dr. C.N. Stefanis, Past President, Greek Society of Neurology and Psychiatry; Dr. C.M. Chung, President, Hong Kong Psychiatric Association; Dr. Shridhar Sharma, representing the Indian Psychiatric Society; Prof. L.S. Gillis, Chairman of the Executive Committee of the Society of Psychiatrists of South Africa; Mr. Gowan T. Guest, President, World Federation for Mental Health; Dr. Tom van der Grintin, General Director, National Institute for Mental Health in the Netherlands; Dr. John Carleton, President, World Association for Social Psychiatry; Dr. John Howells, representing the Royal College of Psychiatrists; Dr. Mauro Villegas, President, Inter-American Council of Psychiatric Associations; Dr. Ayo Binitie, President, Association of Psychiatrists in Nigeria; Dr. Jorge M. Velasco Alzago, representing the Mexican Psychiatric Association; Dr. Robert L. Stubblefield, President, American Academy of Child Psychiatry; Dr. Martin Gershman, Chair, California Chapter, American Academy of Pediatrics; Dr. Nathan T. Sidley, President, American Academy of Psychiatry and the Law; Dr. Paul H. Feldman, President, American Academy of Psychoanalysis; Dr. Joel Yager, President, American Association of Directors of Psychiatric Residency Training; Dr. Mehadin K. Arefeh, President, American Association of Psychiatric Administrators; Dr. Jack C. Westman, President, American Association of Psychiatric Services for Children; Dr. Stanley R. Dean, President, American Association for Social Psychiatry; Dr. Marc Hollender, President, American Board of Psychiatry and Neurology; Dr. Leonard M. Riggs, Jr., President-Elect, American College of Emergency Physicians; Dr. Herbert S. Ripley, President, American College of Psychoanalysts; Dr. Maurice Fox, Regent, American College of Physicians; Dr. Shervert Frazier, President, American College of Psychiatrists; Dr. Lois Skully, immediate Past President, American Medical Women's Association; Ms. Martha Mitchell, Chairperson, Division on Psychiatric and Mental Health Nursing, American Nurses' Association; Dr. Arnold M. Cooper, President, American Psychoanalytic Association; Dr. Nicholas Cummings, immediate Past President, American Psychological Association; Dr. June Jackson Christmas, President, American Public Health Association; Dr. D. Steve Goudelock, President, American Society for Adolescent Psychiatry; Dr. Peter Santucci, President, American Society for Physician Analysts; Dr. Patrick McKegney, President, Association for Academic Psychiatry; Dr. Gerald A. Melchiode, President, Association of Directors of Medical Student Education in Psychiatry; Ms. Julia Robinson, President, Association of Mental Health Administrators; Chaplain Clark S. Aist, President, Association of Mental Health Clergy; Dr. Andrea Delgado, President, Black Psychiatrists of America; Dr. Carolyn B. Robinowitz, Vice President, Council on Medical Specialty Societies; Dr. Robert Gibson, President, Group for Advancement of Psychiatry and Past President of the American Psychiatric Association; Mr. Allen R. Moltzen, Trustee, the Mental Health Association, Ms. Barbara A. Graham, member, Board of Directors, National Association of Social Workers; Mr. Donald Fox, President, National Council of Community Mental Health Centers; Dr. Vertis Thompson, President-Elect, National Medical Association; and Dr. Lewis Robbins, President, National Association of Private Psychiatric Hospitals and Vice-President of the American Psychiatric Association.

Dr. Stone also recognized the past Presidents and members of the Board of Trustees of the American Psychiatric Association, the past Speakers and members of the Executive Committee of the Assembly, past Vice-Presidents of the Association, District Branch Presidents, newly elected APA officers, newly elected Assembly officers, and the chairpersons of APA Councils.

Dr. Stone then introduced the San Francisco Civic Chorale, who performed the "Hallelujah Chorus" from Handel's *Messiah* under the direction of Ms. Winifred Baker.

Linn A. Campbell, M.D., Chairperson of the Task Force on Arrangements, was introduced by Dr. Stone, who thanked the task force for an imaginative job of arranging the leisure time activities.

Dr. Stone extended the Association's appreciation to Allan Beigel, M.D., and the Program Committee for their excellent work. He then presented a Special Commendation for Service to Dr. Beigel.

Vice-President Peter A. Martin, M.D., introduced Dr. Stone, who gave the Presidential Address, "Conceptual Ambiguity and Morality in Modern Psychiatry." Vice-President Lewis L. Robbins, M.D., then introduced Donald G. Langsley, M.D., who responded as the President-Elect. (The texts of these addresses are published elsewhere in this issue of the *Journal*.)

The opening session closed with a benediction by the Reverend Hannibal Williams of the New Liberated Presbyterian Church in San Francisco, and with organ music by John E. Fryer, M.D. Dr. Stone then adjourned the meeting.

EXTRAORDINARY SESSION

Immediately after the Opening Session a special session was held for discussion of equal rights for women and the Equal Rights Amendment. Dr. Langsley, chairperson of the session, introduced Dr. Stone, who outlined the history of APA's support of the ERA. Presentations were given by Judd Marmor, M.D., and Jean Baker Miller, M.D., and were followed by questions and comments from the floor.

BUSINESS SESSION

The business session was convened as a meeting of the Assembly of District Branches acting for the membership in responding to reports of the officers and actions of the Board of Trustees. The meeting followed the Extraordinary Session in the Arena of the Civic Auditorium. Dr. Stone presided. After a memorial to deceased members and Fellows, John S. Visher, M.D., gave a memorial to past Speaker Dr. Warren S. Williams. H. Keith H. Brodie, M.D., speaking for Edward C. Kirby, M.D., Chairperson of the Committee of Tellers, announced the results of the election of officers and trustees, the vote on amendments to the Constitution and By-Laws, and the vote on the referendum to rescind the Board of Trustees' decision not to hold APA meetings in states that have not ratified the Equal Rights Amendment.

The reports of the officers to the membership followed. (The floor was open to questions after each report, and all reports were accepted as read by the Assembly.) After H. Keith H. Brodie, M.D., presented the Secretary's report, Charles B. Wilkinson, M.D., Treasurer, reported a surplus of income over expenses for 1979 due in part to *DSM-III* sales, continuing education course revenues, a strong grant/contract program, and increases in rate schedules for APA products and services. Robert O. Pasnau, M.D., Speaker of the Assembly, focused on seven major problem areas—money, education, politics, bureaucracy, law, internal organization, and future planning. The Assembly's Speaker-Elect, Melvin M. Lipsett, M.D., urged proactive involvement in legislation, research, and public information. Lawrence Hartmann, M.D., Chairperson, Committee on Constitution and By-Laws, presented 6 amendments to the Constitution and By-Laws. Philip M. Margolis, M.D., Chairperson of the Committee on Membership, reported on discussions at the committee's October meeting on criteria for Fellowship and on continuing medical education requirements. Melvin Sabshin, M.D., presented the report of the Medical Director.

Dr. Stone then presented the Speaker's plaque to Robert O. Pasnau, M.D., retiring Speaker, and the Vice-President's badge to Lewis L. Robbins, M.D., the retiring Vice-President. Judd Marmor, M.D., presented the President's badge to Dr. Stone.

Dr. Stone then adjourned the business session and called the annual forum to order. The membership voted to refer to the Board of Trustees their sentiment favoring not holding annual meetings in states that have not ratified the ERA after the 1981 meeting, until the issue is resolved in the nation. There was also discussion of a variety of problems in the JCAH Consolidated Standards. An amendment regarding continuity of care was referred to the Board. Dr. Stone then adjourned the annual forum.

SCIENTIFIC SESSIONS

The scientific sessions began Monday afternoon, May 5. There were 178 papers presented in regular sessions, as well as 380 papers in symposia. There were 43 issue workshops, 109 evening panels, 29 videotaped sessions, 15 films, and 46 papers on new research. With few exceptions, the official annual meeting program met the criteria for continuing medical education Category I credit, as did the 115 continuing education courses offered throughout the week.

There were 26 special lectures. Jeanne Spurlock, M.D., Deputy Medical Director, Minority Fellowship Program, APA, and Clinical Professor of Psychiatry, George Washington University and Howard University, Washington, D.C., spoke on "The Seventies: Societal Changes and Their Impact on Child Psychiatry." Alan M. Dershowitz, LL.B., gave a lecture titled "Law and Psychiatry: Are They More Comfortable Married, Divorced, or Living Together?" The Honorable Nathan J. Stark, Under Secretary, United States Department of Health, Education, and Welfare, gave a speech titled "The Federal Government's Role in Mental Health in the 1980s." Stanley N. Cohen, M.D., Professor of Medicine and Professor and Chairman, Department of Genetics, Stanford University, spoke on "Gene Manipulation of Microorganisms: Current and Future Prospects." Helen Singer Kaplan, M.D., founder of the Human Sexuality Program, New York Hospital-Cornell Medical Center, spoke on "Current Concepts of Human Sexuality."

The APA's Founders Award Lecture, "Que Sera Sera No Tiene Que Ser (Plan or Perish)," was delivered by Jack R. Ewalt, M.D., Past President of APA and Director, Mental Health and Behavioral Sciences, Department of Medicine and Surgery, Veterans Administration. The writer Michael Crichton, M.D., spoke on "Why Work Is Work." James N. Glick, M.D., an orthopedic surgeon in private practice in San Francisco, gave a speech titled "Sports Injuries: Enjoyment with Pain." The Honorable Harry A. Blackmun, Justice of the Supreme Court, spoke on "Psychiatry and the Supreme Court." The Benjamin Rush Lecture, "Medical Evangelism and Psychiatry: The Case of Psychosomatic Medicine," was presented by Nathan G. Hale, Jr., M.D., Professor of History, University of California, Riverside. James O'Toole, Ph.D., Director, Twenty Year Forecast, and

Associate Professor of Management, University of Southern California, spoke on "Work and Love (But Mainly, Work)." Sherven H. Frazier, M.D., Psychiatrist-in-Chief, McLean Hospital, Belmont, Mass., gave a lecture titled "More Violence: The Wave of the Future?" James L. Bernene, M.D., Associate Clinical Professor of Medicine, Yale University, and Director, Medical Residency Program, Greenwich Hospital, Greenwich, Conn., spoke on "Hypoglycemia, Fact or Fantasy?" "The Working Family: A Researcher's View of Health in the Household" was presented by David Reiss, M.D., Center for Family Research, George Washington University, Washington, D.C. The Seymour Vestermark Memorial Lecture, "The Vestermark Tradition, 1960-1980," was presented by Francis Neil Waldrop, M.D. Helen M. Ranney, M.D., Chairperson, Department of Medicine, University of California, San Diego, spoke on "Advances in Hematology: Changes in the Outlook of Hematologic Disease Over Three Decades." Jack Weinberg, M.D., Director, Illinois State Psychiatric Institute, Chicago, and Professor of Psychiatry, Abraham Lincoln School of Medicine, University of Illinois and Rush Medical College, Rush-Presbyterian-St. Luke's Medical Center, gave a lecture titled "No Synonym for the Word Love."

The Solomon Carter Fuller Lecture, "The Rights of Prisoners and Patients in State Institutions," was given by the Honorable Constance Baker Motley, member of the United States Court of Appeals, Second District (New York). Barbara Ehrenreich, who writes on subjects related to women and health, spoke on "Work and Love: Can Women Have Both? (And Are They Worth It When We Get Them?)" "Headache: Current Concepts" was presented by Neil H. Raskin, M.D., Professor and Vice-Chairman, Department of Neurology, University of California, San Francisco, School of Medicine. The Simon Bolivar Lecture, "Value Systems and Sexual Morality," was delivered by Otto F. Kernberg, M.D., Medical Director, New York Hospital-Cornell Medical Center, Westchester Division; Professor of Psychiatry, Cornell University Medical College; and Training and Supervising Analyst, Columbia University Center for Psychoanalytic Training and Research. Herbert S. Kaufman, M.D., who is an allergist in private practice in San Francisco, gave a lecture titled "TB or Not TB: An Overview of Basic Mechanisms of Man's Immunity." George E. Vaillant, Professor of Psychiatry, Harvard Medical School, spoke on "Erikson's Stage of Industry: A Powerful Predictor of Adult Mental Health." Senator Spark Masayuki Matsunaga spoke on "Mental Health in the '80s: A Senator's View." "The Interface of Brain and Behavior: Temporal Lobe Epilepsy" was presented by Bruce O. Berg, M.D., Professor of Neurology and Pediatrics and Director of Child Neurology, University of California, San Francisco. Silvano Arieti, M.D., Clinical Professor of Psychiatry, New York Medical College, presented a lecture titled "The Family of the Schizophrenic: Re-Valuation of the Psychodynamic Role and Participation in Patients' Rehabilitation."

CONVOCATION

The 24th Convocation of Fellows was held in the Continental Ballroom of the San Francisco Hilton Hotel on Monday evening, May 5, at 7:30 p.m. Dr. Stone presided. After the processional march, Dr. Stone called the convocation to order. The invocation was given by the Reverend Leonidas C. Contos of the Greek Orthodox Church of the Holy Cross, Belmont, Calif. Dr. Langsley introduced the Life Fellows and conducted the induction of Fellows. Dr. Stone then introduced the Life Fellows and Life Members who have been in the Association for 50 years (1930-1980): Grace Baker, M.D., New York, N.Y.; Ralph Warren Bohn, M.D., Lansdowne, Pa.; Leon Joseph Saul, M.D., Media, Pa.; Louis Adrian Schwartz, M.D., Rockport, Mass.; and Jean A. Thompson, M.D., Brandford, Conn. Ms. Evelyn Stone, Belmont, Mass., was designated Honorary Fellow; Semyon Gluzman, M.D., Siberia, U.S.S.R. (in exile), was designated Distinguished Fellow; and Stanislaw Dabrowski, M.D., Warsaw, Poland, Professor Detlev Ploog, Munich, West Germany, Jan Frans Schrijvers, M.D., Geel, Belgium, and Costas N. Stefanis, M.D., Athens, Greece, were designated Corresponding Fellows.

The presentation of awards followed. Dr. Stone presented the Distinguished Service Award—given to APA members "whose distinguished careers have ennobled the profession of psychiatry"—to Daniel Blain, M.D., Past President and former Medical Director of APA, and Seymour S. Kety, M.D., Professor of Psychiatry, Harvard Medical School, and Director of the Laboratories for Psychiatric Research, Mailman Research Center and McLean Hospital.

The Foundations' Fund Prize for Research in Psychiatry, established in 1977 to recognize those who have made distinguished contributions to research in psychiatry, was presented by Herbert Pardes, M.D., to Merton M. Gill, M.D., for "distinguished contribution to psychoanalytic theory and to the systematic study of the psychotherapeutic process," to Mardi J. Horowitz, M.D., for "distinguished contribution to an understanding of the psychological processes following serious life events and of the adaptive changes facilitated by psychotherapy," and to Albert J. Stunkard, M.D., for "distinguished contribution to an understanding of eating disorders and the development of research methodology in this and cognate areas."

The Blanche F. Ittleson Prize for Research in Child Psychiatry, established in 1977 to "honor outstanding research in child psychiatry, in particular as it relates to the mental health of children," was presented by Reginald Lourie, M.D., to Stella Chess, M.D., Professor of Child Psychiatry, Department of Psychiatry, New York University Medical Center, and Alexander Thomas, M.D., Professor, Department of Psychiatry, New York University School of Medicine and Attending Psychiatrist, Bellevue Psychiatric Hospital.

Irving Philips, M.D., presented the Agnes Purcell McGavin Award to Albert J. Solnit, M.D., Director, Child Study Center, and Sterling Professor of Pediatrics and Psychiatry at Yale University School of Medicine, for his "outstanding contributions to the prevention of mental disorders in children."

The Isaac Ray Award was presented by Jonas Robitscher, M.D., to Seymour L. Halleck, M.D., Professor, Department of Psychiatry, Associate Director for Recruitment, Residency Training, and Adjunct Professor, University of North Carolina Law School, Chapel Hill, N.C., for his "outstanding contributions to legal psychiatry."

The Seymour D. Vestermark Award was presented by Herbert Pardes, M.D., to Francis N. Waldrop, M.D., recently retired from a career in training and education with Saint Elizabeths Hospital, the National Institute of Mental Health, and the Alcohol, Drug Abuse, and Mental Health

Administration, for "outstanding contributions to undergraduate and postgraduate medical education, to continuing education, and to the education of behavioral scientists for research."

The APA Founders Award, established in 1976 to "honor members of the Association who have made outstanding contributions as author, spokesperson, and advocate in the service of the mentally ill and disabled and to the art and science of helping them," was presented by Shervert H. Frazier, M.D., to Jack R. Ewalt, M.D., Director, Mental Health and Behavioral Sciences, Department of Medicine and Surgery, Veterans Administration, Washington, D.C., and Past President of APA, for "the landmark document, 'Action for Mental Health,' which resulted from the work of the Joint Commission on Mental Illness and Health."

Dr. Frazier also presented the Robert T. Morse Writers Award to Mr. Darrell Sifford, columnist for *The Philadelphia Inquirer,* and the District Branch Newsletter of the Year Award to David A. Rothstein, M.D., Editor, *The Psychiatric Examiner,* Illinois Psychiatric Society. Dr. Rothstein is in private practice in Chicago, Ill.

After the presentation of awards, Dr. Stone introduced Professor Roberto Mangabeira Unger, Harvard Law School, Cambridge, Mass., who gave the William C. Menninger Memorial Convocation Lecture, "Science, Politics, and Moral Vision in Psychiatry." The convocation was then recessed.

MEETINGS OF THE BOARD OF TRUSTEES

The Board of Trustees met in regular session on Sunday, May 4, and on Thursday, May 8. A complete record of Board transactions will be reported by the Secretary (*AJP,* October 1980).

SPOUSES' ACTIVITIES AND ENTERTAINMENT

The Task Force on Arrangements, chaired by Linn A. Campbell, M.D., planned a variety of activities. General tours of San Francisco were given as well as tours of the Monterey Peninsula, Muir Woods and Sausalito, Marin County, Victorian houses of San Francisco, the Filoli House and gardens, the Audubon Canyon Ranch, and the Napa Valley wine country. Other activities included Chinese cooking demonstrations, a visit to the Conservatory and to Asian and French art collections, an architectural antique expedition, a sunset cocktail cruise, and behind-the-scenes observation of local artists at work.

ALLIED MEETINGS

The following professional associations also held meetings in San Francisco during the week of May 3-9: Academy of Orthomolecular Psychiatry, American Academy of Child Psychiatry, American Academy of Psychiatry and the Law, American Academy of Psychoanalysis, American Association for Geriatric Psychiatry, American Association for Social Psychiatry, American Association of Chairmen of Departments of Psychiatry, American Association of General Hospital Psychiatrists, American Association of Psychiatrists from India, American Board of Forensic Psychiatry, American Psychoanalytic Association, American Society for Adolescent Psychiatry, American Society of Physician Analysts, Association for Academic Psychiatry, Association of Korean-American Psychiatrists, Association of Mental Health Clergy, Behavior Therapy and Research Society, Black Psychiatrists of America, International Psychiatric Association for the Advancement of Electrotherapy, National Association of State Mental Health Program Directors, National Association of State Mental Health Research Institutes, National Association of Veterans Administration Chiefs of Psychiatry, National Guild of Catholic Psychiatrists, Navy Psychiatry, Philippine Psychiatrists in America, Psychiatrists for ERA, Research Assessment and Training Unit New York State Psychiatric Institute, Southern Psychiatric Association, and Turkish American Neuropsychiatric Association.

H. KEITH H. BRODIE, M.D.
Secretary, American Psychiatric Association

The American Board of Psychiatry and Neurology

The following successfully completed the Board examination given in Los Angeles, CA, April 14–15, 1980.

PSYCHIATRY

Aflatooni, Saeed, M.D., Roseburg, OR
Africa, Bruce, M.D., Berkeley, CA
Aillon, Gonzalo A., M.D., Duncanville, TX
Allister, Robert J., M.D., San Francisco, CA
Anker, Jeffrey Lawrence, M.D., Boulder, CO
Annis, Frank Lloyd, M.D., Los Altos, CA
Applin, Thomas L., M.D., Bethesda, MD
Arguello, Fares Joseph, M.D., Salt Lake City, UT
Astrada, Carlos A., M.D., Annandale, VA
Auerbach, Eric D., M.D., Rancho Palos Verdes, CA

Baber, Riaz A., M.D., Forest Park, IL
Bagwell, Howard Roberts, Jr., M.D., Portland, OR
Bain, Michael W., M.D., APO, NY
Baker, John W., M.D., Imola, CA
Barnes, Robert F., M.D., Seattle, WA
Bartell, Gary D., M.D., Danville, IN
Barton, Charles C., M.D., Hackensack, NJ
Barza, Allan B., M.D., Walnut Creek, CA
Beltangady, Shamal S., M.D., Houston, TX
Benham, Anne Leland, M.D., Providence, RI
Benson, M. Christina, M.D., Los Angeles, CA
Berger, Fred Keith, M.D., San Diego, CA
Berman, Ralph H., M.D., Santa Cruz, CA
Bien, Ralph Daniel, M.D., Kentfield, CA
Bloomgarden, Robert Evan, M.D., Oakland, CA
Bramhall, Stuart J., M.D., Chico, CA
Brantley, Thomas I., Jr., M.D., Detroit, MI
Brooks, Robert J., M.D., Albany, CA
Browning, Marilyn Crouch, M.D., Sonoma, CA
Bry, Vernon Arthur, Jr., M.D., South Lake Tahoe, CA
Buchanan, Patrick James, M.D., San Francisco, CA
Burchard, Thomas Kirk, M.D., Carmel, CA
Burkins, John Andrew, M.D., APO, NY

Calnaido, Rajadorai, M.D., Shalimar, FL
Carpenter, Frank E., M.D., San Antonio, TX
Carroll, Robert Steven, M.D., West Los Angeles, CA
Casey, Daniel E., M.D., Portland, OR
Chang, Chun Sik, M.D., Dallas, PA
Chen, Li-Chuen, M.D., Danville, IL
Chung, Richard S., M.D., Mission Hills, CA
Clark, James P., M.D., Farmington, NM
Clark, R. Barkley, M.D., Littleton, CO
Cliff, T. Menzie, M.D., San Luis Obispo, CA
Collins, Arthur Cowan, M.D., Albuquerque, NM
Colvin, David Forrest, M.D., Morgantown, WV
Consolver, Jay Paul, M.D., Woodland Hills, CA
Cooper, Ruth Anne, M.D., Berkeley, CA
Goopwood: William E., M.D., Madison, TN
Corelli, Richard J., M.D., Palo Alto, CA
Cruz, Rizalino S., M.D., Lebanon, PA
Cutler, David Lloyd, M.D., Portland, OR

Daghestani, Amin N., M.D., Skokie, IL
Dale, D. Duane, M.D., Portland, OR
Damania, Shireen Rustom, M.D., Cedar Grove, NJ
David, Ariel, M.D., Chicago, IL
Davis, Josh Daniel, Sr., M.D., Tuscaloosa, AL
Davis, Ray Charles, M.D., Houston, TX
Dominguez, Roberto A., M.D., Miami, FL
Dorman, Kenneth Randall, M.D., Austin, TX
Dubriwny, Michael D., M.D., Tulsa, OK
Dudelczyk, Michael Leib, M.D., Santa Fe, NM

Eaton, Edward L., M.D., Topeka, KS
Eaton, Leslie Kay Ellis, M.D., Topeka, KS
Eddleman, Henry Clay III, M.D., Hampton, VA
Eisendrath, Stuart James, M.D., San Francisco, CA
Erickson, Miles Alden, M.D., Ocean Springs, MS
Eyerman, James D., M.D., Los Angeles, CA

Farzana, Farida, M.D., Creve Coeur, MO
Feinstein, Pratarnporn, M.D., East Providence, RI
Feldman, Clark Alan, M.D., Los Angeles, CA
Feldman, Ruben E., M.D., Hanford, CA
Fernando, Wewalage J.E.B., M.D., Newtown, CT
Fesler, F. Anne, M.D., Seattle, WA
Fialkov, Martin J., M.D., Sacramento, CA
Fineberg, Donald Eric, M.D., Santa Fe, NM
Fisher, Donald Eugene, M.D., La Mesa, CA
Foster, David Vernon, M.D., Stoneham, MA
Franchini, R. Gregory, M.D., Albuquerque, NM
Freedman, Robert, M.D., Denver, CO
Friedman, David L., M.D., Pacific Palisades, CA

Garcia, Pedro I., M.D., Los Angeles, CA
Gelker, Peter A., M.D., Santa Ana, CA
Gendel, Michael H., M.D., Denver, CO
George, Robert A., M.D., Tigard, OR
Gerstenberger, Dean L., M.D., Flagstaff, AZ
Gibbs, Donald C., M.D., Kaysville, UT
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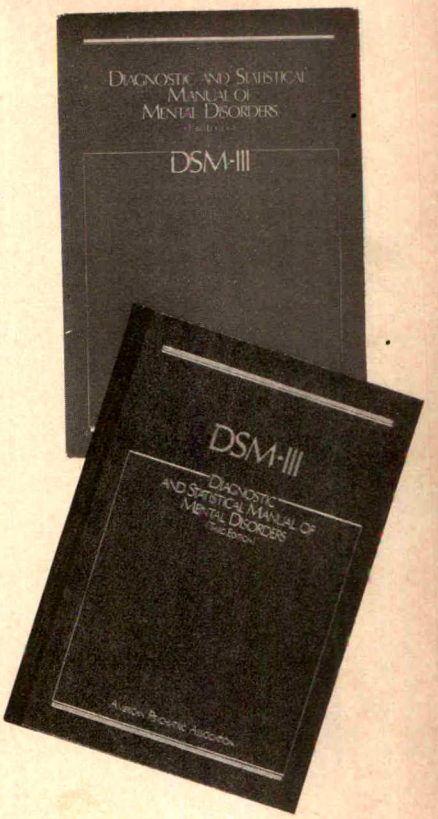
Van Der Heide, Charles J., M.D., Minneapolis, MN[2]

[1]Dr. Fisher's name was omitted from the list of those who passed the examination given in Chicago, IL, October 29-30, 1979 (March 1980 issue).

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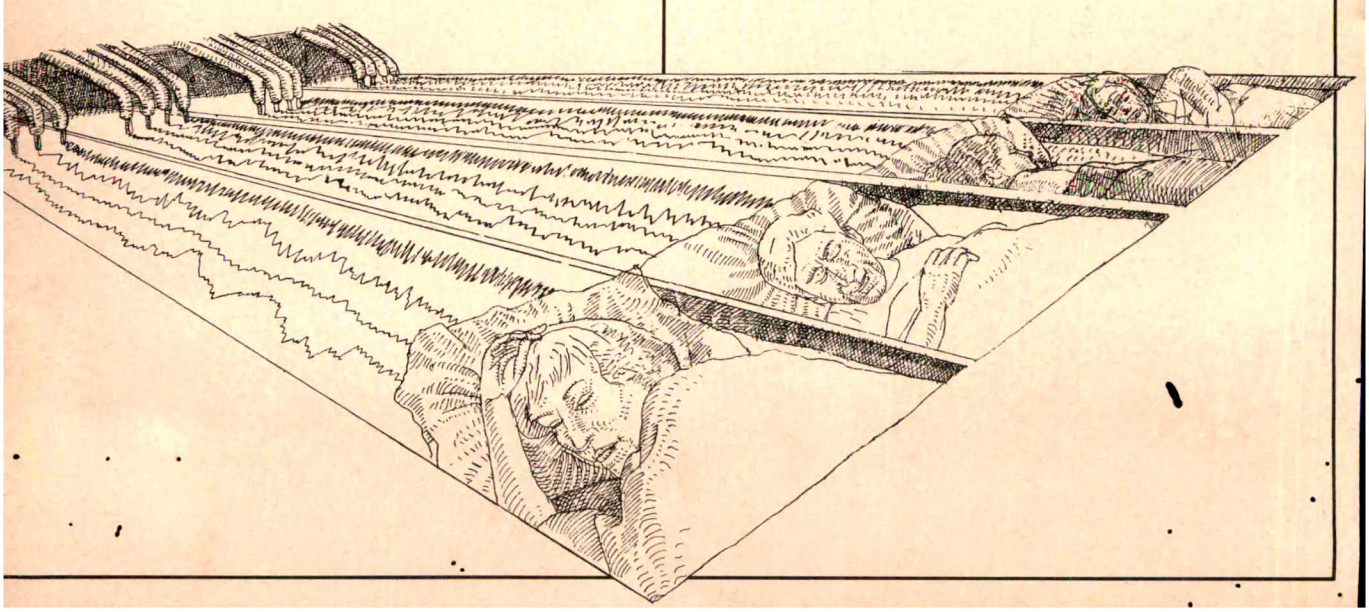
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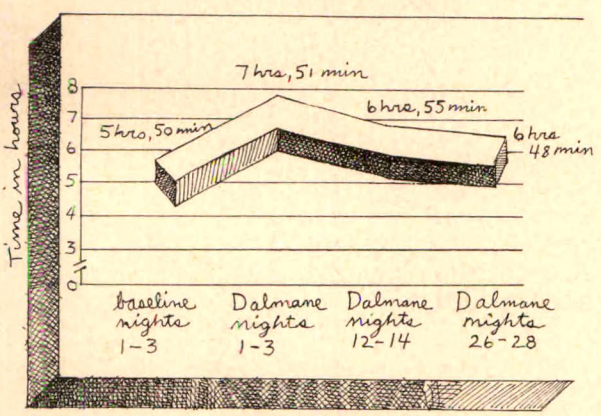
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Adapted from Dement WC, *et al: Behav Med* 5:25-31, Oct 1978

Since most insomnia is transitory in nature, initiate therapy at the lowest effective dose for the shortest time advisable to achieve a predetermined therapeutic goal.

References:
1. Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley NJ
2. Kales A, *et al: Clin Pharmacol Ther 19:* 576-583, May 1976
3. Dement WC, *et al: Behav Med 5:* 25-31, Oct 1978
4. Kales A, *et al: Clin Pharmacol Ther 18:* 356-363, Sep 1975

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☐ Advise patients who ingest alcoholic beverages or other CNS depressants against doing so while on sleep medication. CNS stimulants such as caffeine should also be discontinued.

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Contraindications: Known hypersensitivity to flurazepam HCl.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

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Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies, or with impaired renal or hepatic function. Periodic blood counts and liver and kidney function tests are advised during repeated therapy.
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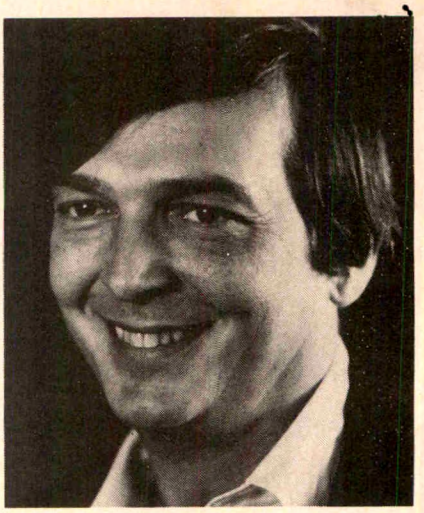
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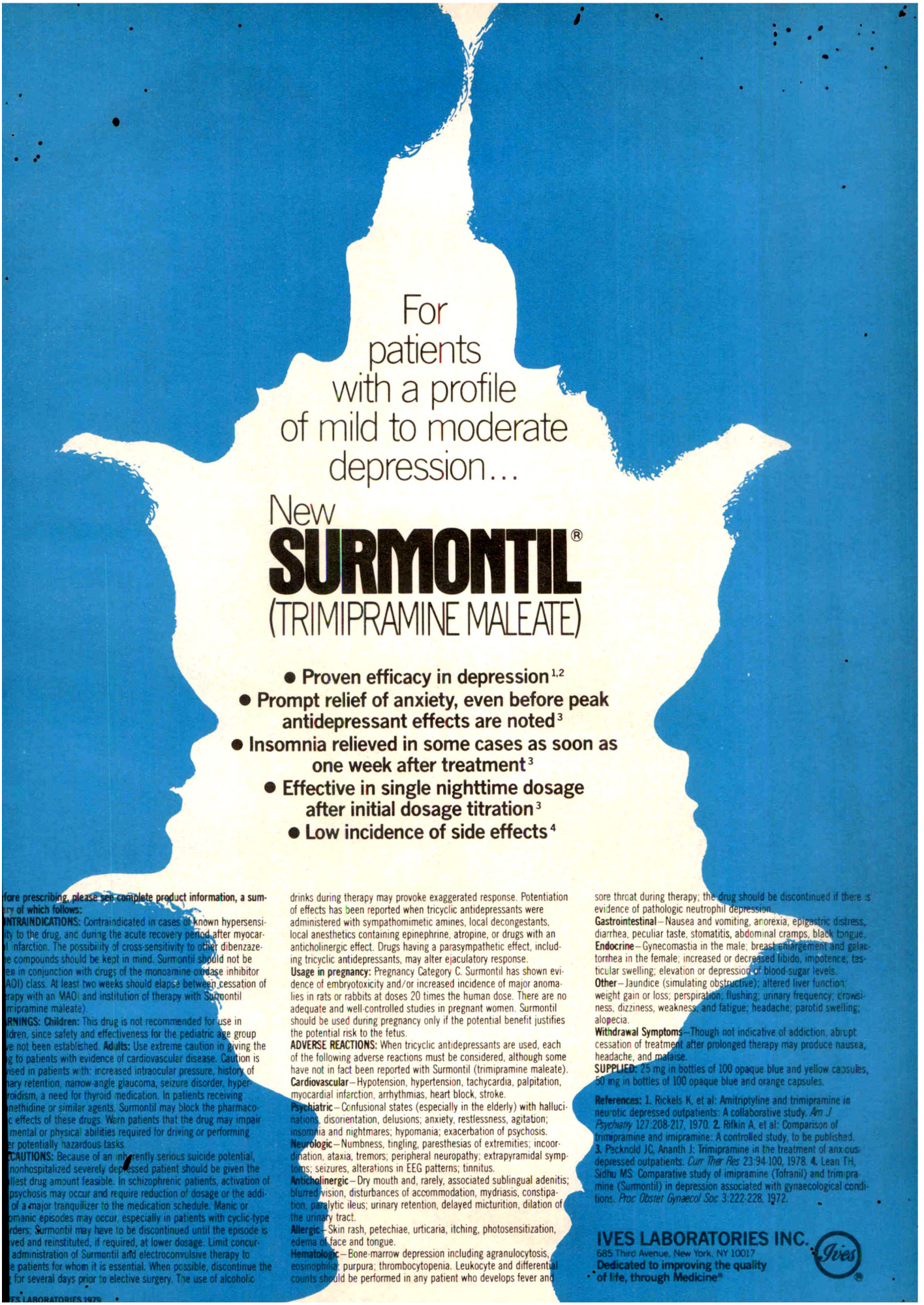
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fore prescribing, please see complete product information, a sum-ary of which follows:

NTRAINDICATIONS: Contraindicated in cases of known hypersensi-ity to the drug, and during the acute recovery period after myocar-l infarction. The possibility of cross-sensitivity to other dibenzaze-ne compounds should be kept in mind. Surmontil should not be en in conjunction with drugs of the monoamine oxidase inhibitor AOI) class. At least two weeks should elapse between cessation of erapy with an MAOI and institution of therapy with Surmontil imipramine maleate).

RNINGS: Children: This drug is not recommended for use in ldren, since safety and effectiveness for the pediatric age group ve not been established. **Adults:** Use extreme caution in giving the g to patients with evidence of cardiovascular disease. Caution is vised in patients with: increased intraocular pressure, history of nary retention, narrow-angle glaucoma, seizure disorder, hyper-roidism, a need for thyroid medication. In patients receiving nethidine or similar agents, Surmontil may block the pharmaco-c effects of these drugs. Warn patients that the drug may impair mental or physical abilities required for driving or performing er potentially hazardous tasks.

CAUTIONS: Because of an inherently serious suicide potential, nonhospitalized severely depressed patient should be given the allest drug amount feasible. In schizophrenic patients, activation of psychosis may occur and require reduction of dosage or the addi-of a major tranquilizer to the medication schedule. Manic or pmanic episodes may occur, especially in patients with cyclic-type rders; Surmontil may have to be discontinued until the episode is ved and reinstituted, if required, at lower dosage. Limit concur-administration of Surmontil and electroconvulsive therapy to e patients for whom it is essential. When possible, discontinue the for several days prior to elective surgery. The use of alcoholic drinks during therapy may provoke exaggerated response. Potentiation of effects has been reported when tricyclic antidepressants were administered with sympathomimetic amines, local decongestants, local anesthetics containing epinephrine, atropine, or drugs with an anticholinergic effect. Drugs having a parasympathetic effect, including tricyclic antidepressants, may alter ejaculatory response.

Usage in pregnancy: Pregnancy Category C. Surmontil has shown evidence of embryotoxicity and/or increased incidence of major anomalies in rats or rabbits at doses 20 times the human dose. There are no adequate and well-controlled studies in pregnant women. Surmontil should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

ADVERSE REACTIONS: When tricyclic antidepressants are used, each of the following adverse reactions must be considered, although some have not in fact been reported with Surmontil (trimipramine maleate).

Cardiovascular—Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric—Confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurologic—Numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures, alterations in EEG patterns; tinnitus.

Anticholinergic—Dry mouth and, rarely, associated sublingual adenitis; blurred vision, disturbances of accommodation, mydriasis, constipation, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic—Skin rash, petechiae, urticaria, itching, photosensitization, edema of face and tongue.

Hematologic—Bone-marrow depression including agranulocytosis, eosinophilia; purpura; thrombocytopenia. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathologic neutrophil depression.

Gastrointestinal—Nausea and vomiting, anorexia, epigastric distress, diarrhea, peculiar taste, stomatitis, abdominal cramps, black tongue.

Endocrine—Gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido, impotence; testicular swelling; elevation or depression of blood-sugar levels.

Other—Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration, flushing; urinary frequency; drowsiness, dizziness, weakness, and fatigue; headache; parotid swelling; alopecia.

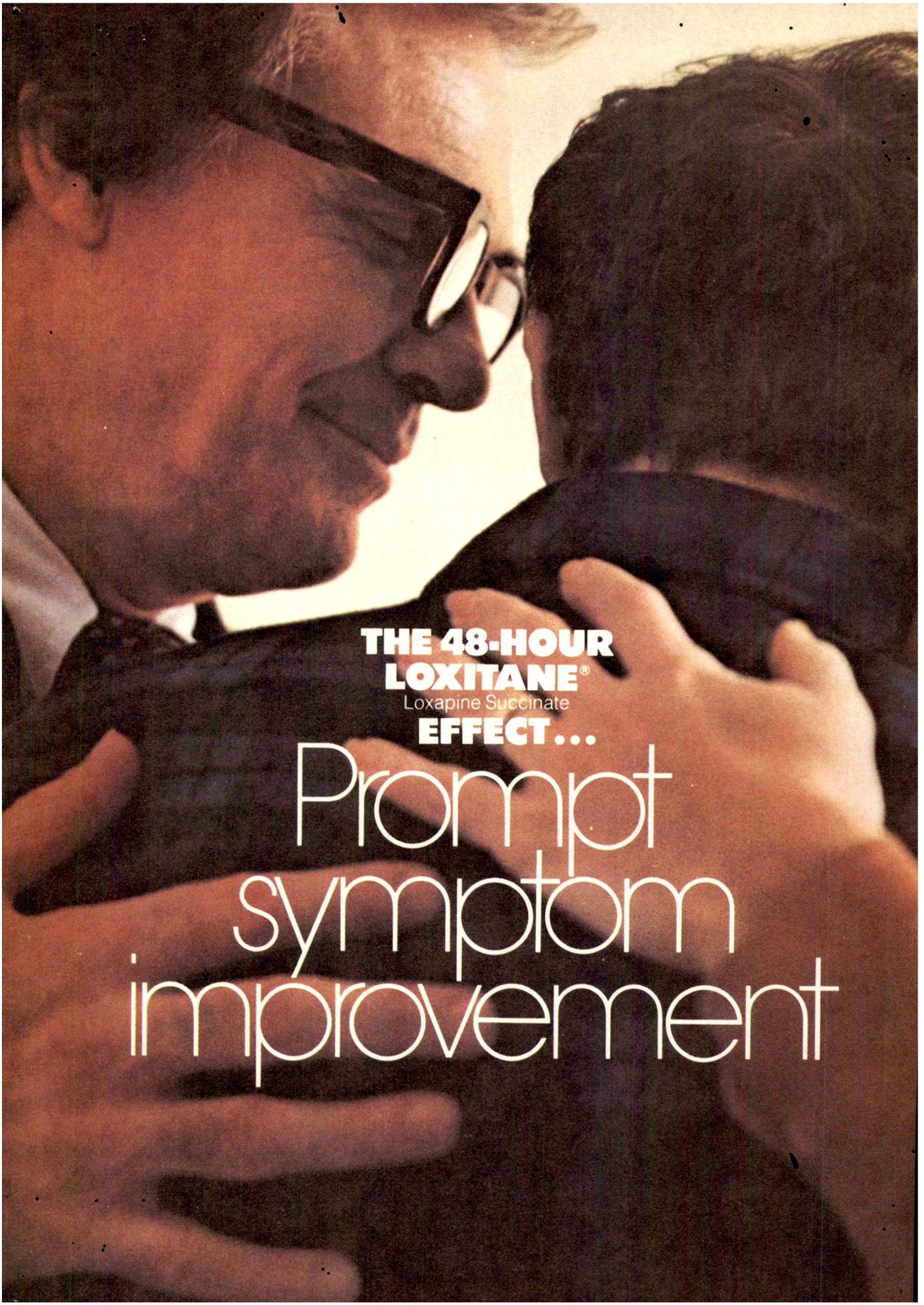
Withdrawal Symptoms—Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache, and malaise.

SUPPLIED: 25 mg in bottles of 100 opaque blue and yellow capsules, 50 mg in bottles of 100 opaque blue and orange capsules.

References: 1. Rickels K, et al: Amitriptyline and trimipramine in neurotic depressed outpatients: A collaborative study. *Am J Psychiatry* 127:208-217, 1970. 2. Rifkin A, et al: Comparison of trimipramine and imipramine: A controlled study, to be published. 3. Pecknold JC, Ananth J: Trimipramine in the treatment of anxious depressed outpatients. *Curr Ther Res* 23:94-100, 1978. 4. Lean TH, Sidhu MS: Comparative study of imipramine (Tofranil) and trimipramine (Surmontil) in depression associated with gynaecological conditions. *Proc Obstet Gynaecol Soc* 3:222-228, 1972.

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THE 48-HOUR
LOXITANE®
Loxapine Succinate
EFFECT...
Prompt
symptom
improvement

Early response—the first step toward early resocialization

After a relatively short time on LOXITANE® Loxapine Succinate, hospitalized schizophrenic patients usually become easier to reach, easier to manage, more sociable, and cooperative. A series of clinical studies[1-4] shows an impressive decrease in conceptual disorganization, thinking disorder, hallucinatory behavior and anxiety-depression during the first 48 hours.

The results in one group of 17 young adult schizophrenics treated at a university hospital typify general clinical experience. In the first 48 hours, these patients achieved a mean overall symptomatic improvement of more than 20% in BPRS.[1] (See chart)

Continued overall improvement... helps facilitate a return to more normal activities

Near maximal control was reported by week four; many behavior patterns approached normal. In other studies, a mean overall improvement of almost 80% was seen by 28 days.[2,3] Prompt therapeutic response and steady progress can help strengthen the patient's capacity to cope with daily living and relationships.

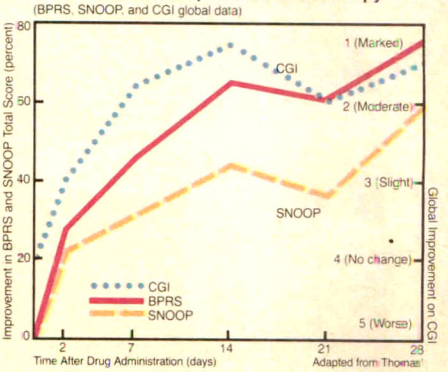
Minimal risk of certain toxicities... encourages compliance

On LOXITANE, physical health is rarely compromised by certain toxicities.* Renal, hematopoietic, cardiotoxicity, phototoxicity and photosensitivity have not been reported to date.* Therapy in most cases is not interrupted by intolerable side effects. Sexual side effects have not been a significant problem; anticholinergic effects are usually mild, and extrapyramidal reactions are generally easily controlled. Well-tolerated LOXITANE means less frequent need to interrupt therapy—fewer problems with patient compliance.

Note: LOXITANE, as with certain other neuroleptics, lowers the convulsive threshold and should be used with extreme caution in patients with a history of convulsive disorders.

*Although not reported to date, the possibility of phototoxicity, cardiac, renal, or blood toxicity cannot be ruled out at this time. Transient liver enzyme changes not definitely related to LOXITANE have been reported.

Mean improvement in 17 schizophrenic patients on LOXITANE® Loxapine Succinate therapy
(BPRS, SNOOP, and CGI global data)



CGI (Clinical Global Impressions)
BPRS (Brief Psychiatric Rating Scale)
SNOOP (Systematic Nursing Observation of Psychopathology)

Loxitane®
LOXAPINE SUCCINATE
Lederle

5 mg, 10 mg, 25 mg, 50 mg capsules

See LOXITANE Brief Summary on following page.

For the difference that begins with rapid action...



Loxitane®
LOXAPINE SUCCINATE
Lederle

Recommended Daily Dosage

Dosage	Initially: 20-50 mg During first 24 hours: up to 100 mg Usual therapeutic level: 150 mg/day Maintenance: 60-100 mg/day Maximum: 250 mg/day Dosage may be divided as necessary. Reduced dosage in geriatrics and adolescents should be considered.

Brief Summary
LOXITANE® Loxapine Succinate Capsules
LOXITANE® C Loxapine Hydrochloride Oral Concentrate
LOXITANE® IM Loxapine Hydrochloride

INDICATIONS: Manifestations of schizophrenia.
CONTRAINDICATIONS: Comatose or severely depressed states; hypersensitivity to the drug.
WARNINGS: Safe use during pregnancy or lactation has not been established; weigh potential benefits against possible hazards. Not recommended for use in children under 16. May impair mental and/or physical abilities especially during early therapy; warn ambulatory patients about activities requiring alertness and concomitant use of alcohol or other CNS depressants. Not recommended for management of behavioral complications in mentally retarded patients.
PRECAUTIONS: Use with extreme caution in patients with a history of convulsive disorders; use with caution in patients with cardiovascular disease or in those with glaucoma or a tendency to urinary retention particularly when on concomitant anticholinergic medication. Loxapine has an antiemetic effect in animals which might occur in man masking signs of overdosage of toxic drugs and obscuring intestinal obstruction or brain tumor. Since possible ocular toxicity cannot be excluded, observe carefully for pigmentary retinopathy and lenticular pigmentation. Slightly higher incidence of extrapyramidal effects possible following IM administration.
ADVERSE REACTIONS: *CNS effects,* other than extrapyramidal, infrequent. Mild drowsiness may occur at beginning of therapy or upon dosage increase, usually subsides with continued therapy. Sedation, dizziness, faintness, staggering gait, muscle twitching, weakness and confusional states have been reported. Extrapyramidal reactions often occur early in treatment, manifested by Parkinson-like symptoms (tremor, rigidity, excessive salivation, masked facies, akathisia); controllable by dosage reduction or antiparkinson drugs at usual dosages. Dystonic and dyskinetic reactions, while less frequently occurring, may be more severe, requiring dosage reduction or temporary withdrawal plus appropriate counteractive drugs. *Persistent Tardive Dyskinesia* may appear during prolonged therapy or following discontinuance, the risk greater in the elderly, especially females, on high dosage. Symptoms, persistent and in some patients apparently irreversible, are characterized by rhythmical involuntary movement of the tongue, face, mouth and jaw sometimes accompanied by involuntary movement of extremities. Since there is no known effective treatment, discontinue all antipsychotic drugs if symptoms appear. Reinstitution of treatment, increased dosage or switching to another agent may mask syndrome. The syndrome may not develop if medication is stopped when fine vermicular movements of the tongue first appear. *Cardiovascular Effects:* Tachycardia, hypotension, hypertension, lightheadedness and syncope. ECG changes, not known to be related to loxapine use, have been reported. *Skin:* Dermatitis, edema of face, pruritus, seborrhea. Possible photosensitivity and/or phototoxicity; skin rashes of unknown etiology seen in a few patients in hot summer months. *Anticholinergic:* Dry mouth, nasal congestion, constipation, blurred vision (more likely to occur with concomitant use of antiparkinson agents). *Other:* Nausea, vomiting, weight gain or loss, dyspnea, ptosis, hyperpyrexia, flushed facies, headache, paresthesia, polydipsia. Rarely, galactorrhea and menstrual irregularity of unknown etiology.

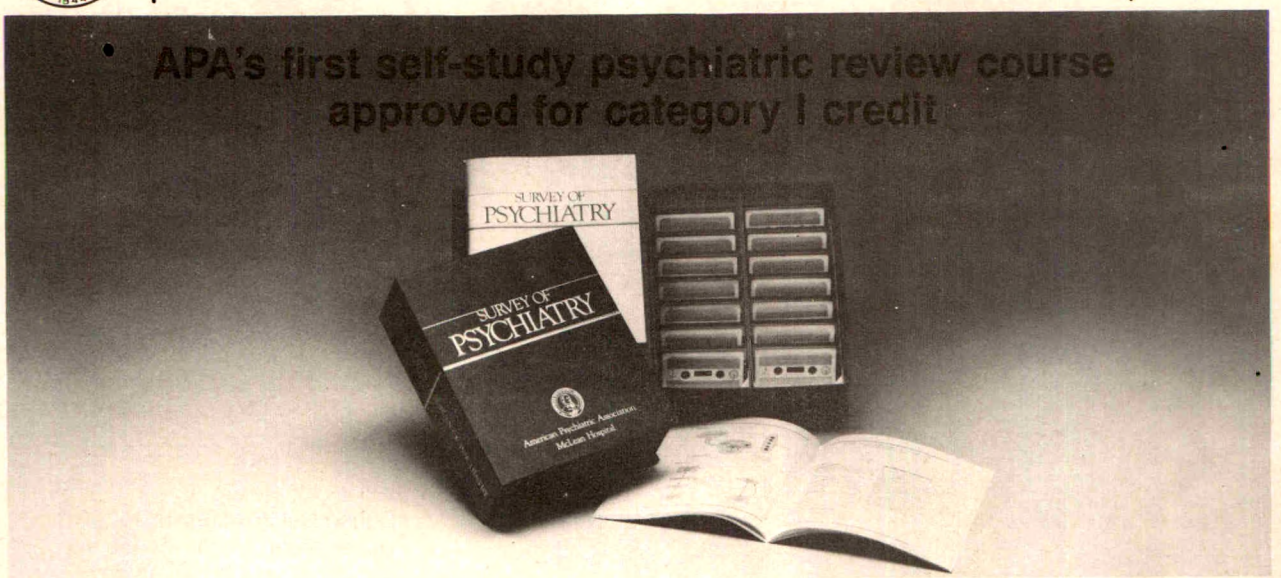
References: 1. Thomas JL: Loxapine oral liquid concentrate in the treatment of young adult patients with acute schizophrenic symptoms, *Curr Ther Res* 25:371-377, March 1979. **2.** Sharma T: Rapid response to loxapine in acute schizophrenia, *Curr Ther Res* 25:366-370, March 1979. **3.** Serban G: Loxapine in acute schizophrenic disorder, *Curr Ther Res* 25:139-143, January 1979. **4.** Zisook S et al.: Loxapine succinate (LOXITANE®) in the outpatient treatment of acutely ill schizophrenic patients, *Curr Ther Res* 24:415-426, August 1978.

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THE CHRONIC MENTAL PATIENT

Report of a Conference held in January 1978
Edited by
John A. Talbott, M.D.

with contributions by James T. Barter, M.D., Paul R. Friedman, J.D., Raymond Glasscote, M.A., Trevor D. Glenn, M.D., Norman V. Lourie, M.S.W., W. Walter Menninger, M.D., Arthur T. Meyerson, M.D., Kenneth Minkoff, M.D., Samuel Muszynski, M.S.W., Lucy D. Ozarin, M.D., Gordon L. Paul, Ph.D., Ronald Peterson, Steven S. Sharfstein, M.D., Judith Clark Turner, Jane Bloom Yohalem, J.D.

This 277-page Report of the Conference, sponsored by APA and President Carter's Commission on Mental Health, identifies the chronic mental patient population, spells out where they are and what their needs and rights are. It specifies what programs work and what programs do not work in meeting the needs of these patients. It elaborates on the obstacles to implementing effective programs and the economic issues involved. It delineates the pros and cons of case management and specifies responsibility for coordinating, implementing, and monitoring services to chronic mental patients.

Finally, it proposes a *Call to Action* which opens with this statement: "There is no more urgent concern than the needs of the chronic mentally ill who suffer from severe, persistent, or recurrent mental illnesses with residual social and vocational disabilities. As a result of the deinstitutionalization programs of the past decade and the continuing growth of high risk populations that generate chronically ill, the problems associated with the care of these patients constitute a national crisis."

Since the Conference, the APA Assembly and the Board of Trustees have both approved the "Call to Action" which calls upon the APA to take the lead in undertaking programs to elevate the prestige and value of work with chronic mentally ill patients.

It follows that all APA members should be thoroughly versed in the current problems of this chronic patient population. This is best accomplished by reading this Conference Report now available from APA Publications Services at $11.00 a copy.

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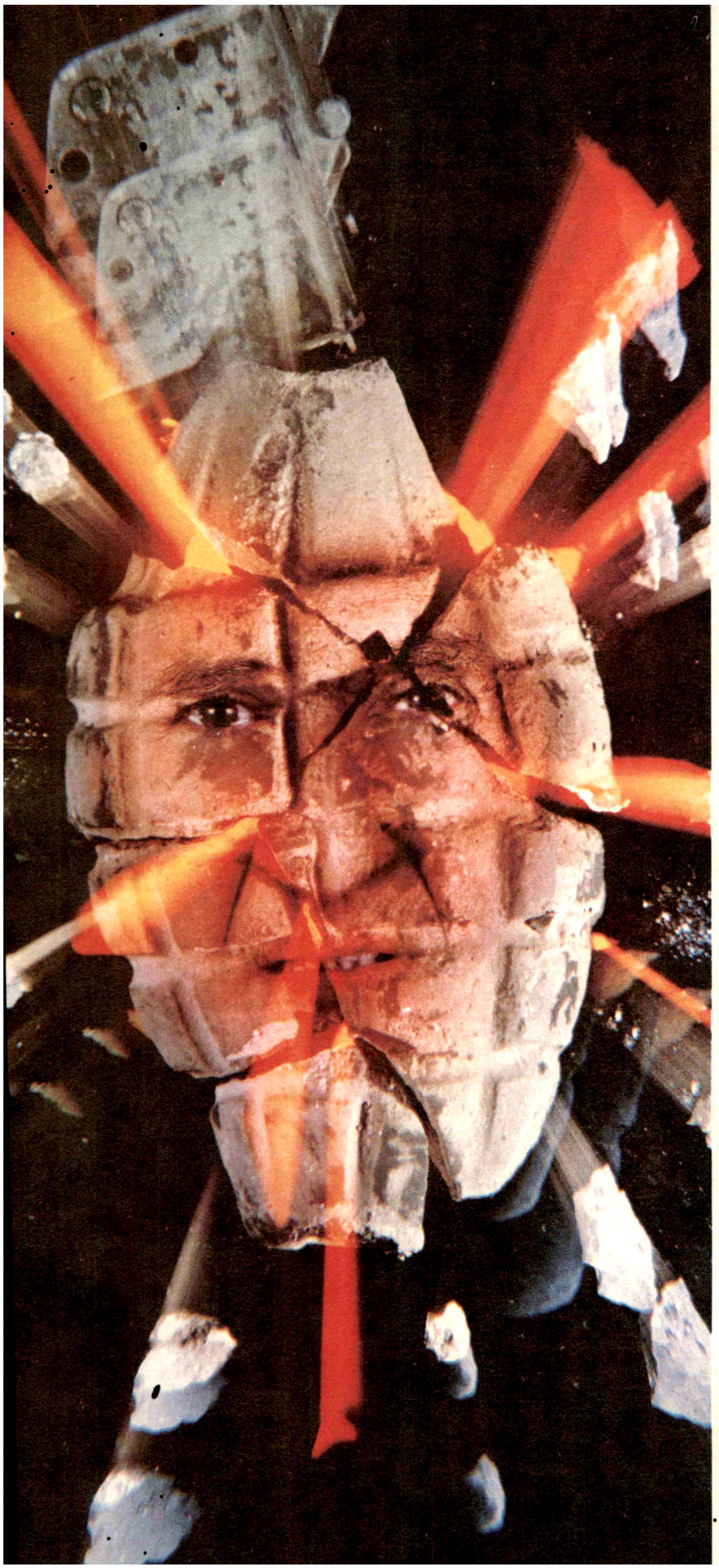
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Introducing...

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(thiothixene HCl)
Intramuscular
For Injection
5 mg/ml



new potency for rapid control of acute psychiatric emergencies

Now, Navane Intramuscular 5 mg per ml provides a more concentrate dosage form for rapid control of severely disturbed behavior associat ed with acute psychosis. Navane Intramuscular, in carefully monitor ed doses, "...can produce significan improvement within hours and increase the accessibility of these [acutely disturbed] patients to mor gradual methods of pharmacologica treatment as well as to socio-enviro mental intervention and psycho-therapy."[1]

Rapid tranquilization usually ca be achieved without excessive seda-tion.[1] Clinically significant hypoten-sion, often seen with other injectab antipsychotic agents, is rare with Navane Intramuscular therapy.[1-3] If extrapyramidal symptoms occur, they are usually readily controlled by dosage adjustment or antiparkin son agents.

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For a brief summary of Navane prescribing information, including adverse reactions and contraindications, please see the following page of this advertisement.

Navane® (thiothixene) (thiothixene hydrochloride)

Capsules: 1 mg, 2 mg, 5 mg, 10 mg, 20 mg Concentrate: 5 mg/ml Intramuscular: 2 mg/ml, 5 mg/ml

BRIEF SUMMARY OF PRESCRIBING INFORMATION
Navane® (thiothixene)
Capsules: 1 mg, 2 mg, 5 mg, 10 mg, 20 mg
(thiothixene hydrochloride) Concentrate: 5 mg/ml,
Intramuscular: 2 mg/ml, 5 mg/ml

Contraindications. Navane (thiothixene) is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy*– Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration of Navane to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children– The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration– As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane (thiothixene). Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudo-parkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leukocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution; Navane For Injection– Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane (thiothixene) Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate– In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Stimulants that may cause convulsions (e.g. picrotoxin or pentylenetetrazol) should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg of thiothixene in bottles of 100, 1,000, and unit-dose pack of 100 (10 x 10's). Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100, 500, and unit-dose pack of 100 (10 x 10's).

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg, and in 30 ml (1 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, and 5 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10 vials. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

Navane (thiothixene hydrochloride) Intramuscular For Injection is available in amber glass vials in packages of 10 vials. When reconstituted with 2.2 ml of Sterile Water for Injection, each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene, and 59.6 mg of mannitol. The reconstituted solution of Navane Intramuscular For Injection may be stored for 48 hours at room temperature before discarding.

References: 1. Stotsky BA: Relative efficacy of parenteral haloperidol and thiothixene for the emergency treatment of acutely excited and agitated patients. *Dis Nerv Syst* 38:967-973, December 1977. 2. Brauzer B, Goldstein BJ: Comparative effects of intramuscular thiothixene and trifluoperazine in psychotic patients. *J Clin Pharmacol* 8:400-403, November-December 1968. 3. Gallant DM, Bishop MP, Bishop C, et al: Thiothixene: A controlled evaluation of the intramuscular antipsychotic preparation. *Curr Ther Res* 10:561-565, November 1968.

For additional information on Navane, please consult your Roerig representative or write to: Roerig Medical Department, 235 East 42nd Street, New York, NY 10017.

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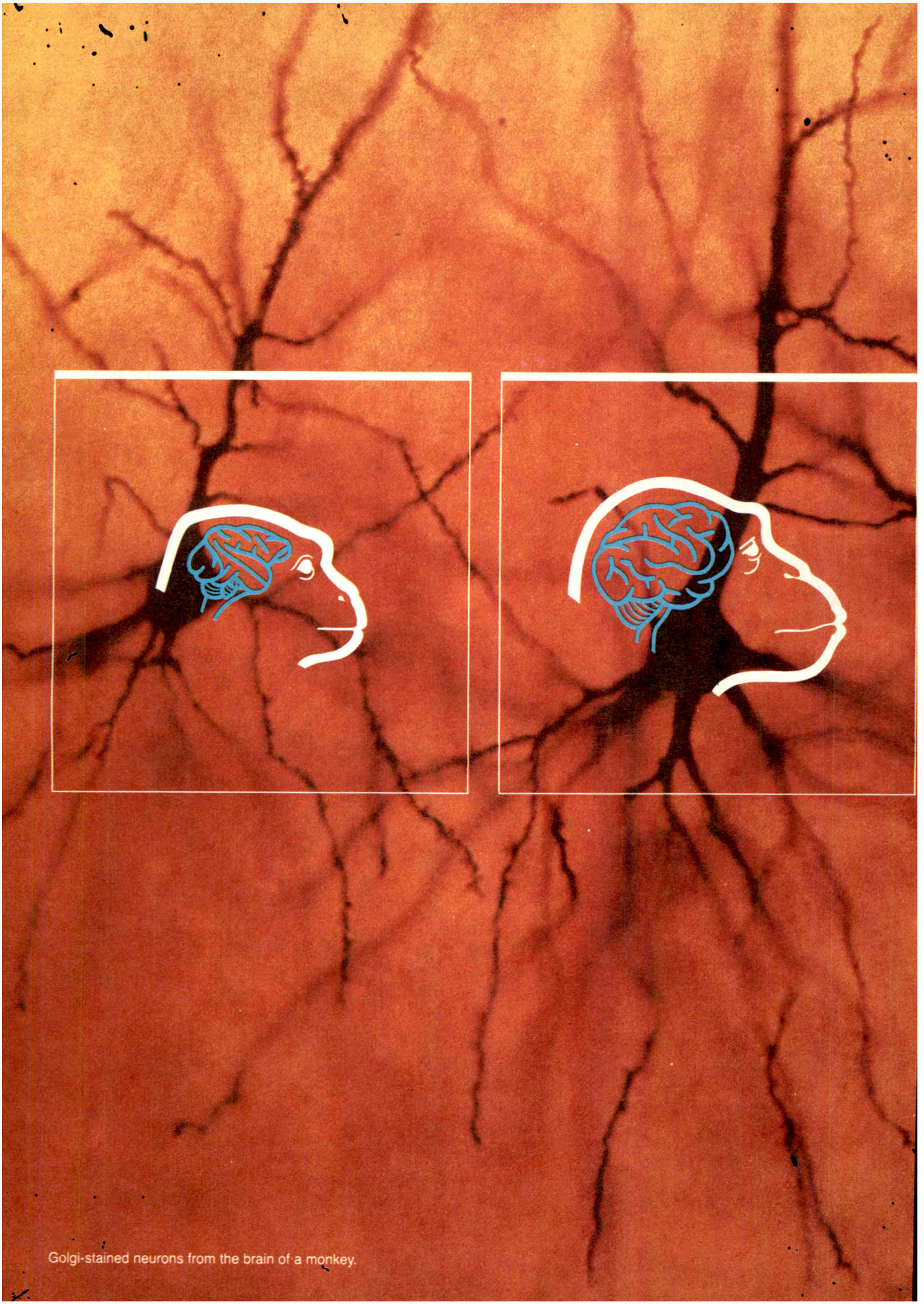
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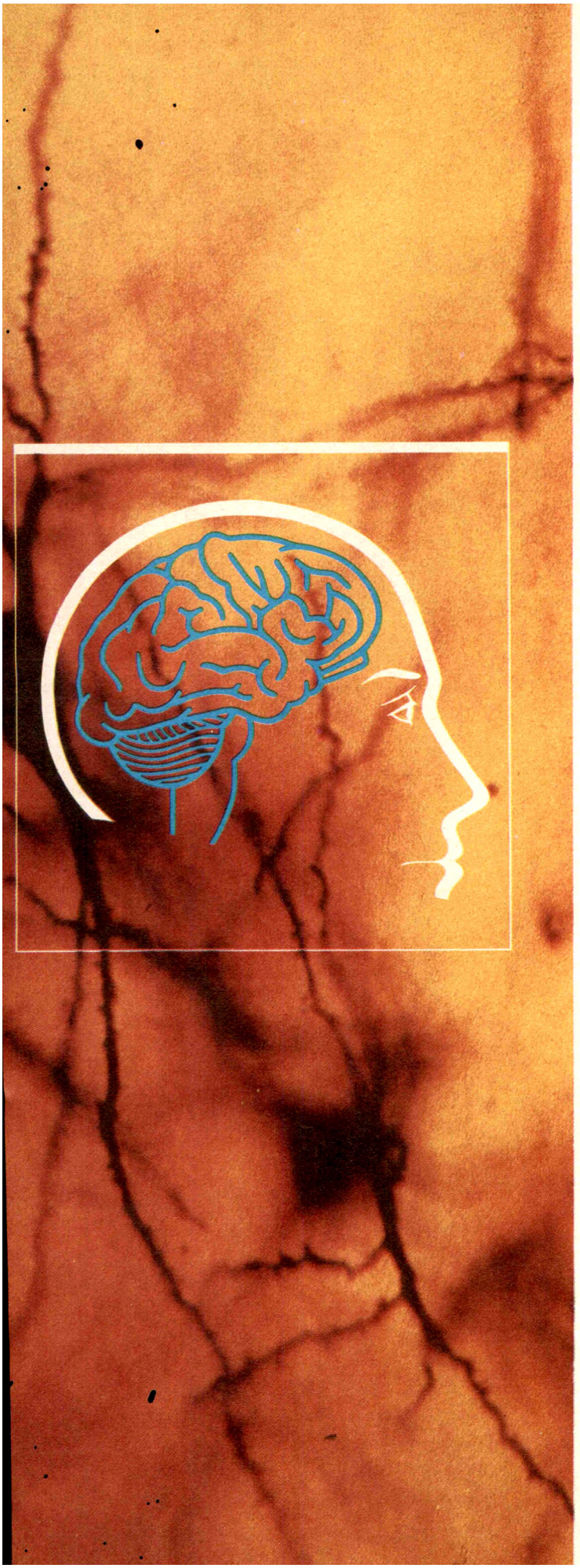
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Please see next page for summary of product information.

Librium® 5mg, 10mg, 25mg capsules
chlordiazepoxide HCl/Roche

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Relief of anxiety and tension occurring alone or accompanying various disease states. Efficacy beyond four months not established by systematic clinical studies. Periodic reassessment of therapy recommended.

Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Warn patients that mental and/or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

Usual Daily Dosage: Individualize for maximum beneficial effects. *Oral–Adults:* Mild and moderate anxiety and tension, 5 or 10 mg *t.i.d.* or *q.i.d.*; severe states, 20 or 25 mg *t.i.d.* or *q.i.d. Geriatric patients:* 5 mg *b.i.d.* to *q.i.d.* (See Precautions.)

Supplied: Librium® (chlordiazepoxide HCl) *Capsules,* 5 mg, 10 mg and 25 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available singly and in trays of 10. Libritabs® (chlordiazepoxide) *Tablets,* 5 mg, 10 mg and 25 mg—bottles of 100 and 500. With respect to clinical activity, capsules and tablets are indistinguishable.

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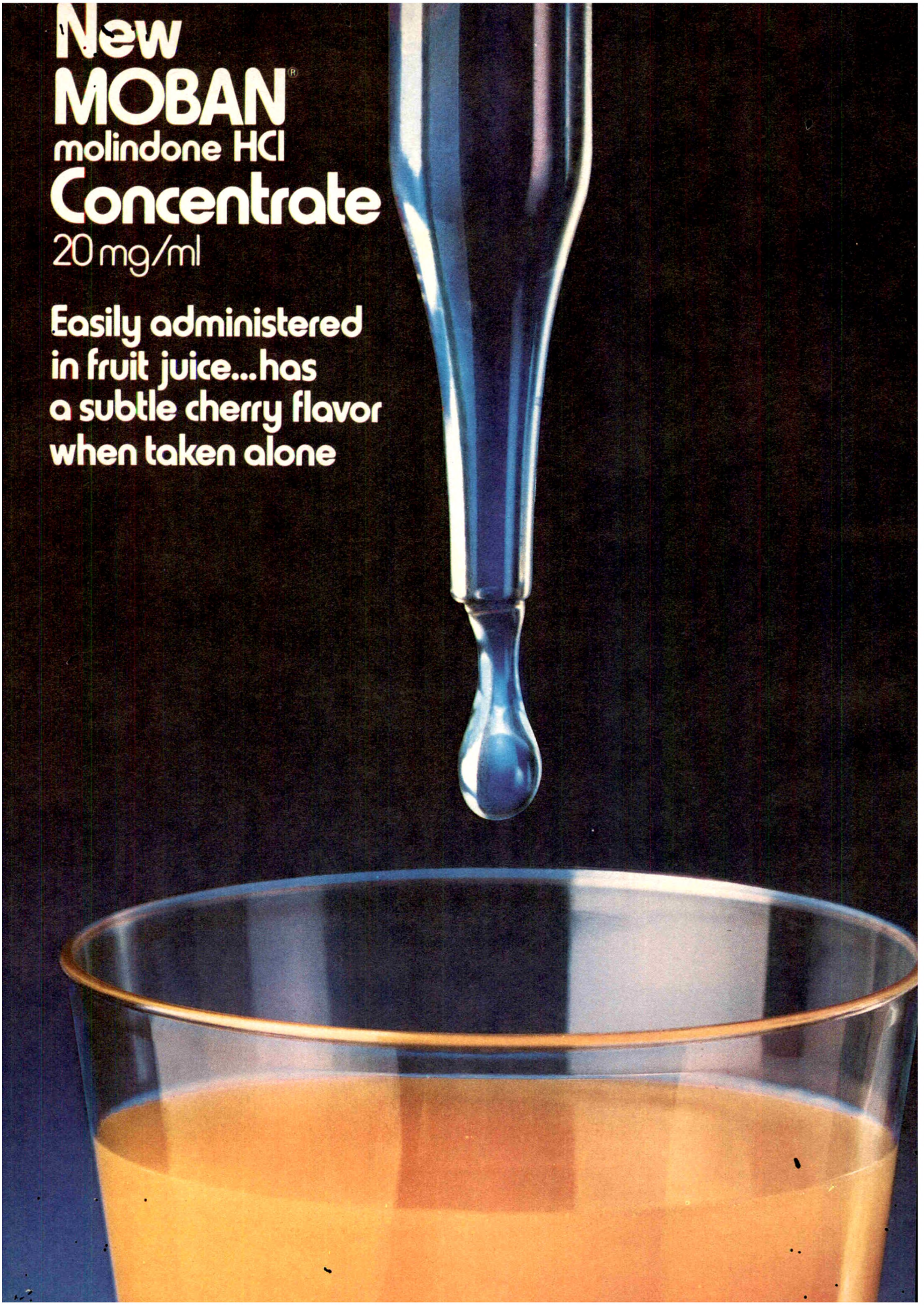
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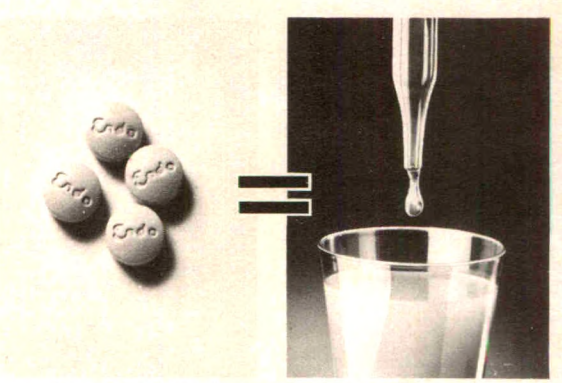
An effective antipsychotic agent with a favorable benefit/risk profile

NO incidence	excessive weight gain hepatotoxic changes ophthalmological changes persistent tardive dyskinesia skin pigmentation thyroid function changes tolerance development
LOW incidence	adverse drug interactions hematological changes hypotension nonpersistent symptoms of tardive dyskinesia sedation
MODERATE incidence	extrapyramidal reactions

This table compiled from clinical and field reports, according to the best available information.

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New MOBAN®
molindone HCl
Concentrate
20 mg/ml



four 25 mg tablets = 5 milliliters (100 mg)

Concentrated for maximum convenience of higher dosages...easy titration... enhanced compliance

Initial dosage with MOBAN®

- usual starting dose is 50-75 mg/day
- may be increased to 100 mg/day in three to four days
- may be adjusted up or down according to response
- some patients with severe symptomatology may require an increase to 225 mg/day
- elderly and debilitated patients should be started on lower dosage

Please see next page for prescribing information.

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A53

An effective antipsychotic agent

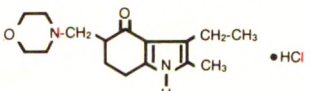
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molindone HCl

New dosage form... Concentrate 20 mg/ml

continuing...Tablets 5 mg, 10 mg, 25 mg

DESCRIPTION MOBAN (molindone hydrochloride) is a dihydroindolone compound which is not structurally related to the phenothiazines, the butyrophenones or the thioxanthenes.

MOBAN is 3-ethyl-6, 7-dihydro-2-methyl-5-(morpholinomethyl) indol-4 (5H)-one hydrochloride. It is a white crystalline powder, freely soluble in water and alcohol and has a molecular weight of 312.67.



MOLINDONE HYDROCHLORIDE

ACTIONS MOBAN (molindone hydrochloride) has a pharmacological profile in laboratory animals which predominantly resembles that of major tranquilizers causing reduction of spontaneous locomotion and aggressiveness, suppression of a conditioned response and antagonism of the bizarre stereotyped behavior and hyperactivity induced by amphetamines. In addition, MOBAN antagonizes the depression caused by the tranquilizing agent tetrabenazine.

In human clinical studies tranquilization is achieved in the absence of muscle relaxing or incoordinating effects. Based on EEG studies, MOBAN exerts its effect on the ascending reticular activating system.

Human metabolic studies show MOBAN (molindone hydrochloride) to be rapidly absorbed and metabolized when given orally. Unmetabolized drug reached a peak blood level at 1.5 hours. Pharmacological effect from a single oral dose persists for 24-36 hours. There are 36 recognized metabolites with less than 2-3% unmetabolized MOBAN being excreted in urine and feces.

INDICATIONS MOBAN (molindone hydrochloride) is indicated in the management of the manifestations of schizophrenia.

CONTRAINDICATIONS MOBAN (molindone hydrochloride) is contraindicated in severe central nervous system depression (alcohol, barbiturates, narcotics, etc.) or comatose states, and in patients with known hypersensitivity to the drug.

WARNINGS Usage in Pregnancy: Studies in pregnant patients have not been carried out. Reproduction studies have been performed in the following animals:

Pregnant Rats oral dose— 20 mg/kg/day—10 days
no adverse effect
40 mg/kg/day—10 days
no adverse effect

Pregnant Mice oral dose— 20 mg/kg/day—10 days
slight increase resorptions
40 mg/kg/day—10 days
slight increase resorptions

Pregnant Rabbits oral dose— 5 mg/kg/day—12 days
no adverse effect
10 mg/kg/day—12 days
no adverse effect
20 mg/kg/day—12 days
no adverse effect

Animal reproductive studies have not demonstrated a teratogenic potential. The anticipated benefits must be weighed against the unknown risks to the fetus if used in pregnant patients.

Nursing Mothers: Data are not available on the content of MOBAN (molindone hydrochloride) in the milk of nursing mothers.

Usage in Children: Use of MOBAN (molindone hydrochloride) in children below the age of twelve years is not recommended because safe and effective conditions for its usage have not been established.

MOBAN has not been shown effective in the management of behavioral complications in patients with mental retardation.

PRECAUTIONS Some patients receiving MOBAN (molindone hydrochloride) may note drowsiness initially and they should be advised against activities requiring mental alertness until their response to the drug has been established.

Increased activity has been noted in patients receiving MOBAN. Caution should be exercised where increased activity may be harmful.

MOBAN does not lower the seizure threshold in experimental animals to the degree noted with more sedating antipsychotic drugs. However, in humans convulsive seizures have been reported in a few instances.

EDO 854M580

The physician should be aware that this tablet preparation contains calcium sulfate as an excipient and that calcium ions may interfere with the absorption of preparations containing phenytoin sodium and tetracyclines.

MOBAN (molindone hydrochloride) has an antiemetic effect in animals. A similar effect may occur in humans and may obscure signs of intestinal obstruction or brain tumor.

ADVERSE REACTIONS—CNS EFFECTS The most frequently occurring effect is initial drowsiness that generally subsides with continued usage of the drug or lowering of the dose.

Noted less frequently were depression, hyperactivity and euphoria.

Neurological—Extrapyramidal Reactions Extrapyramidal reactions noted below may occur in susceptible individuals and are usually reversible with appropriate management.

Akathisia Motor restlessness may occur early.

Parkinson Syndrome Akinesia, characterized by rigidity, immobility and reduction of voluntary movements and tremor, have been observed. Occurrence is less frequent than akathisia.

Dystonic Syndrome Prolonged abnormal contractions of muscle groups occur infrequently. These symptoms may be managed by the addition of a synthetic antiparkinson agent (other than L-dopa), small doses of sedative drugs, and/or reduction in dosage.

Autonomic Nervous System Occasionally blurring of vision, tachycardia, nausea, dry mouth and salivation have been reported. Urinary retention and constipation may occur particularly if anticholinergic drugs are used to treat extrapyramidal symptoms.

Hematological There have been rare reports of leucopenia and leucocytosis. If such reactions occur, treatment with MOBAN may continue if clinical symptoms are absent. Alterations of blood glucose, liver function tests, B.U.N., and red blood cells have not been considered clinically significant.

Metabolic and Endocrine Effects Alteration of thyroid function has not been significant. Amenorrhea has been reported infrequently. Resumption of menses in previously amenorrheic women has been reported. Initially heavy menses may occur. Galactorrhea and gynecomastia have been reported infrequently. Increase in libido has been noted in some patients. Impotence has not been reported. Although both weight gain and weight loss have been in the direction of normal or ideal weight, excessive weight gain has not occurred with MOBAN.

Cardiovascular Rare, transient, non-specific T wave changes have been reported on E.K.G. Association with a clinical syndrome has not been established. Rarely has significant hypotension been reported.

Ophthalmological Lens opacities and pigmentary retinopathy have not been reported where patients have received MOBAN (molindone hydrochloride). In some patients, phenothiazine induced lenticular opacities have resolved following discontinuation of the phenothiazine while continuing therapy with MOBAN.

Skin Early, non-specific skin rash, probably of allergic origin, has occasionally been reported. Skin pigmentation has not been seen with MOBAN usage alone.

MOBAN (molindone hydrochloride) has certain pharmacological similarities to other antipsychotic agents. Because adverse reactions are often extensions of the pharmacological activity of a drug, all of the known pharmacological effects associated with other antipsychotic drugs should be kept in mind when MOBAN is used. Upon abrupt withdrawal after prolonged high dosage an abstinence syndrome has not been noted.

Tardive Dyskinesia Although rarely reported with MOBAN (molindone hydrochloride) symptoms were reversible upon discontinuation of therapy.

Tardive dyskinesia associated with other agents has appeared in some patients on long-term therapy and has also appeared after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). There may be involuntary movements of extremities.

There is no known effective treatment of tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time the syndrome may not develop.

DOSAGE AND ADMINISTRATION Initial and maintenance doses of MOBAN (molindone hydrochloride) should be individualized.

Initial Dosage Schedule The usual starting dosage is 50-75 mg/day.
- —Increase to 100 mg/day in 3 or 4 days.
- —Based on severity of symptomatology, dosage may be titrated up or down depending on individual patient response.
- —An increase to 225 mg/day may be required in patients with severe symptomatology.

Elderly and debilitated patients should be started on lower dosage.

Maintenance Dosage Schedule
1. Mild—5 mg-15 mg three or four times a day.
2. Moderate—10 mg-25 mg three or four times a day.
3. Severe—225 mg/day may be required.

DRUG INTERACTIONS Potentiation of drugs administered concurrently with MOBAN (molindone hydrochloride) has not been reported. Additionally, animal studies have not shown increased toxicity when MOBAN is given concurrently with representative members of three classes of drugs (i.e., barbiturates, chloral hydrate and antiparkinson drugs).

MANAGEMENT OF OVERDOSAGE Symptomatic, supportive therapy should be the rule.

Gastric lavage is indicated for the reduction of absorption of MOBAN (molindone hydrochloride) which is freely soluble in water.

Since the adsorption of MOBAN (molindone hydrochloride) by activated charcoal has not been determined, the use of this antidote must be considered of theoretical value.

Emesis in a comatose patient is contraindicated. Additionally, while the emetic effect of apomorphine is blocked by MOBAN in animals, this blocking effect has not been determined in humans.

A significant increase in the rate of removal of unmetabolized MOBAN (molindone hydrochloride) from the body by forced diuresis, peritoneal or renal dialysis would not be expected. (Only 2% of a single ingested dose of MOBAN is excreted unmetabolized in the urine.)

However, poor response of the patient may justify use of these procedures. While the use of laxatives or enemas might be based on general principles, the amount of unmetabolized MOBAN in feces is less than 1%. Extrapyramidal symptoms have responded to the use of diphenhydramine (Benadryl*) and the synthetic anticholinergic antiparkinson agents, (i.e., Artane*, Cogentin*, Akineton*).

HOW SUPPLIED As tablets in bottles with potencies and colors as follows:

100's and 1000's:
5 mg orange
10 mg lavender
25 mg light green

As a concentrate containing 20 mg molindone hydrochloride per ml in 4 oz. (120ml) bottles.

*Benadryl—Trademark, Parke Davis and Co.
*Artane—Trademark, Lederle Laboratories
*Cogentin—Trademark, Merck Sharp & Dohme
*Akineton—Trademark, Knoll Pharmaceutical Co.

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Ideal for short-term antianxiety therapy



Acts promptly...tapers gently at discontinuation

Tranxene goes to work in minutes

Nordiazepam uptake is rapid, with pharmacologic effects reported within 30 minutes.[1] Peak serum levels are attained within 1-2 hours after dosing.

Allows post-therapy adjustment time

When administration stops, nordiazepam serum levels taper at a gentle rate. This built-in taper gives your patients more time to adjust to cessation of therapy.[2]

1. Sedation reported in patients 30 minutes after dosing. Tranxene Drug Monograph 97-0185, p. 9, 1979.
2. The elimination kinetics of an agent can be closely defined, but these cannot at present be directly related to therapeutic or adverse effects.

Abbott Laboratories, North Chicago, IL 60064

Brief Summary

INDICATIONS — Symptomatic relief of anxiety and tension associated with anxiety disorders, other psychoneurotic disorders, transient situational disturbances, and functional or organic disorders. Symptomatic relief of acute alcohol withdrawal.

Effectiveness in long-term use (over 4 months) not assessed by systematic clinical studies. Physician should periodically reassess usefulness for each patient.

CONTRAINDICATIONS — Known hypersensitivity to the drug. Acute narrow angle glaucoma.

WARNINGS — Not for use in depressive neuroses or psychotic reactions. Caution patients against hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles. Advise against simultaneous use of other CNS depressants, and caution patients that effects of alcohol may be increased. Not recommended for patients under 18. Nervousness, insomnia, irritability, diarrhea, muscle aches, and memory impairment have followed abrupt withdrawal from long-term high dosage. Withdrawal symptoms were reported after abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months. Use caution in patients having psychological potential for drug dependence (dependence has been observed in dogs and rabbits).

Pregnancy and Lactation: Minor tranquilizers should almost always be avoided first trimester. Consider possibility of pregnancy before initiating therapy. Patient should consult physician about discontinuation if she becomes pregnant or plans pregnancy. Do not give to nursing mothers.

PRECAUTIONS — Observe usual precautions in depression accompanying anxiety, or in patients with suicidal tendency, or those with impaired renal or hepatic function. Do periodic blood counts and liver function tests during prolonged therapy. Use small doses and gradual increments in the elderly or debilitated.

ADVERSE REACTIONS —Drowsiness, dizziness, various g.i. complaints, nervousness, blurred vision, dry mouth, headache, mental confusion, insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression, slurred speech, abnormal liver and kidney function, decreased hematocrit, decreased systolic blood pressure.

DOSAGE — ANXIETY Usual daily dose 30 mg or less (start the elderly or debilitated at 7.5-15 mg). Adjust gradually within 15-60 mg daily range. Capsules: divided doses; or once daily h.s. (start patient at 15 mg). Single Dose Tablets, 22.5 mg (for patients stabilized on 7.5 mg t.i.d.) or 11.25 mg: once daily at any hour. ALCOHOL WITHDRAWAL In divided doses: 1st day 30 mg initially, then 30-60 mg; 2nd day 45-90 mg; 3rd day 22.5-45 mg; 4th day 15-30 mg. Then taper to 15-7.5 mg daily, and discontinue as soon as stable.

INTERACTIONS —Potentiation may occur with ethyl alcohol, hypnotics, barbiturates, narcotics, phenothiazines, MAO inhibitors, other antidepressants.

OVERDOSAGE — Take general measures as for any CNS depressant.

SUPPLIED — Tranxene (clorazepate dipotassium) 3.75, 7.5, and 15 mg capsules. Tranxene-SD Half Strength 11.25 and Tranxene-SD 22.5 mg single dose tablets. 0043330

Acts promptly...tapers gently at discontinuation

CLORAZEPATE DIPOTASSIUM

Tranxene® C IV

4306CB



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3.75, 7.5, and 15 mg capsules; 11.25 and 22.5 mg single dose tablets.

See overleaf for brief summary.

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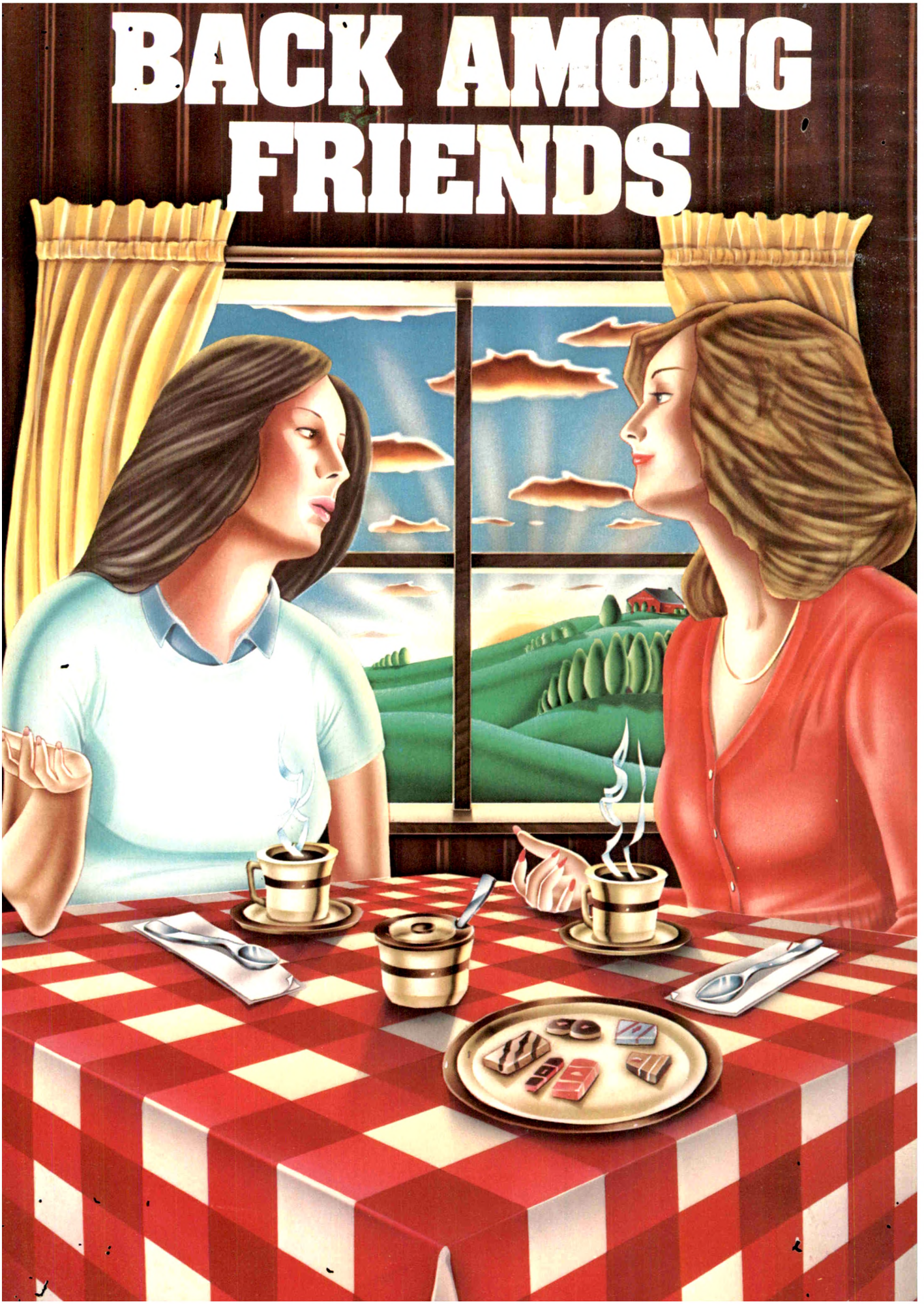
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By Leona L. Bachrach

The Use of ECT in the Treatment of Schizophrenia
by Carl Salzman



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>
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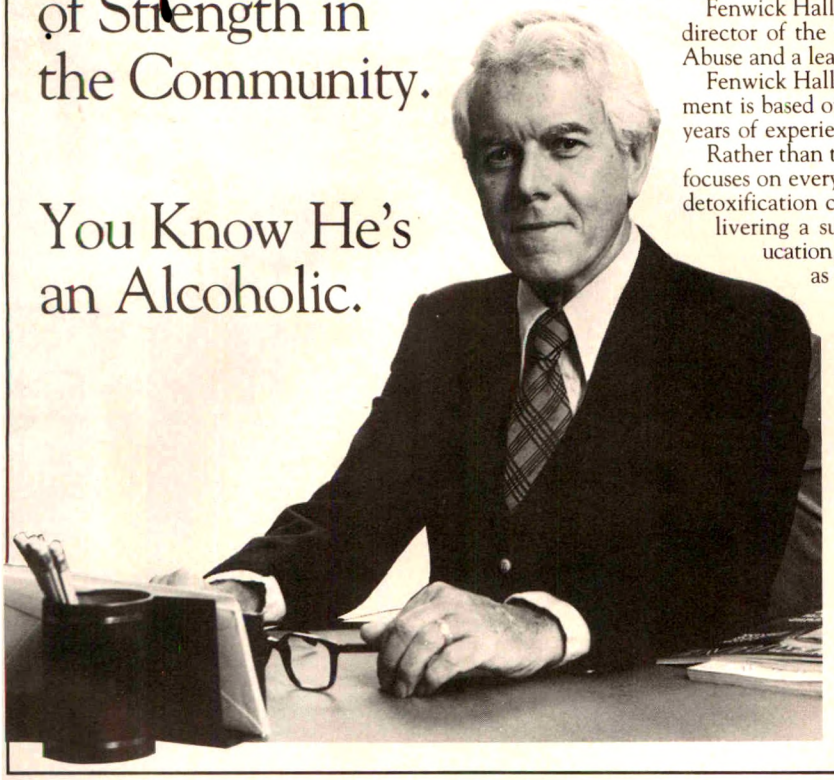
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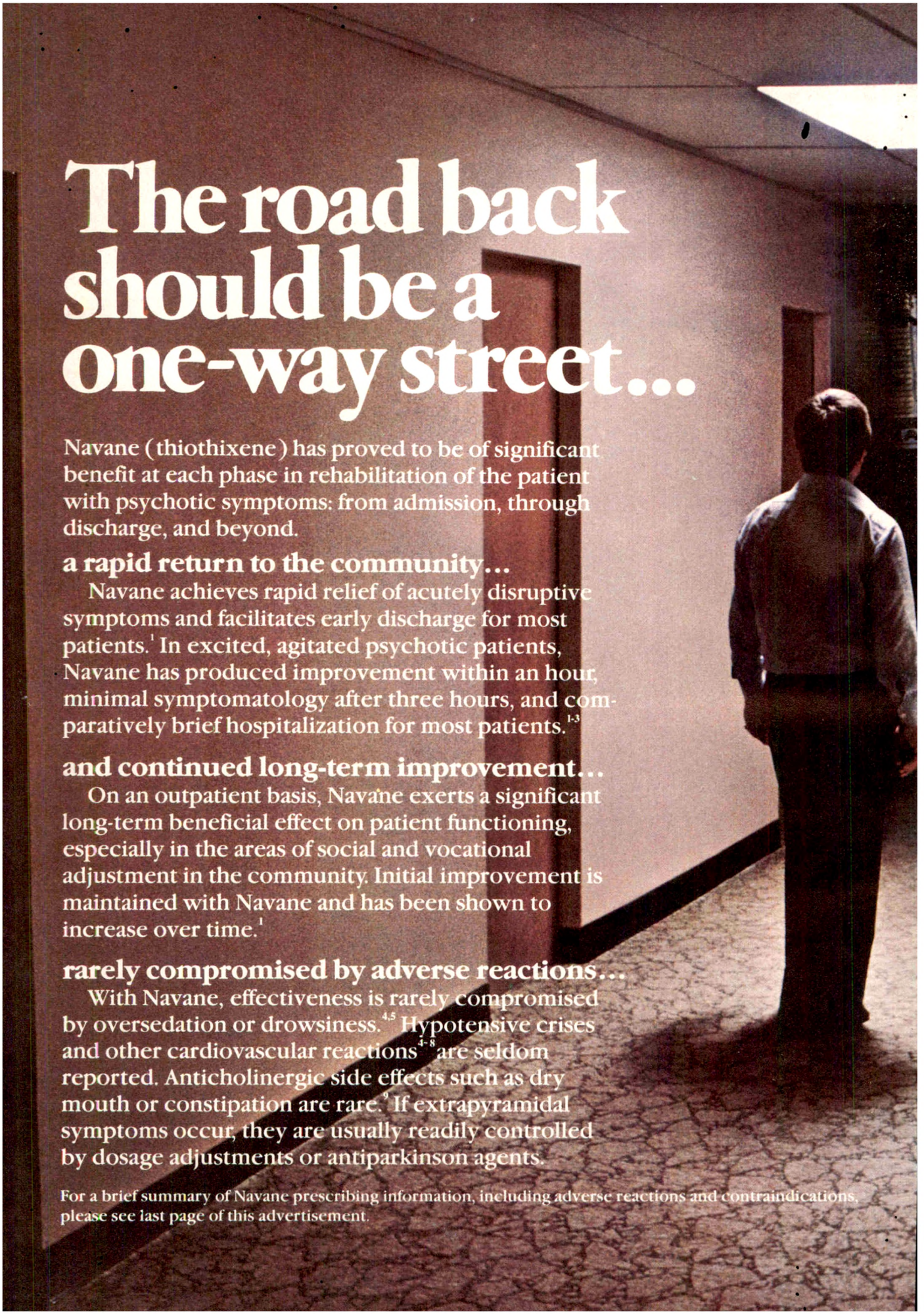
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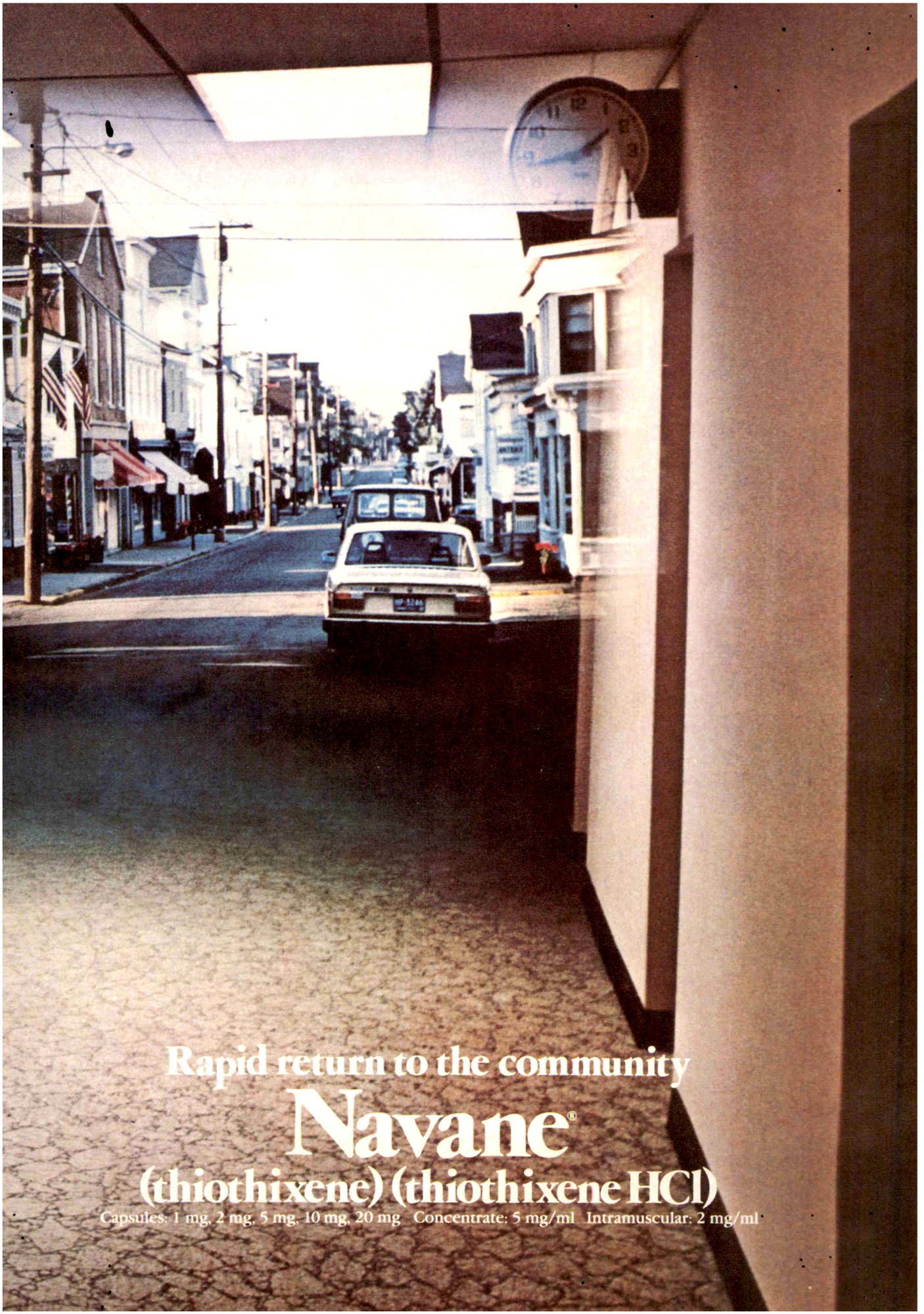
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On an outpatient basis, Navane exerts a significant long-term beneficial effect on patient functioning, especially in the areas of social and vocational adjustment in the community. Initial improvement is maintained with Navane and has been shown to increase over time.[1]

rarely compromised by adverse reactions...

With Navane, effectiveness is rarely compromised by oversedation or drowsiness.[4,5] Hypotensive crises and other cardiovascular reactions[4-8] are seldom reported. Anticholinergic side effects such as dry mouth or constipation are rare.[9] If extrapyramidal symptoms occur, they are usually readily controlled by dosage adjustments or antiparkinson agents.

For a brief summary of Navane prescribing information, including adverse reactions and contraindications, please see last page of this advertisement.


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Contraindications. Navane (thiothixene) is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy*–Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration of Navane to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children–The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration–As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane (thiothixene). Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudo-parkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leukocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution–For Intramuscular Use Only. Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane (thiothixene) Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate–In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Stimulants that may cause convulsions (e.g. picrotoxin or pentylenetetrazol) should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg of thiothixene in bottles of 100, 1,000, and unit-dose pack of 100 (10 x 10's). Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100, 500, and unit-dose pack of 100 (10 x 10's).

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg, and in 30 ml (1 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, and 5 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10 vials. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

References: 1. Rickels K, Norstad N, Downing R: The acutely decompensated schizophrenic patient, presented as a scientific exhibit at the American Psychiatric Association 28th Institute on Hospital and Community Psychiatry, September 20-22, 1976, Atlanta, Ga. 2. Stotsky BA: Relative efficacy of parenteral haloperidol and thiothixene for the emergency treatment of acutely excited and agitated patients. *Dis Nerv Syst* 38:967-973, Dec 1977. 3. Sloan RB, Razani J, Maloney MP, et al: Premorbid adjustment, cognition and outcome in schizophrenia, presented as a scientific exhibit at the 128th Annual Meeting of the American Psychiatric Association, May 5-9, 1975, Anaheim, California. 4. Engelhardt DM, Rudorfer L, Rosen B: Haloperidol and thiothixene in the long-term treatment of chronic schizophrenic outpatients in an urban community: Social and vocational adjustment. *J Clin Psychiatry* 39:834-840, Dec 1978. 5. Itil TM, Unverdi C, Wohlrabe J, et al: Drug therapy of psychosis associated with organic brain syndrome, presented as a scientific exhibit at the American Public Health Association Centennial, Atlantic City, New Jersey, Nov 12-16, 1972. 6. Brauzer B, Goldstein BJ: Comparative effects of intramuscular thiothixene and trifluoperazine in psychotic patients: *J Clin Pharmacol* 8:400-403, Nov-Dec 1968. 7. Dillenkoffer RL, Gallant DM, George RB, et al: Electrocardiographic evaluations of schizophrenic patients, presented as a scientific exhibit at the 125th Annual Meeting of the American Psychiatric Association, Dallas, Texas, May 1-4, 1972. 8. Data on file at Roerig. 9. Goldstein B, Weiner D, Banas F: Clinical evaluation of thiothixene in chronic ambulatory schizophrenic patients, in Lehmann HE, Ban TA (eds): *Modern Problems in Pharmacopsychiatry.* Basel, S Karger, 1969, vol 2, pp 45-52.

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NINGS: Children: This drug is not recommended for use in ren, since safety and effectiveness for the pediatric age group not been established. **Adults:** Use extreme caution in giving the to patients with evidence of cardiovascular disease. Caution is sed in patients with: increased intraocular pressure, history of ary retention, narrow-angle glaucoma, seizure disorder, hyper- idism, a need for thyroid medication. In patients receiving ethidine or similar agents, Surmontil may block the pharmaco- effects of these drugs. Warn patients that the drug may impair ental or physical abilities required for driving or performing potentially hazardous tasks.

CAUTIONS: Because of an inherently serious suicide potential, nonhospitalized severely depressed patient should be given the lest drug amount feasible. In schizophrenic patients, activation of sychosis may occur and require reduction of dosage or the addi- of a major tranquilizer to the medication schedule. Manic or manic episodes may occur, especially in patients with cyclic-type ders; Surmontil may have to be discontinued until the episode is ed and reinstituted, if required, at lower dosage. Limit concur- administration of Surmontil and electroconvulsive therapy to patients for whom it is essential. When possible, discontinue the for several days prior to elective surgery. The use of alcoholic drinks during therapy may provoke exaggerated response. Potentiation of effects has been reported when tricyclic antidepressants were administered with sympathomimetic amines, local decongestants, local anesthetics containing epinephrine, atropine, or drugs with an anticholinergic effect. Drugs having a parasympathetic effect, includ- ing tricyclic antidepressants, may alter ejaculatory response.

Usage in pregnancy: Pregnancy Category C. Surmontil has shown evi- dence of embryotoxicity and/or increased incidence of major anoma- lies in rats or rabbits at doses 20 times the human dose. There are no adequate and well-controlled studies in pregnant women. Surmontil should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

ADVERSE REACTIONS: When tricyclic antidepressants are used, each of the following adverse reactions must be considered, although some have not in fact been reported with Surmontil (trimipramine maleate).

Cardiovascular—Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric—Confusional states (especially in the elderly) with halluci- nations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurologic—Numbness, tingling, paresthesias of extremities; incoor- dination, ataxia, tremors; peripheral neuropathy; extrapyramidal symp- toms; seizures, alterations in EEG patterns; tinnitus.

Anticholinergic—Dry mouth and, rarely, associated sublingual adenitis; blurred vision, disturbances of accommodation, mydriasis, constipa- tion, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic—Skin rash, petechiae, urticaria, itching, photosensitization, edema of face and tongue.

Hematologic—Bone-marrow depression including agranulocytosis, eosinophilia; purpura; thrombocytopenia. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathologic neutrophil depression.

Gastrointestinal—Nausea and vomiting, anorexia, epigastric distress, diarrhea, peculiar taste, stomatitis, abdominal cramps, black tongue.

Endocrine—Gynecomastia in the male; breast enlargement and galac- torrhea in the female; increased or decreased libido, impotence; tes- ticular swelling; elevation or depression of blood sugar levels.

Other—Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency; drowsi- ness, dizziness, weakness, and fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms—Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache, and malaise.

SUPPLIED: 25 mg in bottles of 100 opaque blue and yellow capsules, 50 mg in bottles of 100 opaque blue and orange capsules.

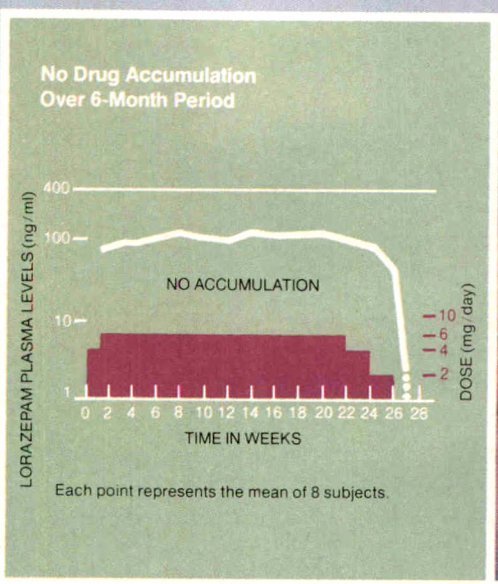
References: 1. Rickels K, et al: Amitriptyline and trimipramine in neurotic depressed outpatients: A collaborative study. *Am J Psychiatry* 127:208-217, 1970. **2.** Rifkin A, et al: Comparison of trimipramine and imipramine: A controlled study, to be published. **3.** Pecknold JC, Ananth J: Trimipramine in the treatment of anxious-depressed outpatients. *Curr Ther Res* 23:94-100, 1978. **4.** Lean TH, Sidhu MS: Comparative study of imipramine (Tofranil) and trimipra- mine (Surmontil) in depression associated with gynaecological condi- tions. *Proc Obstet Gynaecol Soc* 3:222-228, 1972.

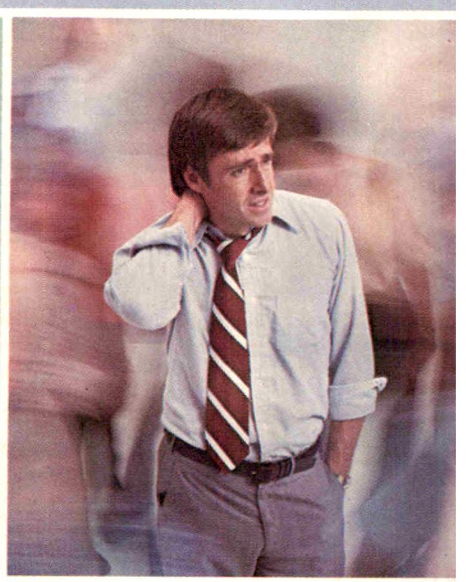
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How do you choose a minor tranquilizer?





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Pharmacokinetics

Ativan (lorazepam) is eliminated more rapidly than other benzodiazepines; generates no clinically active metabolites. The half-life of free lorazepam is about 12 hours; steady-state blood levels are attained in 2-3 days. (Comparable data for diazepam: 20-50 hours and at least 7-10 days.) Ativan shows no evidence of accumulation, even when given in high doses for as long as 6 months. (The pharmacokinetic profile of a drug can define such characteristics as absorption, distribution, metabolism and elimination but cannot, at present, be directly related to its therapeutic effectiveness.)

Anxiety neuroses

In many nationwide studies involving thousands of patients with anxiety neuroses, Ativan (lorazepam) consistently provided significant relief of anxiety, tension, agitation and irritability as measured by standard Hamilton, Global (physician rated) and 35-Item (patient self-rated) scales.

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They're all about the same, aren't they? Until recently, that seemed to sum up the prevalent attitude about the benzodiazepines. After all, they all work similarly and all are effective anxiolytics. With seven of these compounds now available, however, differences have begun to emerge. Foremost among them: different metabolic pathways, which distinguish the relatively short-acting Ativan (lorazepam) from long-acting diazepam, clorazepate and chlordiazepoxide. At the same time, your choice can be made with confidence in the clinical record of Ativan, which confirms its value in anxiety neuroses and in significant anxiety associated with functional or organic disorders, as well as in older patients.

Consider the record of Ativan:



Anxiety in cardiovascular disorders

Ativan (lorazepam) has been specifically evaluated and found effective in seven common protocol, double-blind studies involving 423 patients (211 on Ativan) whose anxiety was related to organic and functional cardiovascular disorders. The cardiovascular component has not, of course, been shown to be significantly benefited by such therapy.



Anxiety in gastrointestinal disorders

So far, nine common protocol, double-blind studies of Ativan have focused on anxious patients with functional or organic gastrointestinal complaints (457 patients, 234 on Ativan). Ativan was clearly effective in reducing the anxiety of these patients. The gastrointestinal component has not, of course, been shown to be significantly benefited by such therapy.



Anxiety in the aging patient

Because its simple metabolism is not readily impaired with advancing age, and accumulation is not likely to present a problem, Ativan is a good choice for older patients. Those who have trouble swallowing solid medication appreciate the small Ativan tablet, which is tasteless and disintegrates within seconds in water or fruit juice.

Ativan® (lorazepam) for Anxiety

See important information on following page.

Indications and Usage: Symptomatic relief of anxiety, tension, agitation, irritability and insomnia associated with anxiety neuroses and transient situational disturbances; anxiety associated with depressive symptoms and as a treatment of symptoms of anxiety if such symptoms are a significant feature of functional or organic disorders, particularly gastrointestinal or cardiovascular.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients on lorazepam not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation.

Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions.

Observe usual precautions with impaired renal or hepatic function.

Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component.

Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 5mg/kg/day. No effect dose was 1.25mg/kg/day (approximately 6 times the maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatric patients requires caution and frequent monitoring for symptoms of upper G.I. disease.

Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug.

In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Overdosage: In management of overdosage with any drug, bear in mind that multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring of vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

Ativan® IV for (lorazepam) Anxiety

Dosage: **Individualize for maximum beneficial effects. Increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; insomnia due to anxiety or transient situational stress, 2-4mg h.s.**

How Supplied: **0.5, 1.0 and 2.0mg tablets.**

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The author's thorough coverage of offender/victim psychology will provide much needed information for psychiatrists working with criminals, delinquents, and their victims. For those interested in doing further research, references are presented for each chapter. *'80, 292 pp., $19.75*

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Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Effective: For the management of the manifestations of psychotic disorders.

Possibly effective: To control excessive anxiety, tension and agitation as seen in neuroses or associated with somatic conditions.

'Stelazine' has not been shown effective in the management of behavioral complications in patients with mental retardation.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose or greatly depressed states due to C.N.S. depressants; blood dyscrasias; bone marrow depression; liver damage.

Warnings: Generally avoid using in patients hypersensitive (e.g., blood dyscrasias, jaundice) to any phenothiazine. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery), especially during the first few days' therapy.

Additive effect is possible with other C.N.S. depressants, including alcohol.

Use in pregnancy only when necessary for patient's welfare.

Precautions: Use cautiously in angina. Avoid high doses and parenteral administration when cardiovascular system is impaired. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Prolonged administration of high doses may result in cumulative effects with severe C.N.S. or vasomotor symptoms. If retinal changes occur, discontinue drug. Agranulocytosis, thrombocytopenia, pancytopenia, anemia, cholestatic jaundice, liver damage have been reported.

Patients on long-term therapy, especially high doses, should be evaluated periodically for possible adjustment or discontinuance of drug therapy.

Adverse Reactions: Drowsiness, dizziness, skin reactions, rash, dry mouth, insomnia, amenorrhea, fatigue, muscular weakness, anorexia, lactation, blurred vision. Neuromuscular (extrapyramidal) reactions: motor restlessness, dystonias, pseudoparkinsonism, persistent tardive dyskinesia.

Other adverse reactions reported with Stelazine (trifluoperazine HCl, SK&F) or other phenothiazines: Some adverse effects are more frequent or intense in specific disorders (e.g., mitral insufficiency or pheochromocytoma).

Grand mal convulsions; altered cerebrospinal fluid proteins; cerebral edema; prolongation and intensification of the action of C.N.S. depressants, atropine, heat, and organophosphorus insecticides; nasal congestion, headache, nausea, constipation, obstipation, adynamic ileus, inhibition of ejaculation; reactivation of psychotic processes, catatonic-like states; hypotension (sometimes fatal); cardiac arrest; leukopenia, eosinophilia, pancytopenia, agranulocytosis, thrombocytopenic purpura; jaundice, biliary stasis; menstrual irregularities, galactorrhea, gynecomastia, false positive pregnancy tests; photosensitivity, itching, erythema, urticaria, eczema up to exfoliative dermatitis; asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions, peripheral edema; reversed epinephrine effect; hyperpyrexia; a systemic lupus erythematosus-like syndrome; pigmentary retinopathy; with prolonged administration of substantial doses, skin pigmentation, epithelial keratopathy, and lenticular and corneal deposits. EKG changes have been reported, but relationship to myocardial damage is not confirmed. Discontinue long-term, high-dose therapy gradually. NOTE: Sudden death in patients taking phenothiazines (apparently due to cardiac arrest or asphyxia due to failure of cough reflex) has been reported, but no causal relationship has been established.

Supplied: Tablets, 1 mg., 2 mg., 5 mg. and 10 mg., in bottles of 100; in Single Unit Packages of 100 (intended for institutional use only); Injection, 2 mg./ml.; and Concentrate (intended for institutional use only) 10 mg./ml.

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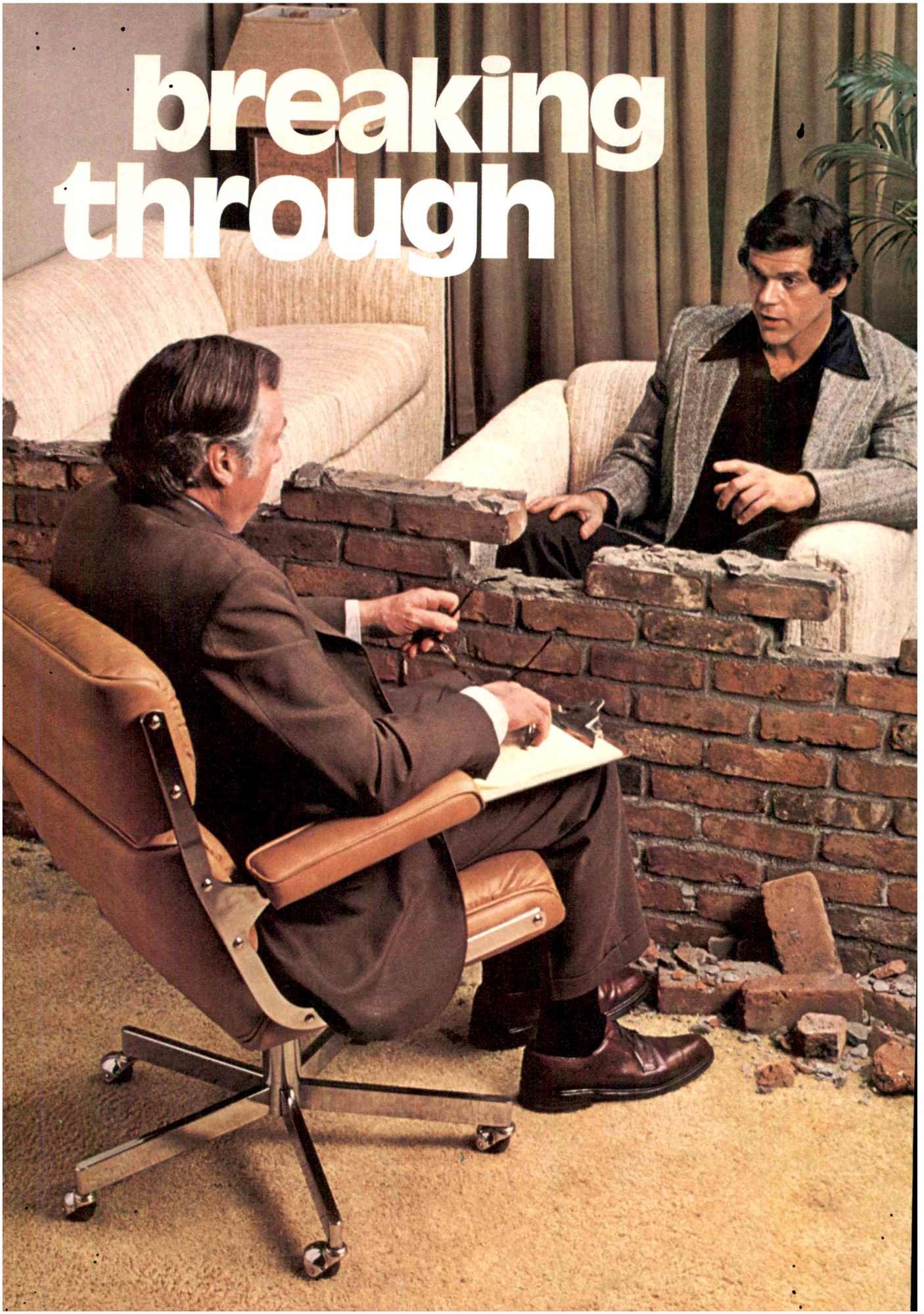
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The tranquilizer component alleviates symptoms of agitation and anxiety within a few days, without apparent dulling of mental acuity. Hypnotic effects from the tranquilizer component appear to be minimal, particularly in patients permitted to remain active. However, TRIAVIL may impair mental and/or physical abilities required for performance of hazardous tasks.

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As symptoms are relieved, many patients often communicate more effectively, become more cooperative, and are able to return to normal daily activities.

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There are now *five* tablet strengths of TRIAVIL for ease of dosage adjustment. For many patients with agitation and depression, you can now initiate therapy with one TRIAVIL® 4-50, containing 4 mg perphenazine and 50 mg amitriptyline HCl, b.i.d. The regimen is simple, economical, and may well enhance patient compliance.

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TRIAVIL is contraindicated in CNS depression from drugs, in the presence of evidence of bone marrow depression, and in patients hypersensitive to phenothiazines or amitriptyline. It should not be used during the acute recovery phase following myocardial infarction or in patients who have received an MAOI within two weeks. Patients with cardiovascular disorders should be watched closely. Not recommended in children or during pregnancy. TRIAVIL may impair mental and/or physical abilities required for performance of hazardous tasks and may enhance the response to alcohol. Antiemetic effect may obscure toxicity due to overdosage of other drugs or mask other disorders. The possibility of suicide in depressed patients remains until significant remission occurs. Such patients should not have access to large quantities of the drug. Hospitalize as soon as possible any patient suspected of having taken an overdose.



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TRIAVIL® 4-10: Each tablet contains
4 mg perphenazine and 10 mg amitriptyline HCl.

CONTRAINDICATIONS: Central nervous system depression from drugs (barbiturates, alcohol, narcotics, analgesics, antihistamines); evidence of bone marrow depression; known hypersensitivity to phenothiazines or amitriptyline. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred from such combinations. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with TRIAVIL. Therapy should then be initiated cautiously with gradual increase in dosage until optimum response is achieved. Not recommended for use during acute recovery phase following myocardial infarction.

WARNINGS: TRIAVIL should not be given concomitantly with guanethidine or similarly acting compounds since TRIAVIL may block the antihypertensive action of such compounds. Use cautiously in patients with history of urinary retention, angle-closure glaucoma, increased intraocular pressure, or convulsive disorders. Dosage of anticonvulsive agents may have to be increased. In patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely. Tricyclic antidepressants, including amitriptyline HCl, have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time, particularly in high doses. Myocardial infarction and stroke have been reported with tricyclic antidepressant drugs. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Not recommended in children or during pregnancy.

PRECAUTIONS: Suicide is a possibility in depressed patients and may remain until significant remission occurs. Such patients should not have access to large quantities of this drug.

Perphenazine: Should not be used indiscriminately. Use with caution in patients who have previously exhibited severe adverse reactions to other phenothiazines. Likelihood of some untoward actions is greater with high doses. Closely supervise with any dosage. The antiemetic effect of perphenazine may obscure signs of toxicity due to overdosage of other drugs or make more difficult the diagnosis of disorders such as brain tumor or intestinal obstruction. A significant, not otherwise explained, rise in body temperature may suggest individual intolerance to perphenazine, in which case discontinue.

If hypotension develops, epinephrine should not be employed, as its action is blocked and partially reversed by perphenazine. Phenothiazines may potentiate the action of central nervous system depressants (opiates, analgesics, antihistamines, barbiturates, alcohol) and atropine. In concurrent therapy with any of these, TRIAVIL should be given in reduced dosage. May also potentiate the action of heat and phosphorous insecticides. There is sufficient experimental evidence to conclude that chronic administration of antipsychotic drugs which increase prolactin secretion has the potential to induce mammary neoplasms in rodents under the appropriate conditions. There are recognized differences in the physiological role of prolactin between rodents and humans. Since there are, at present, no adequate epidemiological studies, the relevance to human mammary cancer risk from prolonged exposure to perphenazine and other antipsychotic drugs is not known.

Amitriptyline: In manic-depressive psychosis, depressed patients may experience a shift toward the manic phase if they are treated with an antidepressant. Patients with paranoid symptomatology may have an exaggeration of such symptoms. The tranquilizing effect of TRIAVIL seems to reduce the likelihood of this effect. When amitriptyline HCl is given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. Paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs.

Caution is advised if patients receive large doses of ethchlorvynol concurrently. Transient delirium has been reported in patients who were treated with 1 g of ethchlorvynol and 75-150 mg of amitriptyline HCl.

Amitriptyline HCl may enhance the response to alcohol and the effects of barbiturates and other CNS depressants.

Concurrent administration of amitriptyline HCl and electroshock therapy may increase the hazards associated with such therapy. Such treatment should be limited to patients for whom it is essential. Discontinue several days before elective surgery if possible. Elevation and lowering of blood sugar levels have both been reported. Use with caution in patients with impaired liver function.

ADVERSE REACTIONS: Similar to those reported with either constituent alone.
Perphenazine: Extrapyramidal symptoms (opisthotonus, oculogyric crisis, hyperreflexia, dystonia, akathisia, acute dyskinesia, ataxia, parkinsonism) have been reported and can usually be controlled by the concomitant use of effective antiparkinsonian drugs and/or by reduction in dosage, but sometimes persist after discontinuation of the phenothiazine.

Tardive dyskinesia may appear in some patients on long-term therapy or may occur after drug therapy with phenothiazines and related agents has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw. Involuntary movements of the extremities sometimes occur. There is no known treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms. It is advised that all antipsychotic agents be discontinued if the above symptoms appear. If treatment is reinstituted, or dosage of the particular drug increased, or another drug substituted, the syndrome may be masked. Fine vermicular movements of the tongue may be an early sign of the syndrome. The full-blown syndrome may not develop if medication is stopped when lingual vermiculation appears.

Other side effects are skin disorders (photosensitivity, itching, erythema, urticaria, eczema, up to exfoliative dermatitis); other allergic reactions (asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions); peripheral edema; reversed epinephrine effect; hyperglycemia; endocrine disturbances (lactation, galactorrhea, gynecomastia, disturbances of menstrual cycle); altered cerebrospinal fluid proteins; paradoxical excitement; hypertension, hypotension, tachycardia, and ECG abnormalities (quinidine-like effect); reactivation of psychotic processes; catatonic-like states; autonomic reactions, such as dry mouth or salivation, headache, anorexia, nausea, vomiting, constipation, obstipation, urinary frequency or incontinence, blurred vision, nasal congestion, and a change in pulse rate; other adverse reactions reported with various phenothiazine compounds, but not with perphenazine, include grand mal convulsions, cerebral edema, polyphagia, pigmentary retinopathy, photophobia, skin pigmentation, and failure of ejaculation.

The phenothiazine compounds have produced blood dyscrasias (pancytopenia, thrombocytopenic purpura, leukopenia, agranulocytosis, eosinophilia); and liver damage (jaundice, biliary stasis).

Pigmentation of the cornea and lens has been reported to occur after long-term administration of some phenothiazines. Although it has not been reported in patients receiving TRIAVIL, the possibility that it might occur should be considered.

Hypnotic effects, lassitude, muscle weakness, and mild insomnia have also been reported.

Amitriptyline: Note: Listing includes a few reactions not reported for this drug, but which have occurred with other pharmacologically similar tricyclic antidepressant drugs and must be considered when amitriptyline is administered. *Cardiovascular:* Hypotension; hypertension; tachycardia; palpitation; myocardial infarction; arrhythmias; heart block; stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth; blurred vision; disturbance of accommodation; increased intraocular pressure; constipation; paralytic ileus; urinary retention; dilatation of urinary tract. *Allergic:* Skin rash; urticaria; photosensitization; edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis; leukopenia; eosinophilia; purpura; thrombocytopenia. *Gastrointestinal:* Nausea; epigastric distress; vomiting; anorexia; stomatitis; peculiar taste; diarrhea; parotid swelling; black tongue. Rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido; elevated or lowered blood sugar levels. *Other:* Dizziness, weakness; fatigue; headache; weight gain or loss; increased perspiration; urinary frequency; mydriasis; drowsiness; alopecia. *Withdrawal Symptoms:* Abrupt cessation after prolonged administration may produce nausea, headache, and malaise. These are not indicative of addiction.

OVERDOSAGE: All patients suspected of having taken an overdosage should be admitted to a hospital as soon as possible. Treatment is symptomatic and supportive. However, the intravenous administration of 1–3 mg of physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage of physostigmine should be repeated as required particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine. On this basis, in severe overdosage with perphenazine-amitriptyline combinations, symptomatic treatment of central anticholinergic effects with physostigmine salicylate should be considered.

J8TR32 (DC6613215)

For more detailed information, consult your MSD Representative or see full Prescribing Information. Merck Sharp & Dohme, Division of Merck & Co., INC., West Point, Pa. 19486.

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A residential treatment center for emotionally disturbed adolescent boys

- I.Q. NORMAL & GIFTED
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- GRADES: 6—12
- 12 MONTH PROGRAM STATE ACCREDITED
- DIPLOMA AWARDED COLLEGE ADMITTED
- STUDENTS: 80
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Residential treatment has become highly specialized in the field of mental health. It is a specific treatment modality for those who need a totally planned and structured environment.

The Brown Schools has developed a wide range of professional services that can be utilized to implement an individually planned residential treatment program. The degree of structure and protection, the intensity of therapy, the methods of education and training are controlled and modified with the resident's changing needs.

Professionals in the areas of psychiatry, psychology, nursing, social work, education, prevocational training, speech pathology, and recreation have developed expertise in the specific area of residential treatment. Each service area is designed as a component of an integrated therapeutic milieu.

The three residential treatment centers of The Brown Schools provide complete programming for those in need of twenty-four hour care. Services are available for children, adolescents, and adults with emotional disturbance, mental retardation, and neurological impairment. Two small group homes in Austin provide for reintegration into the community and complement the services offered in the other centers. For information, write:

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DALMANE
(flurazepam HCl/Roche

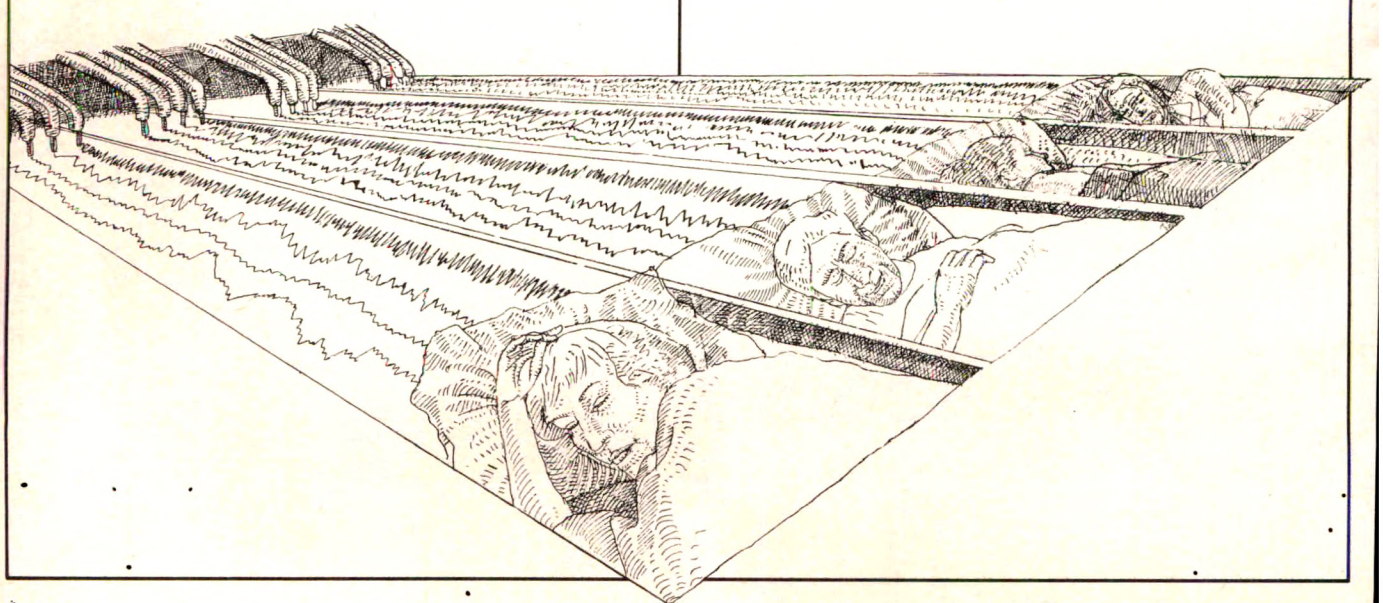
MORE POLYGRAPHIC EVIDENCE

Sleep research laboratory testing of insomnia patients for 995 subject nights demonstrates efficacy of Dalmane (flurazepam HCl/Roche)[1]

In 14 separate studies, subjects given Dalmane were monitored with polygraphic recordings for a total of 995 nights, objectively demonstrating the efficacy of Dalmane.

Efficacy from the first night through 28 nights, without need to increase dosage

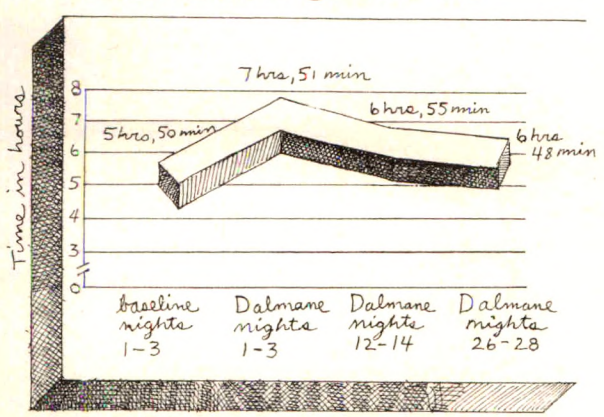
Dalmane (flurazepam HCl/Roche) is the only sleep medication objectively proven effective through 28 consecutive nights of administration in two separate studies in the sleep research laboratory.[2-4] The prolonged use of Dalmane is seldom necessary; should it be, appropriate patient evaluations such as periodic blood counts and liver and kidney function tests are recommended.



OFFERS

OF EFFICACY FOR INSOMNIA

Dalmane® (flurazepam HCl/Roche) improved sleep during short, intermediate and longer-term use



Improvement shown represents mean improvement, in hours, of total sleep time. n=5

Adapted from Dement WC, *et al: Behav Med* 5:25-31, Oct 1978

Since most insomnia is transitory in nature, initiate therapy at the lowest effective dose for the shortest time advisable to achieve a predetermined therapeutic goal.

References:
1. Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley NJ
2. Kales A, *et al: Clin Pharmacol Ther* 19: 576-583, May 1976
3. Dement WC, *et al: Behav Med* 5: 25-31, Oct 1978
4. Kales A, *et al: Clin Pharmacol Ther* 18: 356-363, Sep 1975

Clinical documentation of efficacy in 4950 patients with insomnia[1]

Dalmane (flurazepam HCl/Roche) efficacy was evaluated in 18 clinical studies. These included paired-night, double-blind crossover, double-blind comparison and patient preference studies. Results of these studies demonstrated and confirmed the effectiveness of Dalmane.

Periodic evaluation of patient's sleep habits may help prevent subsequent insomnia

Should insomnia recur, the patient may need guidance in setting up a regular sleep program. This may include establishment of a regular bedtime hour, taking a warm bath prior to retiring and avoidance of alcohol, stimulants or heavy meals late in the evening.

DALMANE® (C IV)

flurazepam HCl/Roche
15-mg and 30-mg capsules

UNMATCHED PROOF, UNMATCHED EFFICACY

Please see summary of product information on following page.

ROCHE®

Protocol for insomnia management

☐ Treat physical and/or psychological factors that may underlie an inability to sleep.

☐ Thoroughly question and evaluate new patients who specifically request sleep medications.

☐ Consider other medications the patient may be taking (including alcoholic beverages) and be aware of possible drug interactions.

☐ Select a sleep medication with objectively *documented* proof of efficacy and a wide margin of safety.

☐ Initiate treatment at the lowest effective dose for the period of time deemed necessary *to achieve a reasonable therapeutic goal.*

☐ Monitor response to therapy and, based upon patient reassessment, determine need for continuing medication on an individual basis.

☐ Advise patients who ingest alcoholic beverages or other CNS depressants against doing so while on sleep medication. CNS stimulants such as caffeine should also be discontinued.

☐ Caution patients against engaging in hazardous occupations requiring complete mental alertness such as operating machinery or driving a motor vehicle shortly after ingesting the drug. In addition, as with all CNS depressants, patients may have reduced motor performance while on hypnotic therapy and should be advised accordingly.

☐ Avoid prescribing for suicidal or dependency-prone individuals whose histories indicate an inability to handle any psychoactive substances including alcohol.

Now available from Roche—important guidance for your patients with insomnia.

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DA-5T

DALMANE® (IV)
flurazepam HCl/Roche

One 15-mg capsule *h.s.*—recommended initial dosage for elderly or debilitated patients.
One 30-mg capsule *h.s.*—usual adult dosage
(15 mg may suffice in some patients).

Before prescribing, please consult complete product information, a summary of which follows:
Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Objective sleep laboratory data have shown effectiveness for at least 28 consecutive nights of administration. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended.
Contraindications: Known hypersensitivity to flurazepam HCl.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

Usage in Pregnancy: **Several studies of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) suggest increased risk of congenital malformations during the first trimester of pregnancy. Dalmane, a benzodiazepine, has not been studied adequately to determine whether it may be associated with such an increased risk. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.
Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies, or with impaired renal or hepatic function. Periodic blood counts and liver and kidney function tests are advised during repeated therapy.
Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase.
Dosage: Individualize for maximum beneficial effect.
Adults: 30 mg usual dosage; 15 mg may suffice in some patients.
Elderly or debilitated patients: 15 mg recommended initially until response is determined.
Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

ROCHE PRODUCTS INC.
Manati, Puerto Rico 00701

ROCHE®



Introducing...

Navane®
(thiothixene HCl)
Intramuscular
For Injection
5 mg/ml



new potency for rapid control of acute psychiatric emergencies

Now, Navane Intramuscular 5 mg per ml provides a more concentrate dosage form for rapid control of severely disturbed behavior associated with acute psychosis. Navane Intramuscular, in carefully monitored doses, "...can produce significant improvement within hours and increase the accessibility of these [acutely disturbed] patients to more gradual methods of pharmacologic treatment as well as to socio-environmental intervention and psychotherapy."[1]

Rapid tranquilization usually can be achieved without excessive sedation.[1] Clinically significant hypotension, often seen with other injectable antipsychotic agents, is rare with Navane Intramuscular therapy.[1-3] If extrapyramidal symptoms occur, they are usually readily controlled by dosage adjustment or antiparkinson agents.

ROERIG Pfizer
A division of Pfizer Pharmaceuticals
New York, New York 10017

For a brief summary of Navane prescribing information, including adverse reactions and contraindications, please see the following page of this advertisement.

Navane® (thiothixene) (thiothixene hydrochloride)

Capsules: 1 mg, 2 mg, 5 mg, 10 mg, 20 mg Concentrate: 5 mg/ml Intramuscular: 2 mg/ml, 5 mg/ml

BRIEF SUMMARY OF PRESCRIBING INFORMATION
Navane® (thiothixene)
Capsules: 1 mg, 2 mg, 5 mg, 10 mg, 20 mg (thiothixene hydrochloride) Concentrate: 5 mg/ml, Intramuscular: 2 mg/ml, 5 mg/ml

Contraindications. Navane (thiothixene) is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy*– Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration of Navane to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children– The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration– As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., glutеus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane (thiothixene). Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudo-parkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leukocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution; Navane For Injection– Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane (thiothixene) Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate– In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Stimulants that may cause convulsions (e.g. picrotoxin or pentylenetetrazol) should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg of thiothixene in bottles of 100, 1,000, and unit-dose pack of 100 (10 x 10's). Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100, 500, and unit-dose pack of 100 (10 x 10's).

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg, and in 30 ml (1 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, and 5 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10 vials. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

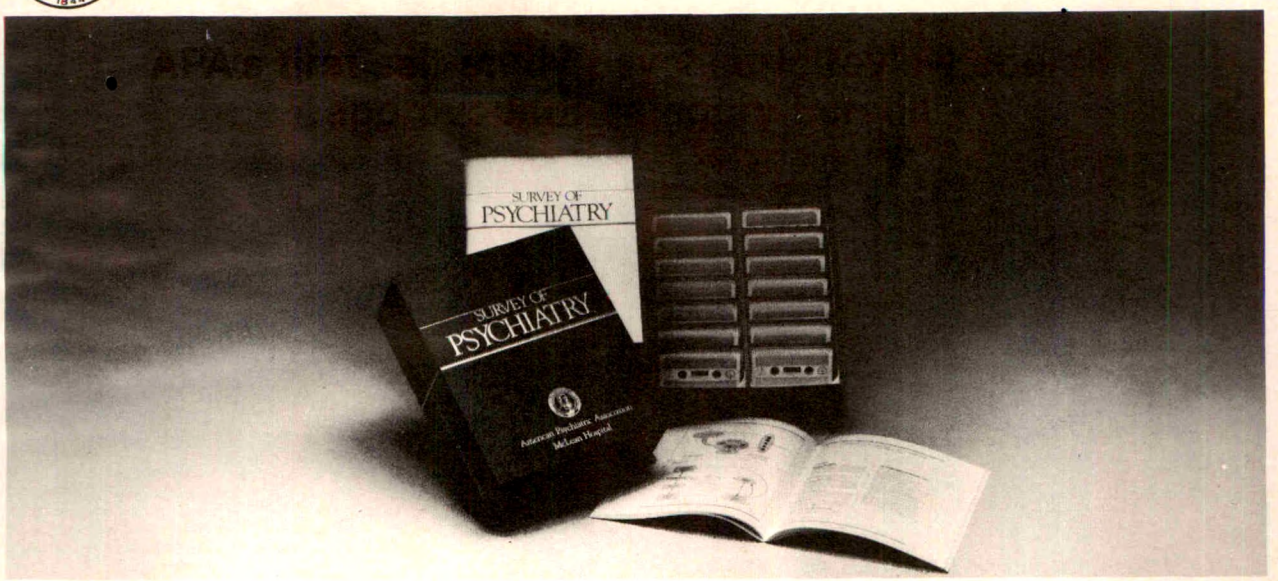
Navane (thiothixene hydrochloride) Intramuscular For Injection is available in amber glass vials in packages of 10 vials. When reconstituted with 2.2 ml of Sterile Water for Injection, each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene, and 59.6 mg of mannitol. The reconstituted solution of Navane Intramuscular For Injection may be stored for 48 hours at room temperature before discarding.

References: 1. Stotsky BA: Relative efficacy of parenteral haloperidol and thiothixene for the emergency treatment of acutely excited and agitated patients. *Dis Nerv Syst* 38:967-973, December 1977. 2. Brauzer B, Goldstein BJ: Comparative effects of intramuscular thiothixene and trifluoperazine in psychotic patients. *J Clin Pharmacol* 8:400-403, November-December 1968. 3. Gallant DM, Bishop MP, Bishop C, et al: Thiothixene: A controlled evaluation of the intramuscular antipsychotic preparation. *Curr Ther Res* 10:561-565, November 1968.

For additional information on Navane, please consult your Roerig representative or write to: Roerig Medical Department, 235 East 42nd Street, New York, NY 10017.

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Brief Summary of Prescribing Information

Indications and Usage: Symptomatic relief of anxiety, tension, agitation, irritability and insomnia associated with anxiety neuroses and transient situational disturbances; anxiety associated with depressive symptoms and as a treatment of symptoms of anxiety if such symptoms are a significant feature of functional or organic disorders, particularly gastrointestinal or cardiovascular.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients on lorazepam not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation.

Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions.

Observe usual precautions with impaired renal or hepatic function.

Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component.

Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day. No effect dose was 1.25mg/kg/day (approximately 6 times the maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatric patients requires caution and frequent monitoring for symptoms of upper G.I. disease.

Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug.

In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Overdosage: In management of overdosage with any drug, bear in mind that multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring of vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

Ativan® IV for Anxiety (lorazepam)

Dosage: **Individualize for maximum beneficial effects. Increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; insomnia due to anxiety or transient situational stress, 2-4mg h.s.**

How Supplied: **0.5, 1.0 and 2.0mg tablets.**

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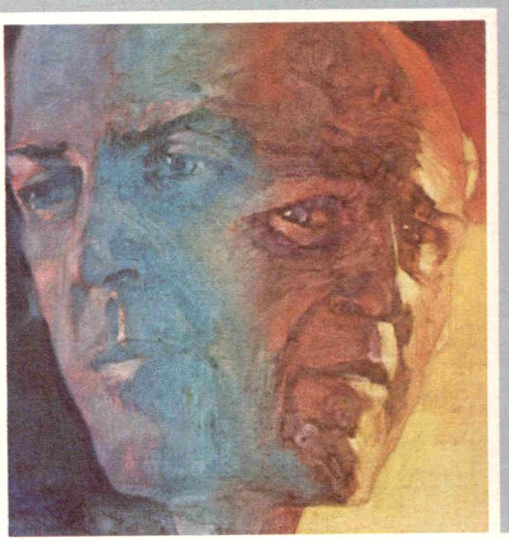
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A mixed syndrome. When drug intervention is indicated in patients whose anxiety is accompanied by depressive symptoms, the choice, essentially, is among three basic strategies: (1) an antidepressant; (2) a tranquilizer; (3) a combination of (1) and (2).

With respect to strategy (2), you should know that Ativan (lorazepam) has been shown to be statistically and clinically significantly effective in alleviating the anxiety component of this mixed syndrome, in common protocol controlled studies of 653 patients (337 on Ativan).

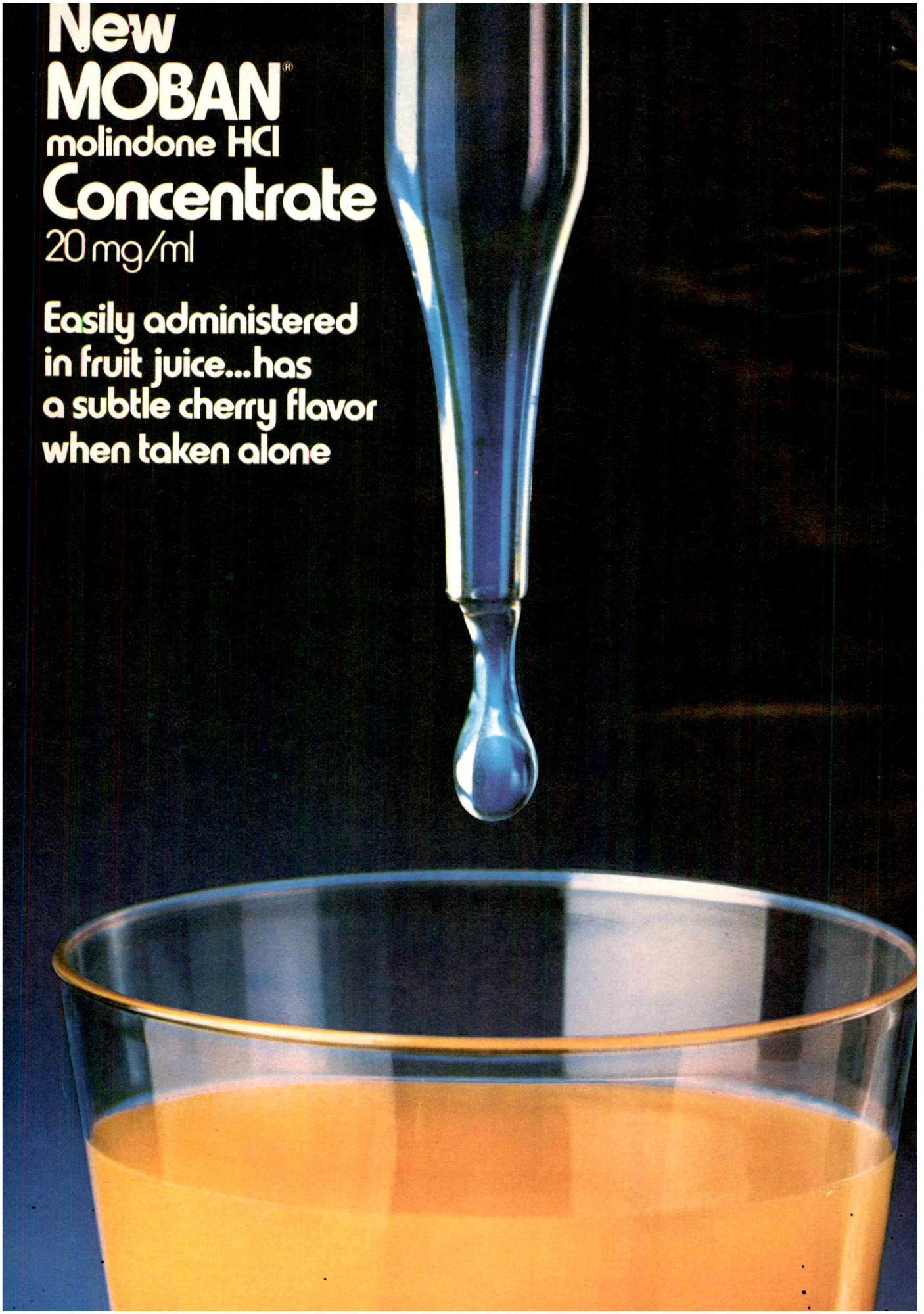
Since Ativan was also found to be compatible with tricyclics, if you opt for strategy (3), your patient has the benefit of rapid anxiolytic action, absence of long-acting metabolites and rapid clearance when you decide to discontinue Ativan. All benzodiazepines, however, including Ativan, produce added CNS depressant effects when administered with alcohol and other CNS depressants.

In either case, Ativan offers you the convenience of a b.i.d. schedule and a flexibility not possible with any fixed combination.



See important information on preceding page.

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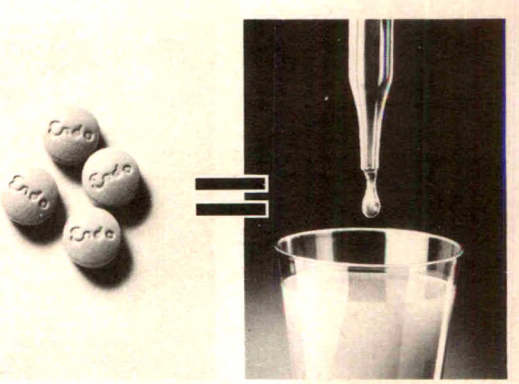
An effective antipsychotic agent with a favorable benefit/risk profile

NO incidence	excessive weight gain hepatotoxic changes ophthalmological changes persistent tardive dyskinesia skin pigmentation thyroid function changes tolerance development
LOW incidence	adverse drug interactions hematological changes hypotension nonpersistent symptoms of tardive dyskinesia sedation
MODERATE incidence	extrapyramidal reactions

This table compiled from clinical and field reports, according to the best available information.

For the schizophrenic patient

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- may be adjusted up or down according to response
- some patients with severe symptomatology may require an increase to 225 mg/day
- elderly and debilitated patients should be started on lower dosage

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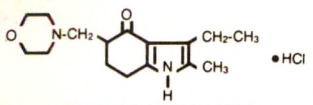
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DESCRIPTION MOBAN (molindone hydrochloride) is a dihydroindolone compound which is not structurally related to the phenothiazines, the butyrophenones or the thioxanthenes.

MOBAN is 3-ethyl-6, 7-dihydro-2-methyl-5-(morpholinomethyl) indol-4 (5H)-one hydrochloride. It is a white crystalline powder, freely soluble in water and alcohol and has a molecular weight of 312.67.



MOLINDONE HYDROCHLORIDE

ACTIONS MOBAN (molindone hydrochloride) has a pharmacological profile in laboratory animals which predominantly resembles that of major tranquilizers causing reduction of spontaneous locomotion and aggressiveness, suppression of a conditioned response and antagonism of the bizarre stereotyped behavior and hyperactivity induced by amphetamines. In addition, MOBAN antagonizes the depression caused by the tranquilizing agent tetrabenazine.

In human clinical studies tranquilization is achieved in the absence of muscle relaxing or incoordinating effects. Based on EEG studies, MOBAN exerts its effect on the ascending reticular activating system.

Human metabolic studies show MOBAN (molindone hydrochloride) to be rapidly absorbed and metabolized when given orally. Unmetabolized drug reached a peak blood level at 1.5 hours. Pharmacological effect from a single oral dose persists for 24-36 hours. There are 36 recognized metabolites with less than 2-3% unmetabolized MOBAN being excreted in urine and feces.

INDICATIONS MOBAN (molindone hydrochloride) is indicated in the management of the manifestations of schizophrenia.

CONTRAINDICATIONS MOBAN (molindone hydrochloride) is contraindicated in severe central nervous system depression (alcohol, barbiturates, narcotics, etc.) or comatose states, and in patients with known hypersensitivity to the drug.

WARNINGS Usage in Pregnancy: Studies in pregnant patients have not been carried out. Reproduction studies have been performed in the following animals:

Pregnant Rats oral dose— 20 mg/kg/day—10 days
no adverse effect
40 mg/kg/day—10 days
no adverse effect

Pregnant Mice oral dose— 20 mg/kg/day—10 days
slight increase resorptions
40 mg/kg/day—10 days
slight increase resorptions

Pregnant Rabbits oral dose— 5 mg/kg/day—12 days
no adverse effect
10 mg/kg/day—12 days
no adverse effect
20 mg/kg/day—12 days
no adverse effect

Animal reproductive studies have not demonstrated a teratogenic potential. The anticipated benefits must be weighed against the unknown risks to the fetus if used in pregnant patients.

Nursing Mothers: Data are not available on the content of MOBAN (molindone hydrochloride) in the milk of nursing mothers.

Usage in Children: Use of MOBAN (molindone hydrochloride) in children below the age of twelve years is not recommended because safe and effective conditions for its usage have not been established.

MOBAN has not been shown effective in the management of behavioral complications in patients with mental retardation.

PRECAUTIONS Some patients receiving MOBAN (molindone hydrochloride) may note drowsiness initially and they should be advised against activities requiring mental alertness until their response to the drug has been established.

Increased activity has been noted in patients receiving MOBAN. Caution should be exercised where increased activity may be harmful.

MOBAN does not lower the seizure threshold in experimental animals to the degree noted with more sedating antipsychotic drugs. However, in humans convulsive seizures have been reported in a few instances.

EDO 854M580

The physician should be aware that this tablet preparation contains calcium sulfate as an excipient and that calcium ions may interfere with the absorption of preparations containing phenytoin sodium and tetracyclines.

MOBAN (molindone hydrochloride) has an antiemetic effect in animals. A similar effect may occur in humans and may obscure signs of intestinal obstruction or brain tumor.

ADVERSE REACTIONS—CNS EFFECTS The most frequently occurring effect is initial drowsiness that generally subsides with continued usage of the drug or lowering of the dose.

Noted less frequently were depression, hyperactivity and euphoria.

Neurological—Extrapyramidal Reactions Extrapyramidal reactions noted below may occur in susceptible individuals and are usually reversible with appropriate management.

Akathisia Motor restlessness may occur early.

Parkinson Syndrome Akinesia, characterized by rigidity, immobility and reduction of voluntary movements and tremor, have been observed. Occurrence is less frequent than akathisia.

Dystonic Syndrome Prolonged abnormal contractions of muscle groups occur infrequently. These symptoms may be managed by the addition of a synthetic antiparkinson agent (other than L-dopa), small doses of sedative drugs, and/or reduction in dosage.

Autonomic Nervous System Occasionally blurring of vision, tachycardia, nausea, dry mouth and salivation have been reported. Urinary retention and constipation may occur particularly if anticholinergic drugs are used to treat extrapyramidal symptoms.

Hematological There have been rare reports of leucopenia and leucocytosis. If such reactions occur, treatment with MOBAN may continue if clinical symptoms are absent. Alterations of blood glucose, liver function tests, B.U.N., and red blood cells have not been considered clinically significant.

Metabolic and Endocrine Effects Alteration of thyroid function has not been significant. Amenorrhea has been reported infrequently. Resumption of menses in previously amenorrheic women has been reported. Initially heavy menses may occur. Galactorrhea and gynecomastia have been reported infrequently. Increase in libido has been noted in some patients. Impotence has not been reported. Although both weight gain and weight loss have been in the direction of normal or ideal weight, excessive weight gain has not occurred with MOBAN.

Cardiovascular Rare, transient, non-specific T wave changes have been reported on E.K.G. Association with a clinical syndrome has not been established. Rarely has significant hypotension been reported.

Ophthalmological Lens opacities and pigmentary retinopathy have not been reported where patients have received MOBAN (molindone hydrochloride). In some patients, phenothiazine induced lenticular opacities have resolved following discontinuation of the phenothiazine while continuing therapy with MOBAN.

Skin Early, non-specific skin rash, probably of allergic origin, has occasionally been reported. Skin pigmentation has not been seen with MOBAN usage alone.

MOBAN (molindone hydrochloride) has certain pharmacological similarities to other antipsychotic agents. Because adverse reactions are often extensions of the pharmacological activity of a drug, all of the known pharmacological effects associated with other antipsychotic drugs should be kept in mind when MOBAN is used. Upon abrupt withdrawal after prolonged high dosage an abstinence syndrome has not been noted.

Tardive Dyskinesia Although rarely reported with MOBAN (molindone hydrochloride) symptoms were reversible upon discontinuation of therapy.

Tardive dyskinesia associated with other agents has appeared in some patients on long-term therapy and has also appeared after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). There may be involuntary movements of extremities.

There is no known effective treatment of tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time the syndrome may not develop.

DOSAGE AND ADMINISTRATION Initial and maintenance doses of MOBAN (molindone hydrochloride) should be individualized.

Initial Dosage Schedule The usual starting dosage is 50-75 mg/day.
- —Increase to 100 mg/day in 3 or 4 days.
- —Based on severity of symptomatology, dosage may be titrated up or down depending on individual patient response.
- —An increase to 225 mg/day may be required in patients with severe symptomatology.

Elderly and debilitated patients should be started on lower dosage.

Maintenance Dosage Schedule
1. Mild—5 mg-15 mg three or four times a day.
2. Moderate—10 mg-25 mg three or four times a day.
3. Severe—225 mg/day may be required.

DRUG INTERACTIONS Potentiation of drugs administered concurrently with MOBAN (molindone hydrochloride) has not been reported. Additionally, animal studies have not shown increased toxicity when MOBAN is given concurrently with representative members of three classes of drugs (i.e., barbiturates, chloral hydrate and antiparkinson drugs).

MANAGEMENT OF OVERDOSAGE Symptomatic, supportive therapy should be the rule.

Gastric lavage is indicated for the reduction of absorption of MOBAN (molindone hydrochloride) which is freely soluble in water.

Since the adsorption of MOBAN (molindone hydrochloride) by activated charcoal has not been determined, the use of this antidote must be considered of theoretical value.

Emesis in a comatose patient is contraindicated. Additionally, while the emetic effect of apomorphine is blocked by MOBAN in animals, this blocking effect has not been determined in humans.

A significant increase in the rate of removal of unmetabolized MOBAN (molindone hydrochloride) from the body by forced diuresis, peritoneal or renal dialysis would not be expected. (Only 2% of a single ingested dose of MOBAN is excreted unmetabolized in the urine.)

However, poor response of the patient may justify use of these procedures. While the use of laxatives or enemas might be based on general principles, the amount of unmetabolized MOBAN in feces is less than 1%. Extrapyramidal symptoms have responded to the use of diphenhydramine (Benadryl*) and the synthetic anticholinergic antiparkinson agents, (i.e., Artane*, Cogentin*, Akineton*).

HOW SUPPLIED As tablets in bottles with potencies and colors as follows:

100's and 1000's:
5 mg orange
10 mg lavender
25 mg light green

As a concentrate containing 20 mg molindone hydrochloride per ml in 4 oz. (120ml) bottles.

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*Artane—Trademark, Lederle Laboratories
*Cogentin—Trademark, Merck Sharp & Dohme
*Akineton—Trademark, Knoll Pharmaceutical Co.

Rev. Nov. 1979 6102-5

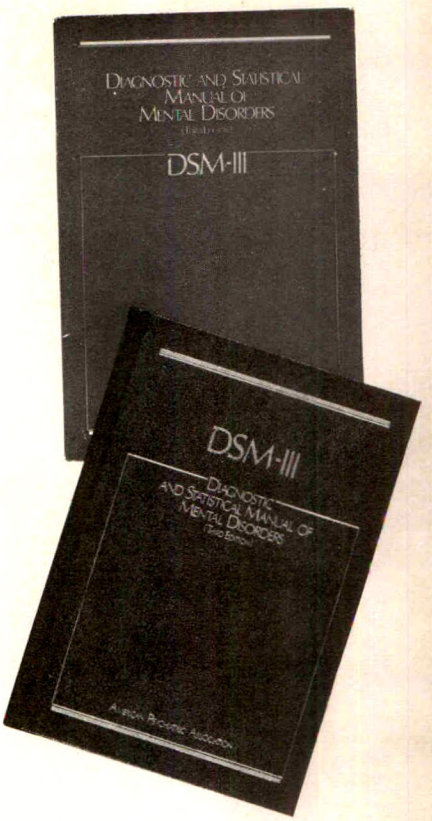
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WARNING
Lithium toxicity is closely related to serum lithium levels, and can occur at doses close to therapeutic levels. Facilities for prompt and accurate serum lithium determinations should be available before initiating therapy.

Indications: Treatment of manic episodes of manic-depressive illness. Maintenance therapy prevents or diminishes the intensity of subsequent episodes in manic-depressive patients with a history of mania.

Warnings: Lithium should generally not be given to patients with significant renal or cardiovascular disease, severe debilitation or dehydration, sodium depletion, or to patients receiving diuretics.

Lithium therapy has been reported in some cases to be associated with morphologic changes in the kidneys.

Caution patient and family to watch for diarrhea, vomiting, tremor, mild ataxia, drowsiness, or muscular weakness as signs of lithium toxicity, and to discontinue therapy and contact a physician should they occur. Patients receiving combined therapy with lithium and an antipsychotic should be monitored closely for early evidence of neurologic toxicity and treatment discontinued promptly if such signs appear. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery).

Lithium should not be used in pregnancy, especially during the first trimester, unless potential benefits outweigh possible hazards.

Not recommended for children under 12.

Precautions: Lithium tolerance is greater during the acute manic phase and decreases when manic symptoms subside.

Lithium therapy may lead to sodium depletion. Normal diet (including salt) and adequate fluid intake (2500-3000 ml) must be maintained, at least during initial stabilization period. Protracted sweating or diarrhea can decrease tolerance; in such cases, administer supplemental fluid and salt.

Each tablet contains 40 mg of sodium chloride, equivalent to 15.7 mg of sodium.

Sweating, diarrhea, and concomitant infection with elevated temperatures may require temporary reduction or cessation of dosage.

Adverse Reactions: Mild to moderate toxic reactions may occur at serum lithium levels from 1.5 to 2.5 mEq/L, and moderate to severe reactions at levels from 2.0 to 2.5 mEq/L. Fine hand tremor, polyuria, and mild thirst may occur during initial therapy and persist. Transient and mild nausea and general discomfort also appear during initial therapy. These effects usually subside with continued treatment or temporary reduction or cessation of dosage. If persistent, discontinue dosage.

Diarrhea, vomiting, drowsiness, muscular weakness, and lack of coordination may be early signs of toxicity and may occur at levels below 2.0 mEq/L. At higher levels, ataxia, giddiness, tinnitus, blurred vision, and a large output of dilute urine may be seen. Serum levels above 3.0 mEq/L may produce a complex clinical picture, involving multiple organs and systems. Serum levels should not exceed 2.0 mEq/L during acute phase.

The following reactions appear to be related to serum lithium levels, including levels within the therapeutic range: Neuromuscular—tremor, muscle hyperirritability (fasciculations, twitching, clonic movements of whole limbs), ataxia, choreo-athetotic movements, hyperactive deep tendon reflex; Central Nervous System—blackout spells, epileptiform seizures, slurred speech, dizziness, vertigo, incontinence of urine or feces, somnolence, psychomotor retardation, restlessness, confusion, stupor, coma; Cardiovascular—cardiac arrhythmia, hypotension, peripheral circulatory collapse; Gastrointestinal—anorexia, nausea, vomiting, diarrhea; Genitourinary—albuminuria, oliguria, polyuria, glycosuria; Dermatologic—drying and thinning of hair, alopecia, anesthesia of skin, chronic folliculitis, exacerbation of psoriasis, xerosis cutis; Autonomic—blurred vision, dry mouth; Thyroid Abnormalities—euthyroid goiter and/or hypothyroidism (including myxedema) with lower T_3 and T_4. I^{131} uptake may be elevated; EEG Changes—diffuse slowing, widening of the frequency spectrum, potentiation and disorganization of background rhythm; EKG Changes—reversible flattening, isoelectricity or inversion of T-waves; Miscellaneous—fatigue, lethargy, transient scotomata, dehydration, weight loss, tendency to sleep.

Reactions unrelated to dosage include: transient EEG and EKG changes, leukocytosis, headache, diffuse nontoxic goiter with or without hypothyroidism, transient hyperglycemia, generalized pruritus with or without rash, cutaneous ulcers, albuminuria, worsening of organic brain syndromes, excessive weight gain, edematous swelling of ankles or wrists, thirst or polyuria, sometimes resembling diabetes insipidus, and metallic taste. A single case of a syndrome resembling Raynaud's has been reported.

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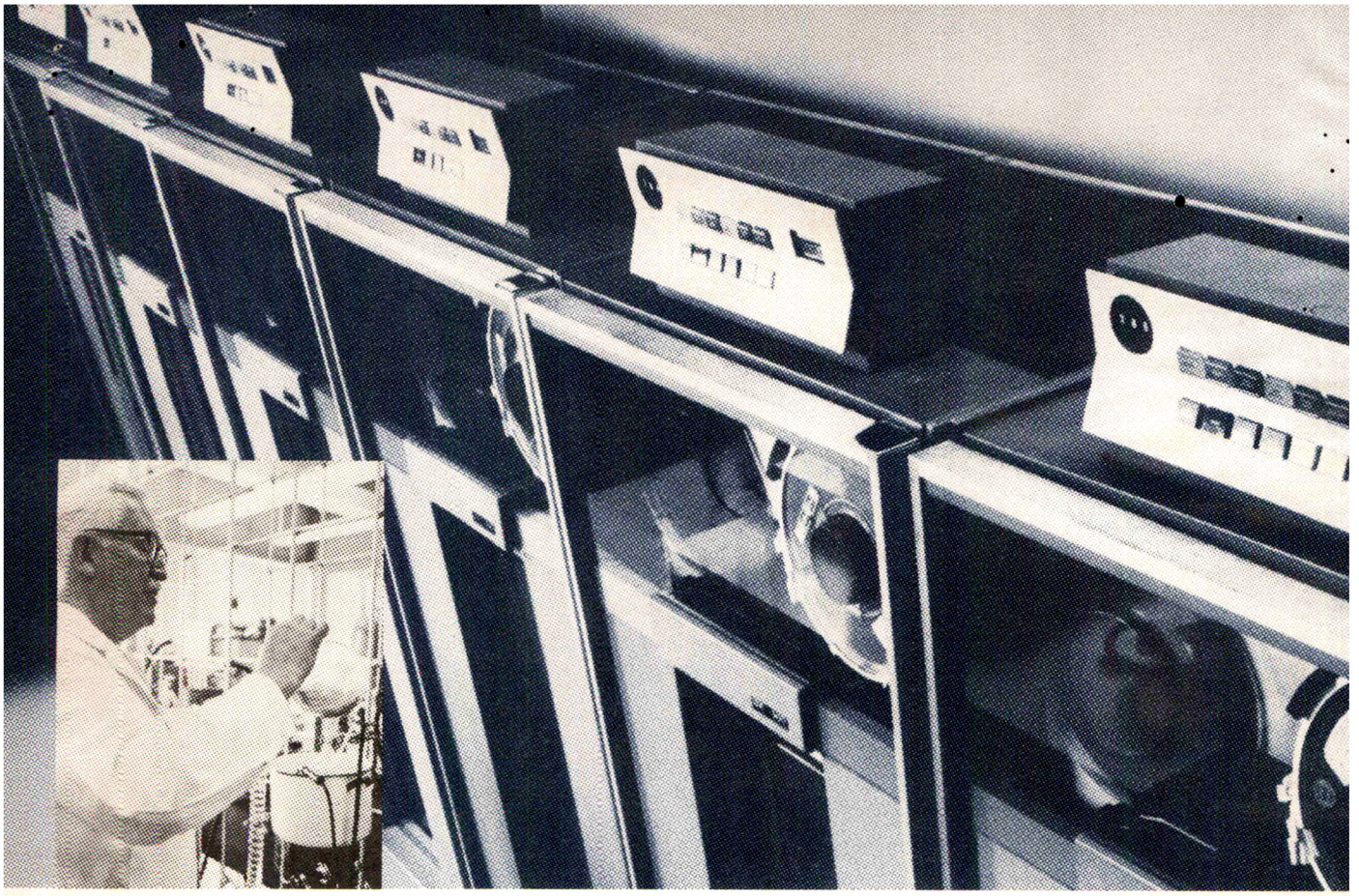
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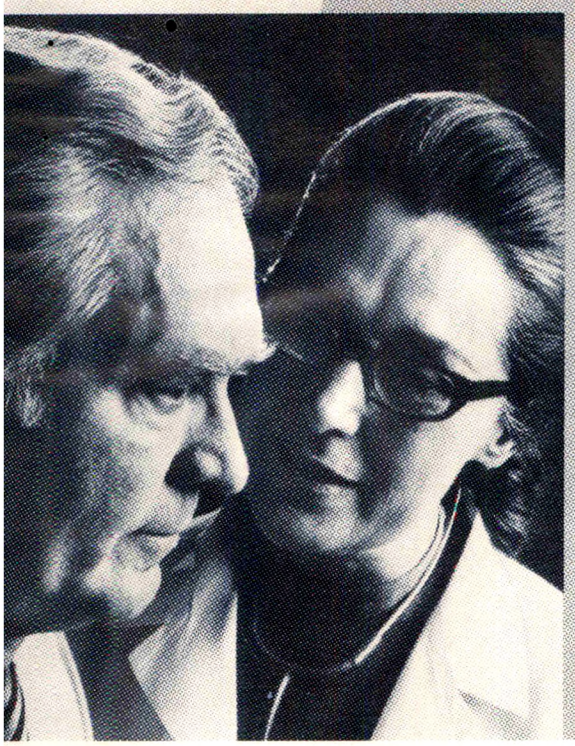
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Indications: Relief of anxiety and tension occurring alone or accompanying various disease states. Efficacy beyond four months not established by systematic clinical studies. Periodic reassessment of therapy recommended.

Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Warn patients that mental and/or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

Usual Daily Dosage: Individualize for maximum beneficial effects. *Oral–Adults:* Mild and moderate anxiety and tension, 5 or 10 mg *t.i.d.* or *q.i.d.*; severe states, 20 or 25 mg *t.i.d.* or *q.i.d. Geriatric patients:* 5 mg *b.i.d.* to *q.i.d.* (See Precautions.)

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Contraindications: Severe central nervous system depression, comatose states from any cause, hypertensive or hypotensive heart disease of extreme degree.

Warnings: Administer cautiously to patients who have previously exhibited a hypersensitivity reaction (e.g., blood dyscrasias, jaundice) to phenothiazines. Phenothiazines are capable of potentiating central nervous system depressants (e.g., anesthetics, opiates, alcohol, etc.) as well as atropine and phosphorus insecticides; carefully consider benefit versus risk in less severe disorders. During pregnancy, administer only when the potential benefits exceed the possible risks to mother and fetus.

Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy, observed primarily in patients receiving larger than recommended doses, is characterized by diminution of visual acuity, brownish coloring of vision, and impairment of night vision; the possibility of its occurrence may be reduced by remaining within recommended dosage limits. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving), and increase dosage gradually. Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension since phenothiazines may induce a reversed epinephrine effect on occasion. Daily doses in excess of 300 mg should be used only in severe neuropsychiatric conditions.

Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; rarely, nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—ECG changes (see *Cardiovascular Effects* below). *Other*—Rare cases described as parotid swelling.

It should be noted that efficacy, indications and untoward effects have varied with the different phenothiazines. It has been reported that old age lowers the tolerance for phenothiazines; the most common neurologic side effects are parkinsonism and akathisia, and the risk of agranulocytosis and leukopenia increases. The following reactions have occurred with phenothiazines and should be considered whenever one of these drugs is used. *Autonomic Reactions*—Miosis, obstipation, anorexia, paralytic ileus. *Cutaneous Reactions*—Erythema, exfoliative dermatitis, contact dermatitis. *Blood Dyscrasias*—Agranulocytosis, leukopenia, eosinophilia, thrombocytopenia, anemia, aplastic anemia, pancytopenia. *Allergic Reactions*—Fever, laryngeal edema, angioneurotic edema, asthma. *Hepatotoxicity*—Jaundice, biliary stasis. *Cardiovascular Effects*—Changes in terminal portion of electrocardiogram, including prolongation of Q-T interval, lowering and inversion of T-wave, and appearance of a wave tentatively identified as a bifid T or a U wave have been observed with phenothiazines, including Mellaril (thioridazine); these appear to be reversible and due to altered repolarization, not myocardial damage. While there is no evidence of a causal relationship between these changes and significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients showing characteristic electrocardiographic changes while taking the drug. While proposed, periodic electrocardiograms are not regarded as predictive. Hypotension, rarely resulting in cardiac arrest. *Extrapyramidal Symptoms*—Akathisia, agitation, motor restlessness, dystonic reactions, trismus, torticollis, opisthotonus, oculogyric crises, tremor, muscular rigidity, and akinesia. *Persistent Tardive Dyskinesia*—Persistent and sometimes irreversible tardive dyskinesia, characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and sometimes of extremities may occur on long-term therapy or after discontinuation of therapy, the risk being greater in elderly patients on high-dose therapy, especially females; if symptoms appear, discontinue all antipsychotic agents. Syndrome may be masked if treatment is reinstituted, dosage is increased, or antipsychotic agent is switched. Fine vermicular movements of tongue may be an early sign, and syndrome may not develop if medication is stopped at that time. *Endocrine Disturbances*—Menstrual irregularities, altered libido, gynecomastia, lactation, weight gain, edema, false positive pregnancy tests. *Urinary Disturbances*—Retention, incontinence. *Others*—Hyperpyrexia; behavioral effects suggestive of a paradoxical reaction, including excitement, bizarre dreams, aggravation of psychoses, and toxic confusional states; following long-term treatment, a peculiar skin-eye syndrome marked by progressive pigmentation of skin or conjunctiva and/or accompanied by discoloration of exposed sclera and cornea; stellate or irregular opacities of anterior lens and cornea; systemic lupus erythematosus-like syndrome.

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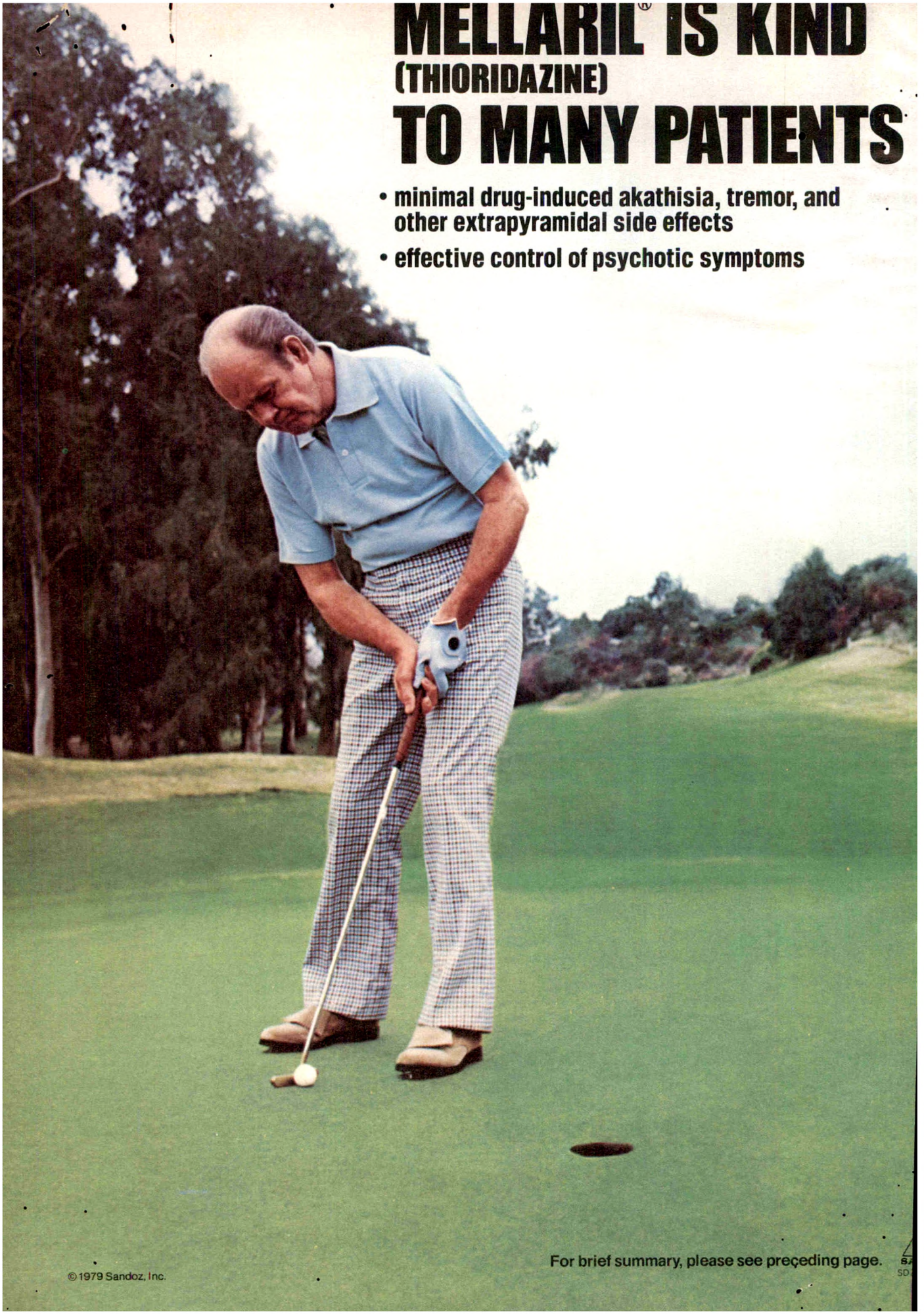
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THE AMERICAN JOURNAL OF PSYCHIATRY

Overview: Model Programs for Chronic Mental Patients

BY LEONA L. BACHRACH, PH.D.

Model programs for chronic mental patients may be viewed from four perspectives: evaluation of individual programs, commonalities in successful programs, generalizability and reproducibility of specific programs, and relevance of model programs to problems of service delivery in mental health systems. Although successful model programs share certain common structural elements, such programs cannot be readily reproduced or generalized. Having limited value for the problems of service delivery in mental health systems, model programs are best seen as experimental efforts, not as solutions. Strategies for translating model-derived knowledge into systems-related action are needed.

In the past two decades a sizable number of "model programs" have been developed to care for chronic mental patients in nontraditional and noninstitutional settings. Considered together, these programs are quite extraordinary. It is unlikely that 20 years ago many caregivers and service planners could have foreseen that so much creativity and innovativeness could be applied to the development of special programs for this patient population. As efforts to reverse the effects of institutionalism continue, the success of these programs provides hope for future service planning.

Simultaneously, however, the literature is replete with descriptions of failures in the care of chronic mental patients. Professional and popular sources alike report that the chronically mentally ill, in general, are not being adequately and humanely served by existing programs and that these patients are regularly overlooked by the mental health service system. When it comes to caring for chronic mental patients, service systems are typically reported to be fragmented and unresponsive, and the most seriously ill are more often than not described as "falling through the cracks."

How can these reports of successful model programs and glaring service system deficits be reconciled? What, precisely, is the relationship between demonstration efforts and the realities of mental health service systems? In this paper I will provide a conceptual framework for evaluating model programs for chronic mental patients and examine their contributions, their limitations, and their relationship to mental health service systems.

In this context the term "model program" is defined as any planned demonstration effort that tests the application of distinctive, often innovative, programmatic strategies to the care of chronic mental patients. Model programs are generally, although not necessarily, small in size, and their participants are usually selected in accord with the program's experimental aims. Often, although again not necessarily, funding of these programs is provided by external agencies for the specific purpose of testing special procedures or treatments.

Model programs for chronic mental patients are not limited to any particular setting. They may be administered under public or private auspices and have been described as occurring in such diverse places as general hospitals, community mental health centers, community action agencies, state mental hospitals, and even as part of a private psychiatric group practice (1-3). One model program reportedly operates on the streets of New York City. Funded by the state's office of mental health, that program uses a mobile van to seek out "shopping bag" people and others in need and to deliver them to treatment settings (4). Not to

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be confused in any way with the "medical" versus "nonmedical" treatment model controversy, model programs for chronic mental patients encompass a wide variety of treatment modalities, some fairly traditional and others decidedly innovative in nature.

Despite enthusiastic reports that many model programs are meeting the diverse needs of chronic mental patients effectively and compassionately, I have suggested elsewhere (5) that there are limits to the generalizability and reproducibility of these programs—that, in fact, they have often been used inappropriately to provide "answers" to questions concerning problems currently experienced by mental health service systems. Actually, model programs may be viewed from a variety of perspectives. In this paper I will discuss four different ways of looking at model programs for the care of chronic mental patients. The conceptual approach used is a layered one in which model programs are assessed through a series of questions representing different levels of abstraction, ranging from the most specific to the most general.

The first level of abstraction is evaluation and focuses on the question of how well any individual model program works. This is the lowest level of abstraction in that it looks at each program separately and makes no attempt at generalization. A second and higher level of abstraction is comparative and inquires as to whether there are principles common to model programs that may be extracted from them. Moving up the ladder of abstraction, a third approach assesses the generalizability of model programs and inquires into their reproducibility in settings other than their original locations. The fourth and highest level of abstraction is, like the first, evaluative, but with a major difference. The question here, instead of looking inward to the workings of individual programs, looks outward and asks, So what? This dimension of relevance focuses on the broader implications of model programs for the delivery of services to the total population of people who are chronically mentally disabled.

LEVEL I: EVALUATION OF INDIVIDUAL PROGRAMS

Every model program for chronic mental patients may be evaluated as an individual entity. Although evaluation of this kind is very important, it will be discussed only briefly because much has already been written on the need for adequate program evaluation (6–8).

Typically, evaluation of individual programs measures programmatic relevance and outcomes according to predefined criteria and is performed in-house or under contract. Evaluation of models, like evaluation of their nonmodel counterparts, may proceed in a variety of ways, depending on what information is being sought. It might, for example, depart from externally determined criteria for the effectiveness of community-based programs such as those proposed by James (9). Or it might inquire into the extent to which the stated objectives of the program are actually being implemented—i.e., the fit between the program's philosophy and its outcomes. Still other kinds of program evaluation might focus on quantifiable changes in patients' clinical status or level of functioning or on patient and/or staff satisfaction with the program's operation. It is for administrators and caregivers in particular programs to determine precisely what questions are important to ask in evaluating programs and how to ask them. This simplest and most specific level of abstraction approaches model programs singly, without reference to or comparison with other programs. In effect, it inquires as to whether a specific program is doing what it has set out to do.

LEVEL II: COMMONALITIES

Another approach assesses model programs comparatively and asks whether there are similarities or commonalities that can be extracted from them for more generalized application. In a recent analysis of diverse model programs selected for their apparent success in meeting the needs of chronic mental patients (5) I concluded that they have certain principles of planning and programming in common. Despite differences in specific locus of care and in control—i.e., whether the program is privately or publicly funded—successful model programs appear to be built on a minimum of eight principles that together form a least common denominator for effective program design for chronic mental patients.

1. Chronic patients targeted. Successful model programs assign top priority to the care of the most severely impaired: they are targeted toward patients who are chronically and persistently ill. If this seems too elementary a principle to merit specific mention, it may be noted that the very failure to assign first priority to chronic patients in many community mental health programs has caused major problems for this population. Zusman and Lamb (10) have contended that although the "basic mission" of deinstitutionalization was originally community-based treatment of chronic patients, "only limited aspects of the original conception have been implemented, while the interest of most workers has gone off in other directions." The result is that community mental health has largely overlooked the chronic patient and has tended instead to focus on those who are "healthy but unhappy." The very existence of chronic mental patients, who are singularly impotent in advocating on their own behalf, has frequently been denied (11, 12). When these patients, with their peculiar combination of service requirements, chronicity, and impotence, have had to compete for scarce resources with others who are less

severely impaired and more socially acceptable, chronic patients have not fared well.

2. *Linkage with other resources.* Realistic linkage with other resources in the community is a second element common to successful model programs for chronic mental patients. This principle is an affirmation of the range and the diversity of treatment needs of those who are chronically ill. In many ways, the planning of relevant deinstitutionalization programs for chronic mental patients is synonymous with planning for human services (11). In addition to psychotherapy, these patients require additional special services that include a "range of living environments and resocialization, vocational rehabilitation, and appropriate work opportunities" (13).

3. *Functional integrity.* Either by itself or in combination with the other resources to which it is linked, a successful model program attempts to provide for its patients the full range of functions that are associated with institutional care. Sociological analyses of the literature reveal that mental hospitals traditionally have fulfilled a variety of functions (14, 15). These include, in addition to long-term comprehensive treatment for disabled individuals, a series of additional functions less readily perceived or acknowledged (e.g., protecting the patient from exploitation or providing him or her with a place of refuge). Much of the difficulty associated with deinstitutionalization efforts may be attributed to the tendency for program planners to overlook, or to neglect to stress, the need for providing out-of-hospital alternatives for the full range of functions performed in hospital settings. Not even the central functions of treatment, asylum, and custody are assured for many deinstitutionalized individuals, not to mention the many peripheral functions. Successful model programs care about the mentally disabled not only as patients but also as social individuals in a complex society. They acknowledge that chronic patients comprise a dependent population for whom the fulfillment of certain basic conditions of human existence must be arranged—conditions that have traditionally, for better or worse, been met within large institutions. These programs are therefore responsive not only to the psychiatric and medical treatment needs of patients but also to their needs for asylum, respite, socialization, and rehabilitation.

4. *Individually tailored treatment.* Model programs that are regarded as successful make a point of providing for their patients personally tailored treatment regimens, whether chemotherapy, psychotherapy, psychosocial rehabilitation, and/or some combination of these and other treatment modalities. This principle of reaching out to patients on an individualized basis necessarily activates certain other elements of treatment that are widely understood as fundamental to the effective care of chronic mental patients. Program elements like 24-hour crisis intervention and case management are automatically assumed and assured when the principle of individualized programming is at work. Individualized programming renders successful model programs the conceptual opposite of "dumping" (indiscriminate and wholesale placement of patients either in hospitals or in the community without consideration for their specific needs).

5. *Cultural relevance and specificity.* Successful model programs are tailored to conform to the local realities of the communities in which they are located. Indeed, the very uniqueness of model programs stands out as one of their underlying similarities. Every model program is a very special event that can be studied and possibly adapted but not precisely duplicated. Because, as Abbott (16) has pointed out, the provision of community-based services for chronic mental patients "has major social components," any successful program for their care "must reflect the character of the community in which the patients are being served."

6. *Specially trained staff.* There is a need in successful model programs for trained staff who are attuned to the unique survival problems of chronic mental patients living in noninstitutional settings. This principle is of such central importance that Polak and Kirby (17) have written that any program lacking staff "who are trained and comfortable working with disturbed patients in community settings . . . is likely to have poorer outcome than psychiatric hospitalization." Special training includes strategies for dealing not only with problems in patient care but also with staff issues, like burnout, that affect role performance.

7. *Hospital liaison.* Successful model programs for chronic mental patients are tied in some manner (and sometimes reluctantly) to a complement of hospital beds. This principle is consistent with the growing view that there are certain patients for whom periods of hospital care continue to be a necessity (11). Irrespective of what specific reasons are considered appropriate for hospitalization—whether for the protection of the patient or of society or whether to provide the patient with diagnostic or intensive treatment services—successful model programs reflect the ever-increasing departure from the polarized antihospital stance that characterized the early years of the deinstitutionalization movement (14, 18, 19).

8. *Internal evaluation.* Descriptions of successful model programs indicate the presence of a kind of ongoing internal assessment mechanism that permits continuous self-monitoring. This kind of evaluation may be something quite apart from formal program evaluation, which tends to be too complex—with too slow a "turnaround" time—to be of use in the kind of equilibrating function that his principle implies. Internal evaluation in these models measures programmatic activities against previously agreed on standards and thus avoids the disjunction among planning, management, and evaluation that tends to characterize evaluative efforts for larger and more complex programs (6). Because model programs are usually relatively

small in size and uncomplicated in structure, they afford unique opportunities for feedback.

In summary, a search for commonalities in model programs that are considered successful demonstrates that there are certain principles that characterize them. The eight principles extracted and discussed here appear to be basic elements in programs that minister effectively to chronic mental patients by acknowledging that they have severe and recurrent problems, that they are often functionally impaired and in need of assistance in gaining access to the most basic of life's entitlements, and, most of all, that their individuality is of greater significance in effective treatment than is any categorical label, such as "schizophrenic," that might be applied to them.

These principles are all concerned with the structural aspects of programming for chronic mental patients and do not extend to program specifics like treatment modalities used or manpower patterns employed. They are simple enough to transcend differences in communities and in demographically distinctive target populations. Indeed, it is apparent that these principles are so simple that they may prove to be disappointing to those who look to model programs to provide instant solutions for their own communities' problems. Moreover, these principles show great similarity not only to the structural properties of effective mental health systems (20, 21) but also to the principles Talbott (22) has put forth as necessities for effective programming in state hospitals of the future. That is noteworthy: it reinforces the notion that the guiding principles of effective planning for chronic patients transcend locational considerations and have more to do with good and humane medical practices than with geography.

Finally, because these principles are structural and are not concerned with the substantive aspects of programming, they can potentially be applied in other setings—i.e., they can be generalized.

LEVEL III: REPRODUCIBILITY AND GENERALIZABILITY

Mental health planners have for some time sought to develop model programs for chronic patients that can be readily reproduced in a variety of settings. Thus Greenblatt and Budson (23), in a descriptive evaluation of five model programs, have asked, "Once demonstration of an effective modality has been made, how do we export it?" In the present context, the critical question is, May we generalize beyond the structural aspects of programming for chronic patients, as described above? It would, of course, be very comforting to be able to point to a laundry list of program specifics and proclaim that if one but includes all of them in a program design, a successful program will be born. Perhaps because the need for solutions to the many problems that plague deinstitutionalization is so acute (14), the assumption is often made that if a model program is proven successful in one place it can be exported to and duplicated in another. The widespread search for reproducible model programs represents a quest for quick-and-easy solutions to alleviate what has often amounted to widespread neglect of chronic mental patients. There is a hope that through identification and diffusion of appropriate models the care of chronic patients will be made more humane.

Unfortunately, there do not appear to be any simple answers. Just as the design of services for chronic patients has too often departed from information derived from "an unwarranted emphasis on the single-episode user of services, a deficiency of interest in people with lifelong disorders, and unwarranted expectations about the effects that programs of general social betterment can have on serious mental illness (24), so have hopes for diffusing model programs been based on the untested assumption that such a process is feasible. Although the structural principles discussed above are generalizable, it is exceedingly difficult—perhaps even dangerous—to attempt to transplant entire programs to alternate settings. Accordingly, those who seek easy answers and choose to view model programs as ready-made solutions for their own communities' problems should proceed with great caution. There are compelling reasons for not attempting to duplicate model programs in other settings.

For one thing, as suggested by the principle of cultural relevance and specificity, any program, model or not, has a cultural context. Abbott (16) has aptly pointed out that "certain models are not applicable to certain communities" and that "a system of care natural to one community may not be appropriate in another because of diverse social, cultural, and political elements." Separation of program specifics from the culture base in which they naturally occur can lead to irrelevant or inappropriate programming. Polak and Kirby (17), in their description of a community-based network of facilities for chronic patients in Denver (a program frequently held up as an extremely successful model) have questioned whether their program is reproducible, and they have specifically speculated about its suitability for rural areas.

No less important a consideration is the fact that many model programs have proceeded with special start-up funds and personnel. Attempts to adapt these programs to the realities of local budgets and resources will inevitably mean cutting out portions of the plan, and it is difficult to foresee the effects of cuts that are made for financial or any other reasons. Test and Stein (25), in discussing the renowned Training in Community Living program in Madison, Wis., have suggested that their own program differs from traditional programs along so many dimensions that it is difficult to sort out "which of the factors carry the greater part of the variance in accounting for the favor-

able results." These authors have said, simply, that beyond such elementary principles as those noted above, we do not know what it is that makes model programs work. Extricating from a complex field of variables a few program specifics has a potential for causing harm.

It is also impossible to know how much of a program's success comes from its having been singled out as a model to be funded and implemented and how much comes from actual program elements. Almost certainly there is a "Hawthorne effect" present in many model programs, a situation in which it becomes difficult to distinguish the positive effects of planned program principles from those which are brought about by "the mere fact of participating in an experiment" (26). Norman Ellis of the University of Alabama, in personal communications to me, has described an unpublished study of profoundly mentally retarded adults, previously considered untrainable, who showed great improvement in self-help, social, and interpersonal skills simply as the result of increased attention from staff. No new program initiatives had been undertaken in this instance, and no new money had been spent, but clinically assessed improvements nevertheless occurred.

An additional limitation on reproducibility comes from precisely how services are delivered and precisely who delivers them in model settings. As Rossi (27) has asserted, "It is difficult to separate the treatment from the manner of delivery." Model programs often attract what Rossi has called "exceptionally devoted persons," so that efficacious results are as likely to come from their ministrations as from the program itself. It is important to realize that the precise method of delivering services to chronic patients is of critical importance in a program's success and that this feature cannot be faithfully reproduced with any degree of certainty.

Finally, it is also important to realize that descriptions of model programs, although "exciting on their own terms," as Mechanic (28) has noted, "have yet to demonstrate that [they] can maintain their early momentum over long periods of time or communicate their enthusiasm to others."

In sum, it appears to be very difficult, if not impossible, to transplant a model program, either in its entirety or in some part, to a new setting. Several factors inhibit such diffusion; these include 1) the difficulty of separating a specific program from its culture base, 2) the difficulty of deciding what program elements are the most important ones to try to duplicate, 3) the danger of interfering with the program's Gestalt, 4) the existence of a "Hawthorne effect" that makes it difficult to assess what changes come from the program itself and what changes derive from the experimental milieu, and 5) the problem of separating program elements from the personnel who execute them.

The limited generalizability of model programs has been noted by Suchman (7), who has suggested that every model is, in effect, the statement of a hypothesis with "almost no generalizability" (p. 79). This is not to say, of course, that models have no values; that is hardly the case. But the value of model programs must be sought in something other than their reproducibility. Beyond the supply of some replicable structural fundamentals, the value of model programs should be sought in their experimental yield.

LEVEL IV: RELEVANCE

The fourth and final level of abstraction in analyzing model programs is, like the first, concerned with evaluation, but with a unique difference. Whereas traditional forms of evaluation look inward to criteria of program success, this kind of evaluation looks outward and uses events in the external world as yardsticks. Such reality testing is of particular importance in mental health planning because it examines the relationship of the model program to the broader scope of mental health service delivery. Since I have been unable to find in the literature a name for this variety of program evaluation, I will call it, for want of a standard term, "impact evaluation" to differentiate it from the better known process and outcome procedures. The difference between impact evaluation and other kinds of program evaluation may be approached metaphorically through the paradox that the operation was a success but the patient died. A given model may be very successful indeed, but does this necessarily mean that deinstitutionalization is "working"? Or does it mean only that a particular model program is working?

Impact evaluation is distinguished by the kinds of questions it poses. While the traditional evaluation might ask, Does this program work? impact evaluation asks, What does this program do to meet the needs of the total population of chronically disabled people? This latter question is one of great importance in view of the fragmented nature of mental health systems. Unless it is asked, a specific program runs the risk of going off into its own orbit with little meaning for the various needs of the population it is intended to serve. In the case of model programs, the question is even more apposite because these experimental endeavors have a strong potential for removing themselves from the mainstream of service delivery problems.

In this context one may ask of model programs, What was the question? If the question is, Do model programs prove that chronic mental patients can be treated successfully in a variety of nontraditional settings? the answer, based on examination of a number of operating programs, must obviously be yes—or, at least, yes, given the presence of certain fundamental program elements. If, however, the question is Can model programs by themselves provide ready solutions for the problems of treating these patients in a

mental health system? the answer must be negative. Beyond their demonstration of some very elementary principles of successful programming, model programs tell us only that individual model programs can work. The inductive leap from this position to the notion that because a given model is successful for a specific target population it can solve the range of problems associated with deinstitutionalization is based on faulty logic: the conclusion does not follow from the evidence at hand. Yet the search for model programs as "answers" to the problems of deinstitutionalization continues. Perhaps this is a natural consequence of the fact that the problems in deinstitutionalization are massive and complex, and the need for quick solutions is felt acutely.

Difficulties in providing services for chronic mental patients have often come from the existence of an "inverse system of care," in which "the most trained and skilled clinicians deal with the most articulate, interesting and likely to succeed clientele," while the existence of those patients most in need is largely ignored (29). Such denial of the most seriously ill exists against a backdrop of diverse planning needs for several subgroups of the total population of chronically ill people, all of whom have different service requirements. In addition to patients released from mental hospitals, planners must consider chronically ill people who are long-time residents of mental hospitals and remain on their rolls, those who are now entering mental hospitals and will become long-stay patients, those who are now entering mental hospitals and will stay for only a short while, and those who are in the community and who, as a direct consequence of deinstitutionalization practices, have never been hospitalized (30).

Each of these demographic subgroups represents, in its own way, fallout from the deinstitutionalization movement. There are problems associated with patient tracking that must be solved so that services can be provided for two rather diametrically different classes of patients who present specific problems for service delivery systems: those who are highly visible as they go again and again through the revolving doors of the service system and those who become virtually invisible when they exit through the doors of mental hospitals and then get lost. The problem of responding to the needs of all the chronically mentally ill affects model programs only indirectly, if at all, but it must be the constant preoccupation of service systems.

To complicate matters even further, it appears that the principles demonstrated to be common to successful model programs may even at time collide head-on with the needs and resources of mental health systems. The very factors that provide high marks for one may give opposite ratings for the other. Thus, for example, improvements in patients' levels of functioning that are directly related to the highly individualized treatment plans and the specially trained staff found in model programs may well meet with a "so what" kind of response in an era of rapid and continuing deinstitutionalization, particularly when "Proposition 13 fever" is rampant. In the face of a nationwide problem of crisis proportions, it is no wonder that model programs which serve only a few patients are viewed by many planners with skepticism.

It must be apparent that model programs provide answers to only some kinds of questions and that these questions are apt to have relatively little to do with the global needs of program planning for chronic mental patients. It would seem then, as suggested earlier, that the appropriate perspective from which to view model programs is for their heuristic possibilities in limited circumstances. In the words of Suchman (7), the model program's value lies precisely in its ability to "indicate the probable success of the planned program, to try out procedures, and to suggest modifications" (p. 140).

MODELS VERSUS SYSTEMS

In summing up the discussion of commonalities above I noted that although it is possible to reach some conclusions about the structural aspects of successful model programs, more specific program practices are not so readily generalizable. There is a reason for this. Questions concerning the "how" of programming for chronic mental patients must be determined largely by such extra-program considerations as timing, available resources, attitudes, and other local conditions. The answers to "how" questions are necessarily highly idiosyncratic.

Understanding these limits on the generalizability of model programs leads, of course, to the question, What is the use of these programs? The answer depends on what is being sought. If one insists on looking at model programs as prototypes for diffusion, he is probably headed for disappointment. If, on the other hand, one alters his perspective and thinks of model programs as hypotheses, not as answers, their value becomes more apparent. Every successful model program may be understood as a hypothesis, as a test for a series of assumptions regarding the effective care of chronic mental patients in a specific setting. Each is an ongoing research effort with evaluative potential.

Model programs have taught us much about the basic ingredients of programs that care, humanely and effectively, for chronic mental patients. Whatever the size, scope, treatment philosophy, affiliation, or location of a particular model program for these patients, a sine qua non of its success appears to be the application of the eight principles outlined above. Understanding these principles throws into bold relief some of the oversights that plague the provision of services to the chronically mentally ill. This is, in itself, a major contribution and a giant step forward; but, beyond this, model programs are not equipped to meet the aggre-

gate demands for services to chronic patients within a defined geographic area. What this means, quite bluntly, is that, to the extent that models and mental health systems are confused, a serious error is made. Major differences exist between mental health systems and model programs, which are important to grasp.

There is, first of all, a difference in the selection of patients to be served in model programs and in mental health systems. Models choose their target populations to conform to their program goals. If patients of a certain age or with a certain institutional history would strain the limits of the experiment's design, those patients need not be admitted to the program. The Soteria model, for example, limits its subjects to unmarried schizophrenic patients between the ages of 16 and 30 who have had more than one hospitalization of brief duration (31). A mental health system does not have the freedom to choose its patients in this way.

Similarly, some models focus on subjects' rehabilitation and exclude custodial or maintenance-level patients who are not considered appropriate candidates for skills training. A mental health system, however, must ultimately face the need to provide for the care of even those who, according to current knowledge, show little hope of successful rehabilitation. A mental health system runs the risk of falling into a seductive trap if it pretends that the needs of all patients can be served through model efforts. Rossi (27) has noted that "a delivery system can simulate success by delivering treatments to individuals who are most likely to recover" or, in the case of chronic mental patients, to exhibit marked and measurable improvement.

The service system, unlike the model program, frequently has difficulty in defining the limits of its task—in identifying precisely what people comprise its target population and how to locate them. Case-finding problems of models are much less complex than the overall epidemiological concerns of mental health systems. In fact, to the extent that there is a class of chronic patients who appear to fit nowhere at all (32), the elevation of model programs to systems solutions has potential for harming rather than helping by diverting attention from the very existence of these patients.

Moreover, model programs and mental health systems are typically accountable to different "masters." Models are primarily responsible for testing and evaluating innovative approaches for the care of those who are chosen for their special programmatic efforts. Mental health systems, however, are most often accountable to governing bodies, like legislatures, for whom mental health services, particularly those targeted toward the care of chronic patients, represent only a portion of their concerns. These bodies are not expected to be experimental in their orientation; they are more concerned with demonstrations of service comprehensiveness and cost effectiveness than with the efficacy of treatment procedures or with the more abstract notion of quality of care.

There are also differences in resource allocation patterns between models and mental health systems. In a model program resource allocation is focused, so that funding, personnel, and material resources are all directed toward patients selected for participation in the program. In a mental health system, however, there is competition for resources among different kinds of patients. There is, indeed, a hierarchy of patient needs in which priorities must be assigned. Miller (29) has cautioned us to "question models that smack of 'triage,' " but the assignment of priorities within a mental health system is essential. The problem is how to make those assignments equitably so that the most seriously ill are not denied services.

Even the kinds of evaluation performed in model programs and in mental health systems differ markedly. Although evaluation efforts of the kind described in the discussion of level I above—assessments of program outcome according to predefined criteria—may be sufficient for model programs, the goals of evaluation in mental health systems must necessarily be much broader and must deal with issues of accountability at a variety of levels (33–37).

To summarize, model programs and mental health systems vary in a number of critical ways. Systems have to serve all in need, not only those who fit in with some predetermined experimental aims. Systems must serve even those who show little hope of improvement. Although model programs can focus their resources on their selected patients, mental health systems have to deal with competition from different kinds of patients, and while mental health systems must account to governing bodies like legislatures, a model program's primary responsibility is to the controlled testing of an innovative approach.

In short, the problems that characterize the delivery of services in mental health systems are numerous and diverse. Model programs do not suffice to provide answers for these problems; they must be dealt with in other ways. Generally too few in number to have widespread effect on the problems of mental health systems and too limited in concept to be reproduced in other settings, model programs remain, in effect, interesting and informative experiments. Although they have been important to the development of principles of programming for chronic mental patients, they do not, in any sense, provide the global answers that are needed to solve the problems of deinstitutionalization.

The differences between the problems that affect model programs and those which affect mental health systems are exemplified in a U.S. General Accounting Office report on the Veterans Administration personal care home program (38), a highly successful model program for foster care placements of chronic patients that is now nearly 30 years old. That report states that, although many more patients qualify for care in personal care homes than are now enrolled in that program, they tend to remain in hospitals for such reasons

as insufficient funds, unavailability of enough suitable community facilities to effect more widespread placements, lack of adequate commitment to the program from the VA system's central office, and patient or family resistance to out-of-hospital placements. Clearly, this is a case where the model program, effective as it is, fails to provide an answer to the problems of the VA system of care.

CONCLUSIONS

This discussion leads logically to several conclusions. First, as Zusman and Lamb (10) have so aptly written, the concept of deinstitutionalization has not yet been given a fair test. The movement has somehow been diverted in community mental health programming from its original purpose so that the complex needs of the most seriously ill have been overlooked. This should not necessarily be construed to mean that most of the chronically mentally ill cannot be treated in community settings some day. It does mean that past errors and oversights—i.e., the confounding variables in deinstitutionalization—must be acknowledged and dealt with so that the problem can be given the cautious and objective evaluation it deserves.

Second, what is derived from model programs depends very much on what questions are asked. There are productive ways to view model programs. One way is to look at the scientific yield of each model's experimental efforts. Another way is to look at several successful models in concert to determine what elements they share. The latter approach yields some structural principles that characterize successful model programs and that are generalizable; they may be applied beyond model settings.

Third, when model programs are stretched in such a way that they are required to provide more than their natural limits permit, they lose some of their value. Planners of mental health services have too often looked to model programs to offer answers that they cannot, by their nature, provide. Specifically, for a variety of sociological and economic reasons, individual model programs cannot, as entities, be successfully generalized to other settings. Beyond the shared structural principles that they so ably demonstrate, their diffusibility is quite limited. If they are to be used in other settings, they must be specifically adapted to these new places. Moreover, there are even substantial risks in adapting these programs, because of difficulties inherent in selecting and extracting particular program elements.

Fourth, it is inappropriate to view model programs as solutions to the problems that mental health systems face. Model programs are too selective in their patient populations and too focused in their designs to be used as systems solutions. Accordingly, it is possible that a point in the testing of models has been reached where these experiments show much of a sameness. Perhaps, in other words, we have reached a plateau, and more model programs have relatively little new information to provide. If this is so, it is time to reassess the value of investing additional scarce resources in the piloting of more and more model programs. The question is no longer whether model programs are valuable and worthwhile—we know that they have been—but, rather, what the magnitude of our future investment in them should be.

The model programs that are pursued in the future should clearly attempt seriously to test new hypotheses—to examine previously untested structural fundamentals with the potential for adding materially to what is already known about the basic principles of effective planning. Unless there is some reason to believe that a new model can do this, it may be wiser to weigh the value of its implementation against the potential benefits to be derived from focusing on service systems.

There is little question that it is important now to direct more energy toward applying some of what is already known to mental health systems. The widespread absence of the eight general principles of effective programming from mental health service systems is glaringly evident. How, then, may the knowledge derived from existing models be applied to planning within mental health systems? What are the best ways to convert the structural principles of model programs into viable service system elements? How may mental health systems apply those principles in the face of the special problems imposed by their need to establish priorities for resource allocation? How may the program evaluation efforts of mental health systems be redesigned to yield assessments of those program principles while they also address the systems' special needs for accountability?

It seems that these are relevant questions for the 1980s. Although the literature is now beginning to focus more intensively on the problems of mental health systems and their solutions, there is a real need to develop strategies for translating model-derived knowledge into systems-related action. It will take fundamental changes in attitudes and funding practices to shift from the habit of looking to models as solutions toward integrating what is already known from them into service delivery practices. But a continuing failure to place model programs in perspective will only divert attention from the more global needs of the population of chronically mentally ill people and will prolong the serious problems that are now, unfortunately, widely identified with the deinstitutionalization movement.

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The Use of ECT in the Treatment of Schizophrenia

BY CARL SALZMAN, M.D.

ECT has been replaced by neuroleptics for the treatment of schizophrenia. The production of serious drug-related side effects, particularly tardive dyskinesia, raises the question of the efficacy and toxicity of ECT versus neuroleptics. Most of the studies in the English literature on the use of ECT in the treatment of schizophrenia are unacceptable according to contemporary criteria; the question of ECT versus neuroleptic drugs thus remains unanswered. In the few acceptable published studies, clinical response to ECT was inversely proportional to duration of schizophrenic symptoms. Schizophrenic patients with affective and catatonic symptoms responded best; those with chronic symptoms rarely responded. ECT does not alter the fundamental psychopathology of schizophrenia.

Electrically induced convulsions, administered by Cerletti and Bini in 1938 to a catatonic schizophrenic patient, introduced ECT into clinical psychiatry (1). ECT was a modification of chemically induced seizures. It was based on an earlier erroneous belief that schizophrenia and epilepsy could not coexist in the same patient as well as the observation that patients with psychotic symptoms sometimes improved after spontaneous or accidentally induced seizures (2). It was reasoned that the induction of epileptic-like seizures, either chemically or electrically, would thus be an effective treatment for schizophrenia.

USE OF ECT BEFORE THE INTRODUCTION OF NEUROLEPTICS

One of the earliest and most repeated observations was the favorable response to ECT of acutely ill schizophrenic patients compared with those who had a long and chronic illness. Most authors defined acute illness as one year or less of symptoms. Those who had been ill less than 6 months had the best response of all, whereas patients who were ill more than 2 years showed very poor response (3, 4). Early studies of ECT with schizophrenic patients also emphasized the symptomatic improvement achieved with this treatment even if hospital discharge was not attained. This improvement, seen in acutely ill as well as agitated patients, led to a dramatic decrease in the use of barbiturates, restraints, wet packs, and isolation (5). Patients with catatonic or affective symptoms responded particularly well to ECT (5, 6). In an early paper Pacella and Barrera (7) commented that patients who improved "almost always had strong affective components of depression, guilt, worthlessness, hopelessness, self-condemnation and suicidal tendencies." However, there was considerable disagreement among authors about the role of ECT with the paranoid patient. The earliest follow-up studies of successfully treated patients with acute schizophrenia suggested a low relapse rate. However, subsequent follow-up studies reported relapse rates as high as 83% (8). These follow-up studies led to the conclusion that ECT was useful primarily for patients who had a favorable prognosis and who would have recovered with other treatments or with no treatments (9). The results of ECT with patients who were chronically ill with schizophrenia and who had a poor prognosis were uniformly discouraging.

Early Studies

In the early reports of ECT efficacy in schizophrenia, diagnosis of schizophrenic patients was by clinical judgment rather than by adherence to rigorous diagnostic criteria. Similarly, the standards for remission, recovery, or improvement often were not specified or were made by clinical judgment and thus varied from report to report. The number of treatments also varied greatly from study to study, and control groups were rare. Nevertheless, the results of these early clinical reports were similar enough to draw general inferences regarding the role of ECT in the treatment of schizophrenia. A number of these observations are still valid today, despite more rigorous and sophisticated clinical and experimental methodology.

1. ECT offers symptomatic relief for acutely ill patients. Social and self-care behavior improve, and hospital discharge rates increase.
2. The shorter the duration of illness, the better the

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outcome with ECT. Patients who have been ill less than one year do much better than patients ill more than one year.

3. Acutely ill patients with catatonic or affective symptoms respond best.

4. Chronic, deteriorated patients and patients who have been symptomatic for more than 2 years have a poor response to ECT.

5. Relapse rates are high for all patients who improve. As the duration of illness lengthens, the probability of relapse increases.

ECT IN THE ERA OF NEUROLEPTICS

Studies of ECT Without Drug Comparisons

Lehmann (10) introduced the replacement of ECT by neuroleptics as a treatment for schizophrenia. He reported that "the time required for full recovery (with chlorpromazine) is shorter than with electroconvulsive treatment, and furthermore, the patient's insight is often better because awareness and confusion do not develop under treatment." Drugs soon replaced ECT for the treatment of schizophrenia in hospitals throughout the world. Nevertheless, studies of ECT in schizophrenia continued. Small and associates (11) reported an overall patient discharge rate of nearly 30% at 60–90 days after ECT or fluorothyl convulsive treatment. This rate was lower than many of those reported in the early years of ECT treatment. Doongaji and associates reported a 50% reduction in symptoms (12) in acutely ill schizophrenic patients, but after 3 months only 21 of the 46 patients available for follow-up had sustained their improvement.

Wells (13) reported a 10-year survey of ECT in the treatment of nonchronic schizophrenic patients who had not responded to phenothiazines. A total of 276 patients received an average of seven treatments, and 75% were thought to have a decrease in symptoms; catatonic and schizoaffective patients did best. However, 2 years later 46% of the 95 patients available for follow-up had relapsed. Wells concluded that only patients suffering from a first episode of schizophrenia that included depressive symptoms were good ECT responders. In a retrospective survey of 118 consecutively admitted patients, Folstein and associates (14) found that good response to ECT was predicted by the presence of affective disorder. These factors predicted good therapeutic outcome regardless of whether the patient was also diagnosed as schizophrenic. In contrast, patients who had only Schneiderian first-rank symptoms for schizophrenia did not improve with ECT. Dempsey and associates (15) presented the favorable outcome of 5 acute schizophrenic patients with a history of drug abuse. These patients were initially diagnosed as schizoaffective and failed to respond to phenothiazines, but all responded well to a course of eight sessions of ECT followed by maintenance lithium carbonate therapy. In addition to these studies, reports by other experienced clinicians have been enthusiastic about the role of ECT as the primary treatment for acute schizophrenia (16–19).

In addition to the many recent studies of ECT efficacy in acute schizophrenia, several reports have found ECT to be useless in chronic schizophrenic patients (20). Although symptoms of agitation and depression are diminished by ECT (21), behavioral and affective improvement is not lasting (22).

Comment

Although ECT has been replaced by neuroleptics for the treatment of schizophrenia, there has been a recent reexamination of ECT as the primary treatment form for acute schizophrenia. Despite improvements in research design and increased clinical sophistication about schizophrenia, the overall results of these recent reports do not alter the early conclusions regarding ECT. Acute schizophrenic patients, particularly those with affective symptoms, respond rapidly to ECT. Relapse rates are still high, and chronic schizophrenic patients are still refractory to ECT. ECT can offer a temporary diminution of agitation and depression in all schizophrenic patients.

Studies Comparing ECT with Neuroleptics

After the early demonstrated efficacy of chlorpromazine and other neuroleptics for the treatment of schizophrenia, the obvious research task was to determine whether there was a differential efficacy between drugs and ECT. Two types of studies were conducted to answer this question. In the first type, ECT-treated patients were compared concurrently with patients receiving neuroleptic drug treatment. In the earliest study of this type (23), relapse rates were lowest for the ECT group (31%) compared with the insulin group (55% relapse rate) and the chlorpromazine-treated patients (91% relapse rate). Langsley and associates (24) found similar rates of improvement in a group of patients receiving ECT and those who received neuroleptic treatment. Fifty-two acutely ill schizophrenic patients who received 15–20 unmodified sessions of ECT were compared with 54 similar patients who received daily doses of 800 mg of chlorpromazine. The treatments were equal in improving affective symptoms, but chlorpromazine patients stayed in the hospital a shorter time, had less fear, and seemed to experience better relief of subjective distress. However, overall ratings by psychiatrists failed to differentiate between the treatments. Ayres (25) studied 263 schizophrenic patients who received one of several treatments. Only 9% of the 89 patients who received ECT alone needed rehospitalization; 20% of those who received chlorpromazine alone required rehospitalization. Ayres did not provide data on diagnostic types of schizophrenia, length or severity of illness, dose of chlorpromazine, or number of ECT sessions adminis-

tered. King (26) and Childers (27) also observed equal improvement rates in groups of acute schizophrenic patients who received ECT and those who received chlorpromazine. King found that ECT patients remained in the hospital longer than the drug-treated patients.

The second type of study consisted of retrospective comparisons of schizophrenic patients who were hospitalized before 1955 and therefore treated primarily with ECT and of similar patients who were hospitalized after 1955, when drugs were used. Achte and Apo (28) compared 427 schizophrenic patients hospitalized from 1950 to 1952 with 542 schizophrenic patients hospitalized from 1957 to 1959, after the introduction of neuroleptics. For the first-admission patients, 93%–95% were discharged within 2 years regardless of the type of somatic treatment received. Relapse rates were high in both groups. Within 3–5 years 39%–47% of the discharged patients had been readmitted. Pritchard (29, 30) conducted a similar study of 41 patients who were hospitalized from 1952 to 1953 and 47 patients hospitalized from 1956 to 1957. The first group which received ECT primarily, stayed out of the hospital longer after discharge than did the later group of discharged patients. Within the second group, ECT was judged slightly better than chlorpromazine in regard to patient outcome and follow-up. In both groups of patients, those with the shortest duration of symptoms had the best long-term results. Pritchard concluded, however, that "overall there is little true difference between groups in duration of stay in the hospital, or in readmission rates."

Rovere (31) studied 105 schizophrenic patients treated from 1950 to 1960, as neuroleptic drugs were being introduced. He noted that the recidivism rate, which was 70% in 1950, had decreased to 30% by 1960. He concluded that when drugs replaced ECT in the treatment of schizophrenia, the recovery period was shorter and recidivism was less frequent. A similar examination of the decade from 1953 to 1964 also revealed a trend toward shorter hospital stays as treatment methods shifted from only ECT in 1953 to only phenothiazines in 1964 (32). Lassenius and associates (33) compared three groups of first-admission schizophrenic patients who were admitted from 1944 to 1946, 1955 to 1956, and 1959 to 1960. ECT and other shock treatments, used as the primary treatment for schizophrenia between 1944 and 1946, were gradually replaced by drugs so that by 1959–1960 schizophrenic patients received mainly pharmacotherapy. The authors noted that drug treatment of schizophrenia has been associated with a reduction in hospital and other institutional care and consider pharmacotherapy to be the treatment of choice for this disorder.

Comment

These comparative and retrospective studies as a group suggest that schizophrenic patients in general respond similarly to ECT and pharmacotherapy. However, the studies contain many methodologic deficiencies that militate against a definite conclusion. Since great variability exists in diagnostic criteria, the number of ECT sessions, and the dosage of drugs used, as well as in the overall treatment philosophy of different institutions, these studies can only be said to approximate each other. However, it is possible to conclude with certainty that none of these studies suggests an overwhelming therapeutic advantage in acutely ill schizophrenic patients either for ECT or for pharmacotherapy. Furthermore, there seems to be no differential prophylactic effect on readmission rates between the treatments. However, drug-treated patients definitely have shorter hospital stays than do ECT patients.

The Camarillo Study

In 1965 May and Tuma (34) published the first of a series of reports concerning five treatment modalities for schizophrenic patients at Camarillo State Hospital (Camarillo, Calif.). The patients for their study were considered to be in the middle section of the prognostic range—they were neither extremely chronic nor so acutely ill that they were likely to rapidly improve regardless of treatment. The five treatment methods were individual psychotherapy, pharmacotherapy, pharmacotherapy combined with individual psychotherapy, ECT, and milieu therapy. The range of ECT sessions was 10–48, with a mean of 22. The primary neuroleptic used was trifluoperazine in the range of 10–40 mg/day, with an average daily dose of 25.5 mg. Many outcome measures were employed in a sophisticated research design in order to compare the five treatment modalities.

On all measures of immediate outcome, pharmacotherapy with or without psychotherapy was superior to ECT. Of the 48 patients treated with drugs, 46 (95%) were discharged within 1 year, compared with 42 (95.5%) of the 44 patients who received drugs combined with psychotherapy and 37 (78.7%) of the 47 patients who received ECT (35). May concluded that "it is reasonably clear that electroconvulsive therapy cannot be considered to be desirable as an alternative or serious rival to ataraxic drugs alone or to psychotherapy plus drugs." He hypothesized that ECT may be useful for the patient who develops serious toxic effects from drugs or who fails to respond to drugs alone or to drugs plus psychotherapy. It is possible, however, that some patients will respond only to ECT.

The original report (34) contained a 3-year follow-up which showed there were no statistical differences between the ECT patients and the two drug groups in the number of days spent in the hospital since original treatment. A subsequent study (36), which extended the follow-up to 5 years, found that drug-treated and ECT-treated patients seemed to spend approximately the same number of days out of the hospital after their

initial period of treatment. ECT patients may, in fact, do slightly better than the drug-treated patients. May and associates (36) concluded that it seems unlikely that the shorter stay of ECT patients represents a conscious avoidance of the hospital because of fear of the treatment. They did not present data to support this conclusion but stated only that the ECT was delivered under humane conditions. These follow-up findings led Shader (37) to hypothesize that if patients "are given ECT and reintegrate rapidly and therefore have brief initial hospitalization, this rapid pulling together helps patients to remain out of the hospital over the subsequent years."

Comment

The Camarillo study represents the most sophisticated and carefully controlled comparison of ECT versus other treatment modalities. The overall conclusion is that ECT has been justifiably replaced by neuroleptic medication. However, more than three-fourths of the mid-prognostic range schizophrenic patients who received ECT did respond to it, and as a group they remained out of the hospital as long as or slightly longer than drug-treated patients.

ECT Combined with Neuroleptics

Studies of the efficacy of ECT in combination with neuroleptics have attempted to determine whether the combination is more therapeutic than either treatment alone. In early studies (3) the two treatments seemed to enhance each other and produced as much as an 83% improvement in acute schizophrenic symptoms. Less ECT was used, which shortened the period of post-ECT confusion and thereby reduced the length of hospitalization. The treatment was also found beneficial for chronic schizophrenic patients (38–41). One of the more authoritative early supports for combined ECT and chlorpromazine treatment came from Rohde and Sargant (42). These authors routinely used 300 mg of chlorpromazine per day and ECT in the treatment of schizophrenia. The authors compared the combined results with those of ECT alone. At discharge, 69% of the ECT-alone patients still had evidence of schizophrenia, compared with only 29% of the combined treatment group. ECT in combination with chlorpromazine also decreased the length of hospital stay. The beneficial effects of the combination treatment were still apparent 2 years later, at which time 51% of the ECT group were still psychotic, compared with only 20% of the combined treatment group. This early enthusiasm for the ECT-drug combination was not unanimous (43, 44), and serious side effects were sometimes observed (45, 46). In reviewing the early literature, Grinspoon and Greenblatt (47) wrote that ECT plus phenothiazines caused an increased risk and that "one cannot be assured that combined therapy counterbalances what may be an increased risk."

Contradictory reports on the efficacy of combining ECT with antipsychotic drugs have continued to appear, although more authors have favored the combination. Childers (27) reported the results of a comparative study of four treatment methods for acute schizophrenia. Group A (N=20) received 12 ECT sessions and had a 55% improvement rate; group B (N=20) received 20 mg of fluphenazine per day for 30 days, with a 45% improvement rate; group C (N=20) received 1000 mg of chlorpromazine for 30 days, also with a 45% improvement rate; and group D (N=20) received 1000 mg of chlorpromazine as well as ECT, with an 80% improvement rate. No follow-up data were provided, nor were the criteria specified for diagnosis of schizophrenia or for improvement. Nevertheless, the authors concluded that the combination of neuroleptics and ECT "appreciably increases the percentage who attain a moderate or better improvement." There were no severe side effects from the combination.

Kelly and Sargant (48) and Roth and associates (49) noted immediate but transient improvement when ECT was combined with neuroleptics in the treatment of chronic schizophrenic patients. When compared with the results of ECT alone, however, both forms of treatment produced improvement followed by relapse, although the patient's hospital stay was shortened by the combination. Regestein and Roper (50), however, reported lasting remission of schizophrenia in an 8-month follow-up of a single patient. In a placebo-controlled study of chronic schizophrenic patients, Gambill and Wilson (51) found that ECT alone and drug (prochlorperazine) alone were far better treatments than the combination of ECT and active drugs, which was only slightly better than placebo.

Achte (52) retrospectively compared schizophrenic patients who were in Helsinki hospitals in 1960 and received ECT combined with neuroleptics with patients in 1950 who received ECT and other "shock" treatments. Although the discharge rate was higher for the combination treatment group, 5 years later the overall improvement rate was not statistically different from that of the earlier group. Nevertheless, Achte concluded that the prognosis for schizophrenia was more favorable in 1960 than in 1950.

Smith and associates (53) studied both acute and chronic patients and found an immediate favorable response with combined treatment. They compared 29 patients who received 12 ECT sessions plus 400 mg daily of chlorpromazine with 25 patients who received only 655 mg of chlorpromazine per day. Although both groups improved significantly, the combination group started to improve within 1 week and continued to improve faster than the drug group. Patients who received the combined treatment were discharged sooner and were readmitted less often. At 6-month or 1-year follow-up, there was no difference in the overall ratings of psychopathology between the two groups. Within 1 year after the original admission 8 (33%) of

the patients treated with drugs alone had been readmitted, compared with only 2 (10%) of the patients receiving combination therapy. These data strongly favored the combination of ECT and chlorpromazine for the treatment of schizophrenia.

The combination of drugs and ECT was found to be superior to each treatment alone in hospitals in Pakistan (54) and in Cairo (55). Weinstein and Fischer (56) unequivocally supported the use of ECT combined with drugs. They did not offer data, but presented four case histories and their extensive clinical experience to illustrate that the combination is particularly useful for patients who do not initially respond to ECT and then reach a plateau when treated with phenothiazines, like the patients of Witten (57). ECT "lifted" these patients off the plateau, reduced their need for high drug doses, and prevented relapse. Wessels (58) found unilateral and bilateral ECT combined with thioridazine to be equally efficacious for the treatment of acute schizophrenia.

Two critical reviews of the ECT-drug combination literature have been less enthusiastic about the therapeutic efficacy of combining the two treatments. Turek (59) criticized most reports as having an uncontrolled research design, variable number of treatments, heterogeneous diagnostic groups, and no follow-up. He concluded that more rigorous research is needed before the combination of ECT and neuroleptic drugs can be considered better than either treatment alone, particularly for chronic schizophrenic patients. Abrams (60) also reviewed the literature and commented that the data supporting the combination therapy are unacceptable and more research is needed.

Comment

Although early reports were enthusiastic, most of the later studies of the therapeutic efficacy of ECT combined with neuroleptic drugs have been equivocal. However, the excellent study of Smith and associates (53) strongly favored the combination. As with other treatment forms, the combination is most beneficial for acutely ill patients. Further carefully controlled studies are necessary before the combination can be judged superior to either treatment alone.

The Camarillo study suggests that neuroleptic drug administration after a course of ECT may be beneficial as a follow-up treatment for schizophrenic patients in the middle section of the prognostic range. The 3- and 5-year follow-up data were based on patients who received treatment of their physician's choice after the initial hospitalization with ECT. In practice, most patients who needed posthospital treatment probably received antipsychotic medication.

The potential toxicity of combining ECT and neuroleptic drugs is still unknown. Most later reports have stressed the safety of the combination. However, neuroleptic drugs that have hypotensive properties may augment the initial blood pressure drop after ECT. In older patients and those with vulnerable cardiovascular systems, a hypotensive crisis can result. Such an effect is more common when ECT is combined with reserpine, and this combination is no longer used.

REGRESSIVE AND MULTIPLE MONITORED ECT

Regressive ECT

An interesting modification of standard ECT procedure was introduced in 1946 as a possible treatment for chronic schizophrenia. This technique consisted of a large number of ECT sessions in a short period of time, usually 2–4 a day on a daily basis. The treatments were given until an acute brain syndrome was produced. In this state the patient was regressed and exhibited memory loss, marked confusion, disorientation, lack of verbal spontaneity, and slurred speech to the point of complete dysarthria. The patient was also apathetic, incontinent of urine and feces, often required spoon feeding, and occasionally was not even able to swallow, thus requiring tube feeding. Neurological signs included ataxia, spastic rigidity, hyperreflexia, occasional ankle clonus, and pathological reflexes (61). As the patient recovered, usually within 1–4 weeks, the symptoms of schizophrenia were often absent or diminished. The drastic nature of the treatment was justified by the treatment-resistant and hopeless nature of the chronic schizophrenic patient who had been hospitalized for many years. Most (although not all) early studies concluded that regressive ECT was useless with chronic schizophrenic patients.

After these negative accounts, regressive ECT was not discussed in the literature until 1973. Exner and Murillo (62, 63) then published a study of 32 chronic schizophrenic patients who received twice-daily ECT for 7 days a week to a total of 26 convulsions. They compared these patients with 21 process schizophrenic patients who received an unspecified dose of haloperidol for an equivalent period of time. Those treated with regressive ECT showed significantly greater improvement on all outcome measures including ratings of psychopathology, self-reports of symptom distress, family reports of patient behavior, and indices of social adjustment. Hospital discharge rates were higher among the regressive ECT patients. At 3-year follow-up these patients were doing remarkably well and significantly better than the drug-treated control group. Many of them were working and continuing to live out of the hospital. Spensley (64) criticized this study for using inadequate dosages of haloperidol, thus biasing the results in favor of the regressive ECT patients. The authors responded that not only did they use adequate doses of haloperidol, but the drug-treated patients also "manifested complete remissions and were discharged from the hospital" (65). This comment raises serious questions about the chronicity of the patients included

in the study. It would be very unlikely to have such a high remission rate for chronic schizophrenic patients, regardless of the treatment they received.

Multiple Monitored ECT

Multiple monitored ECT is a technique in which treatments are given several times a day, but at a lower electrical intensity than that used with conventional ECT. Seizure activity is monitored by EEG, while cardiac response is monitored by ECG. In the original description, Blachly and Gowing (66) reported the technique to be as safe as or safer than conventional ECT, with less memory loss. White and associates (67) noted that all 4 acute schizophrenic patients given this treatment had a complete remission, and 3 of 5 chronic schizophrenic patients improved. Bridenbaugh and associates (68) reported that 14 of 17 acutely ill schizophrenic patients given multiple monitored ECT had a marked improvement. During a follow-up period of unspecified length, there was no recurrence of illness. Abrams (69, 70) gave 38 patients with mixed diagnoses 4–6 sessions of multiple monitored ECT and reported that the side effects were not different from those of conventional ECT, although the course of treatment was accelerated in many cases. This technique therefore is considered to produce results at least as good as those of conventional ECT and uses less power to induce seizures (71).

Comment

Regressive ECT is a drastic procedure that has produced equivocal results. The majority of observations have been negative. Most of the favorable reports either have commented on the temporary nature of the improvement or lacked follow-up data. While Murillo and Exner's report (63) is provocative, it may have serious methodologic faults concerning the diagnosis of patients. It seems reasonable, therefore, to consider regressive ECT as an experimental procedure requiring further careful research evaluation.

Multiple monitored ECT seems to hold promise as a variant of the conventional technique of ECT administration. It is not widely used and should be considered as experimental until additional reports of its use and efficacy are available.

DISCUSSION

Scientific Acceptability of the Literature on the Use of ECT with Schizophrenia

Contemporary standards for clinical research studies in psychiatry include rigorous diagnostic criteria, standardized treatment methods, control groups, independent or double-blind raters, specific outcome criteria, and adequate follow-up. Most of the early clinical reports in this review understandably do not meet these criteria. Riddell (72) reviewed 42 clinical research reports that had been published before 1962 and found only 10 of them scientifically acceptable. She stated, "It is apparent that not only is there a distressing lack of acceptable research into ECT as a therapeutic agent, but that very few valid conclusions can be reached from the studies which have been carried out." Riddell called for more rigorous research.

Unfortunately, many of the subsequently published research reports of ECT efficacy in schizophrenia also cannot meet current standards of acceptability, since only 18 have employed a control group for comparative purposes. Eight of these have been retrospective studies (13, 28–30, 32, 33, 49, 52, 73). Although such studies are useful in establishing trends in treatment efficacy, variable diagnostic criteria and treatment methods limit the conclusions that can be drawn.

Prospective studies that compare concurrent treatment methods are more useful in providing data about differential treatment efficacy. Twelve of the prospective studies published since 1963 have included a comparison or control group of patients. Five of these (12, 55, 58, 74, 75) studied electrode placement rather than different treatment modalities. A sixth study (11) compared ECT with fluothyl-induced convulsions. Reports of comparative studies by Childers (27) and by Murillo and Exner (63) did not include adequate diagnostic information and thus are unacceptable. Neither Gambill and Wilson (51) nor Rahman (54) included follow-up data in their reports.

Only two prospective studies that used a control population meet contemporary standards for research criteria (34, 53). Both studies used rigorous diagnostic and outcome criteria as well as a follow-up of 1 or more years.

It is discouraging to note, therefore, that with these two exceptions there have been no completely acceptable controlled prospective studies of the use of ECT for schizophrenia even though ECT has been used as a treatment for schizophrenia for over 40 years.

Treatment of the Acute Schizophrenic Patient

Despite the scientific acceptability of only a few ECT studies, the overwhelming impression to be derived from the remaining anecdotal and clinical reports is that ECT can be an effective treatment for schizophrenia. Rates of remission or marked improvement range from 40% to 80% for immediate outcome. For patients who have been ill for more than 1 year, ECT, like other treatments, is not particularly effective. However, ECT has become firmly established as a treatment for catatonic symptoms.

If lack of readmission is taken as the criterion for treatment success, then the Camarillo comparative study strongly suggests that ECT does approximately as well as neuroleptic medication 3 to 5 years after initial treatment. On this basis neuroleptic drugs are only slightly favored over ECT. On a cost-effectiveness

basis, however, ECT is clearly inferior to drugs alone or to drugs plus psychotherapy (76).

If the treatment outcomes are approximately equal several years later, then differential production of side effects must be considered in order to establish clinical prescribing preference. ECT produces memory loss, which is usually transient. Memory loss is correlated with electrode placement, as well as with the frequency and number of seizures. The treatment of schizophrenia may require more ECT than does affective illness. Regressive ECT or maintenance ECT is particularly likely to add to the overall number of sessions a schizophrenic patient receives in a lifetime. It is possible, therefore, that a young acute schizophrenic patient who had recurrent episodes of illness may require several courses of ECT during a lifetime, thus increasing the total number of seizures. The question is, therefore, Is ECT's effect on memory cumulative over a lifetime, as radiation is for carcinogenesis?

Although a comprehensive examination of this question lies outside the scope of this review, four reports have studied the effect of ECT on memory functioning in schizophrenic patients. Perlson (77) found no intellectual deterioration as measured by psychological tests in a 27-year-old acute schizophrenic patient who received 152 sessions of ECT. However, Rabin (78) administered the Rorschach to 6 chronic schizophrenic patients who received a total of 100–134 ECT sessions and found characteristic organic personality patterns in these patients. Goldman and associates (79) administered the Bender-Gestalt test and Benton Visual Retention Test to 16 chronic schizophrenic patients who received 15–219 ECT sessions during their lifetime (mean=69.5). It had been 10–15 years since the patients' last course of ECT. The test scores of this group were compared with scores from 16 chronic schizophrenic patients who had never received ECT and who were matched for age, race, and level of education with the ECT recipients. The authors admitted that matching did not extend to the degree of pretreatment psychopathology or length of hospitalization. The ECT-treated group had significantly higher errors on the two tests than did the non-ECT group, leading the authors to conclude that cognitive impairment results from the cumulative irreversible brain damage of ECT.

Templer and associates (80) attempted to replicate this study and added the Wechsler Adult Intelligence Scale to the test battery. Twenty-two chronic schizophrenic patients who received 40–263 ECTs (mean=58.5) were compared with 22 patients who received no ECT and were matched for age, sex, race, and level of education. The authors successfully replicated original findings of inferior test performance by the ECT recipients. However, when the difference in degree of pretreatment psychopathology was controlled for, the two groups' performance on the Benton test and Wechsler scale was similar. The ECT patients' inferior performance on the Bender-Gestalt test suggested, but did not conclusively prove, permanent brain damage.

These two studies do not resolve the question of cumulative, irreversible cognitive impairment resulting from ECT use. Decreased intellectual function may have antedated the ECT, arisen from non-ECT sources, or may have resulted from inadequate oxygenation during ECT administration. The results of these studies, therefore, cannot be accepted as definitive evidence that ECT causes irreversible brain damage and/or permanent memory loss. However, the lack of unequivocal evidence of no association between ECT and memory loss casts doubt on the wisdom of using ECT with young acute schizophrenic patients, who may require a total of 50 or more treatments during their lifetime.

In deciding between ECT and neuroleptic treatment for young acute schizophrenic patients, the clinician must also consider the possibility of tardive dyskinesia in recipients of ataraxic drugs. ECT does not produce tardive dyskinesia (81). If ECT may be equivalent to drugs in long-term efficacy, would not this differential side effect favor the use of ECT? This question, like that of permanent cumulative memory loss, cannot be satisfactorily answered. The need for further research in these areas is clear.

Quality of Life After Treatment with ECT

Studies of ECT efficacy in schizophrenia that have included follow-up have almost universally used hospital readmission, ability to work, or ward behavior as the outcome criteria. Even the Camarillo study considered lack of readmission or fewest days of rehospitalization as the measure of long-term therapeutic success. This review suggests that ECT has some success in keeping patients out of the hospital. Although relapse rates are high, they seem to be correlated with duration of illness.

To my knowledge, no study to date has examined the quality of the treated patient's life after ECT. Yet there are many other factors of a schizophrenic patient's life that must be considered besides hospital readmission and work. The maintenance or development of a close human relationship, which is often impaired in schizophrenia, has not been described following ECT. There are no data on the effect of ECT on low self-esteem, lack of autonomy, autism, or pathological fear and suspiciousness, all of which contribute to a schizophrenic patient's unhappiness. Thus, it is conceivable that a patient who is successfully treated with ECT (judged by the criterion of a low rehospitalization rate) may still be leading a miserable, isolated, and fearful existence. Stated differently, there are no data to suggest that ECT qualitatively alters any aspect of schizophrenia other than the acute symptoms that require hospitalization.

The lack of data indicating the superiority of ECT

over drugs in terms of quality of life, long-term side effects, length of initial hospitalization, immediate reduction of symptoms, or cost-effectiveness suggests that ECT cannot be considered superior to neuroleptic drugs. Only the comparative follow-up data from the Camarillo study suggest that ECT may be equally efficacious to drugs; further comparative follow-up data are needed.

Treatment of Chronic Schizophrenia

Questions of cumulative memory loss after ECT as well as tardive dyskinesia after long-term high doses of neuroleptics obviously apply to the chronic schizophrenic patient. This review suggests that conventional ECT as well as regressive ECT is not therapeutic for the patient who has nonaffective schizophrenic illness of more than 2 years' duration. Although ECT may sometimes produce a diminution in symptoms, this response is usually transient and relapse rates are high. It is also possible to conclude that the likelihood of any favorable response to ECT decreases as the duration of illness increases.

Nevertheless, *some* patients who are chronically ill do occasionally show therapeutic response to ECT (82, 83). The rate of "improvement" of chronic schizophrenic patients following ECT has varied from 8% (84) and 11% (85) to nearly 50% (39). Since improvement is usually not adequately defined, no conclusions can be made regarding approximate percentages of response among chronic patients. Furthermore, relapse rates are particularly high for patients with long-standing illness. It is unlikely, therefore, that an estimate of response rate in chronic schizophrenia can be higher than 5%–10%. However, even this low rate of response would mean the discharge of a substantial number of patients from chronic institutionalization.

Since any response to ECT among chronic schizophrenic patients is likely to require more treatments than are needed for the acute schizophrenic or affectively ill patient, the risk of possible cumulative memory loss is obviously increased. Considering the overall poor response to ECT, it is unclear whether this form of treatment should ever be considered for this population. Is ECT, with its poor likelihood of response and variable incidence of memory loss, preferable to long-term neuroleptic medication, with progressive risk of tardive dyskinesia? Only future longitudinal comparative studies of the two treatments in chronic schizophrenia will conclusively answer this question.

Since a long course of treatments is to be expected if a chronic schizophrenic patient is to be treated successfully, it is particularly important to obtain informed consent from both patient and family. The explanation of the procedure must stress the risks of ECT as well as the risks of not receiving ECT, such as long-term hospitalization or tardive dyskinesia as a result of pharmacotherapy (86, 87).

CONCLUSIONS

The literature reporting the therapeutic efficacy of ECT for schizophrenia is predominantly unacceptable according to contemporary research criteria. In particular, most articles vary in the degree to which they specify criteria for the diagnosis of schizophrenia, outcome, or follow-up.

ECT has been almost completely replaced by pharmacotherapy for the treatment of acute schizophrenia and the management of chronic schizophrenia. This may be due to the poor scientific quality of the ECT studies, as well as the potential toxicity of ECT, difficulty of administration compared with drugs, and, thus, declining acceptability to patients and providers. Although a few authors still strongly advocate a primary role for ECT in the treatment of schizophrenia (19, 88–90), a secondary role for ECT is proclaimed in standard psychiatric textbooks (91–96), contemporary reviews (97–99), and in the report of the APA Task Force on Electroconvulsive Therapy (100).

As ECT has been replaced by pharmacotherapy, the length of hospital stay has declined and hospitals have unlocked their doors. This is a correlational relationship and has not been proven as cause and effect.

ECT reduces acute schizophrenic symptoms and in some cases produces a complete remission. The degree of ECT's therapeutic effect is inversely related to the duration of schizophrenic symptoms. The best results are with patients who have been ill 1 year or less. ECT is not as immediately effective as pharmacotherapy for the treatment of schizophrenic patients in the middle section of the prognostic range.

ECT is universally acclaimed as an excellent treatment for acute catatonic withdrawal or excitement.

ECT has been found useful for schizophrenic patients with depressive affect and symptoms of low self-esteem, hopelessness, and suicidal thoughts.

Although opinion is divided concerning ECT for patients with acute paranoid reactions and acute schizophrenia with paranoid features, most authors have found ECT to be beneficial.

ECT offers little hope for lasting improvement in chronic schizophrenic patients. It controls behavior and temporarily reduces symptoms, but relapse rates are very high. An occasional chronic patient will respond with lasting and significant improvement.

Opinion is divided regarding the safety and efficacy of combining ECT with neuroleptic drugs. Some reports have been enthusiastically in favor of the combination. One excellent study indicated that the combination reduces both hospital stay and readmission rate. The combination may also be employed for patients who have not responded to either treatment alone. There is no contraindication to following a course of ECT with ataraxic drugs.

Regressive ECT has produced mixed results in chronic schizophrenic patients in whom it has been

predominantly tested. It should be considered an experimental procedure at this time. Multiple monitored ECT holds promise as a new technical variation for ECT administration, but it is not conclusively superior to conventional use of ECT for schizophrenia.

There are a few suggestions that schizophrenic patients who receive many (i.e., 50) sessions of ECT during a lifetime show cumulative, irreversible brain damage as measured by paper-and-pencil tests. These data must be replicated and extended by future research.

Follow-up studies of schizophrenic patients who have been successfully treated with ECT tend to suggest a high relapse rate. In the Camarillo comparative study, patients receiving ECT and neuroleptic drugs had a nearly equivalent number of hospital readmission days. No follow-up study considers the quality of life after successful ECT. It is impossible, therefore, to state whether ECT can help schizophrenic patients live more gratifying, less lonely and frightened lives, even if they are discharged from the hospital.

Given the variable quality of reports, often contradictory findings, and abundance of different and passionate opinions regarding ECT in schizophrenia, further research is indicated.

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Effects of Lithium Carbonate on Memory and Other Cognitive Functions

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The authors studied the effects of lithium carbonate on memory and cognitive function in 16 psychiatric patients, who received lithium for 2 weeks and placebo for 2 weeks in a double-blind cross-over design. At the end of each treatment phase, subjects were administered a battery of memory and cognitive tests. As reported previously, lithium induced slowing of performance on certain of the perceptual motor tests; however, lithium did not cause memory impairment or a change in self-assessment of memory functions.

There has been considerable interest recently in effects of lithium carbonate on personality and cognitive function that might occur in conjunction with its therapeutic effects. Despite individual reports of memory complaints during lithium therapy (1–3), few comprehensive studies have formally assessed the effects of lithium on memory or other cognitive functions. Two double-blind studies of healthy volunteers, each using one memory test, found that lithium affected the ability to recall a list of words (4, 5). Moreover, psychiatric patients taking lithium for more than 1 year learned and recalled words more poorly than did patients not taking lithium (6), and 20%–30% of patients reported some degree of memory problems after 6 months of lithium treatment (7). Other studies have found that memory was unaffected by lithium when compared with either tricyclic drugs (8) or a drug-free condition (9, 10). Even after several years of treatment, lithium reportedly did not alter memory problems, according to patients' self-reports (11).

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Other cognitive tests have repeatedly demonstrated that lithium can impair performance (12–17). Judd and associates (16, 17) have suggested that lithium may affect those tests requiring speed, primarily causing a slowing of performance secondary to a slowing in the rate of cognitive processing.[1] To explore this issue more comprehensively we assessed the effects of lithium on psychiatric patients, by using a battery of six memory tests, a self-rating instrument for memory complaints, and a battery of seven cognitive and perceptual motor tests.

METHOD

Subjects and Protocol

Sixteen psychiatric inpatients (15 men, 1 woman) from the San Diego Veterans Administration Medical Center voluntarily participated in the study. The subjects averaged 41 years of age (range, 22–55 years), had completed an average of 13.9 years of education (range, 10–18 years), and were diagnosed with Research Diagnostic Criteria (18)—13 had a diagnosis of alcoholism, 2 were diagnosed bipolar I (bipolar with mania), and 1 was given a diagnosis of schizoaffective disorder, manic type. Of the 13 alcoholic patients, 4 were diagnosed as having secondary major depressive disorder, 4 had secondary minor depressive disorder, and 2 were diagnosed bipolar I. Patients with affective disorder usually were euthymic, although several were depressed or mildly hypomanic at the time of the study. Informed consent was obtained from all subjects.

All subjects were withdrawn from all psychotropic medications at least 7 days before the study began. A double-blind, counterbalanced cross-over design was then used in which each subject was given oral lithium carbonate two times a day for 2 weeks in an effort to achieve desired serum levels. Half of the subjects received placebo for the 2 weeks preceding active lithium treatment, and half received placebo for 2 weeks following lithium treatment. Serum lithium levels at the end of the active lithium treatment phase averaged

[1] L.L. Judd, L.Y. Huey, D.S. Janowsky: Lithium and its effects on cognitive processing, 1979 (unpublished manuscript).

0.94 mEq/liter (range, 0.5–1.2 mEq/liter) and 0.03 mEq/liter (range, 0–0.1 mEq/liter) at the end of the placebo phase. A battery of memory tests and a battery of cognitive and perceptual motor tests were administered on days 11–13 of each 2-week phase of the study. All 16 subjects were given the memory tests, and 11 were also given the cognitive and perceptual motor tests.

On 1 of the 2 days following testing, some of the subjects received a single 3-hour challenge drug infusion or ingestion (methylphenidate, N=3; physostigmine, N=5; ethanol, N=5); the results of these studies will be described elsewhere. Since these drugs were given after testing, the subjects were always free of all drugs other than lithium or placebo for at least 12 days before testing.

Memory Battery Tests

Tests 1–6 were administered in two equivalent forms 2 weeks after treatment with lithium or placebo. These tests are known to be sensitive to the amnesic effects of bilateral ECT (19 and unpublished observations).

Test 1: Recognition of 32 items. The subjects looked at 32 items (objects, faces, nonsense drawings, words) and later attempted to select each item from a set of three (19). Retention was tested immediately after learning and again 10–12 minutes later. The score was the number of items identified out of 32.

Test 2: Paired associate learning. The subjects saw 10 pairs of unrelated nouns on three successive trials (20). After each trial the subjects saw the first word and were asked to recall the second. The score was the number of words recalled out of 10.

Test 3: Recall of a paragraph. Immediately and 10–12 minutes after hearing a short story of five lines, the subjects tried to recall as much as they could (19). The score was the number of story segments recalled.

Test 4: Drawing a geometric figure from memory. The subjects copied the Rey-Osterrieth figure (21) or the equivalently difficult Taylor figure (22) and then 10–12 minutes later, without being forewarned, tried to reproduce it from memory. The score was the number of properly located line segments based on a maximum score of 36 points.

Test 5: Remote memory for public events. This test consisted of 23 questions about persons or events in the news from 1950 to 1976. There were 6 questions about the 1950s, 9 about the 1960s, and 8 about the 1970s. The subjects were asked a specific question about each event and then to recall as much as they could about it (20). The score was the total number of facts recalled from each decade per event.

Test 6: Detection task. Of the 16 subjects, 13 took this test, which has been described in detail previously (23). The subjects read a list of 6–17 words, 2 of which 1) were the same, 2) rhymed, or 3) came from the same semantic category. In each list 0, 3, or 6 filler words intervened between the 2 critical words. The subjects knew which type of detection they were to make (repetition, rhyme, or category) and were asked to signal when they detected the second critical word. The score was the percentage of correct detections.

Test 7: Self-rating scale for memory complaints. The subjects filled out an 18-item questionnaire that was sensitive to the memory complaints associated with ECT (24) to assess aspects of their memory abilities "now compared to before I began to feel bad and came to the hospital." Ratings were made on a scale from −4 (worse than ever before) to +4 (better than ever before). A list of all test items has appeared elsewhere (24).

Cognitive and Perceptual Motor Battery Tests

All tests were administered in two equivalent forms 2 weeks after treatment with lithium or placebo. Additional information and original sources for all tests have been presented elsewhere (16, 25).

Test 1: Porteus maze. The subjects were presented a labyrinth maze printed on paper and instructed to start in the center and trace a path to the outside edge without crossing any lines or lifting the pencil from the page. The score was the time required for completion, the number of wrong turns, and the number of times that lines were crossed.

Test 2: Serial sevens. The subjects were instructed to count backwards by sevens from a given number between 500 and 506. The score was the number of correct responses and the percent of errors in a 2-minute period.

Test 3: Number learning task. The subjects were given seven trials to learn correctly a 14-digit number, and seven more trials to learn a second number. The score for each learning test was the total number of digits correctly recalled in seven trials.

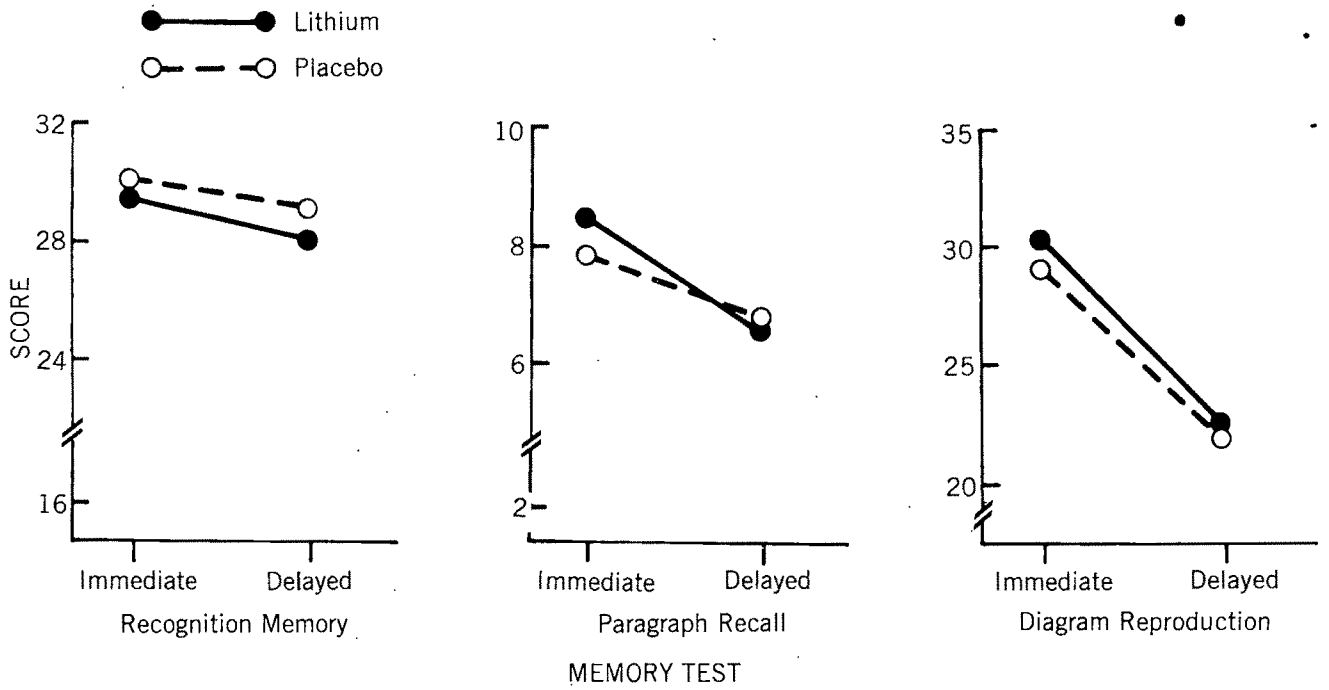
Test 4: Speed of closure. The subjects were instructed to scan 22 rows of 46 letters for 2 minutes and then to circle any 4-letter word. The score was the number of letters scanned, the number of correct words identified per 100 letters scanned, the percent of correct words not identified (omission errors), and the percent of "words" incorrectly identified (commission errors).

Test 5: Minnesota clerical. The subjects scanned a list of number pairs for 8 minutes. Some of the pairs were identical and others were not, and the subjects were asked to check the identical pairs. The score was the number of pairs scanned, the percent of identical pairs not checked (omission errors), and the percent of nonidentical pairs checked (commission errors).

Test 6: Digit symbol substitution. This paper-and-pencil timed test, selected from the Wechsler Adult Intelligence Scale, required subjects to convert a sequence of symbols into a sequence of digits according to a code printed at the top of the page. The score was based on the number of correct responses in 90 seconds.

Test 7: Trail-making A and B. These were two pa-

FIGURE 1
Effects of Lithium and Placebo on Recognition Memory (Test 1), Paragraph Recall (Test 3), and Diagram Reproduction (Test 4) Memory Tests for 16 Psychiatric Inpatients



per-and-pencil timed tests from the Halstead-Reitan Battery, in which subjects connected as quickly as possible a sequence of numbers, or numbers and letters, arranged out of order on a page. The score was the amount of time required for completion.

RESULTS

The data for each test were submitted to an analysis of variance with one repeated factor (drug condition: lithium or placebo) and one independent factor (drug order: lithium-placebo or placebo-lithium). There were no significant effects of lithium on any of the memory tests (see figure 1 for the results of tests 1, 3, and 4). In addition, for paired associate learning (test 2), scores improved from an average of 4.6 correct on the first trial to 8.6 correct on the third trial, and there was no effect of drug on learning ($F=2.4$, $p>.10$). On test 5 (remote memory) subjects recalled an average of 2.0 facts about each event from the period 1950 to 1976, and there was also no effect of drug condition on recall ($F=1.0$, $p>.10$). On test 6 (short-term detection), subjects were best at detecting repetitions of words (91% correct), worst at detecting rhymes (63%), and intermediate at detecting words belonging to the same semantic category (73% correct). In addition, performance declined from 96% correct, when the two words to be detected were read in immediate succession, to 57% correct when six words intervened between the critical words. Performance on this task was almost identical under lithium and placebo conditions, and the average difference in performance across the 9 experimental conditions (3 types of detection×3 delays) was 3.6%. Order effects emerged for three of the six tests (tests 2, 3, and 6); i.e., subjects receiving lithium-placebo performed differently than those receiving placebo-lithium. On two occasions the lithium-placebo order yielded better performance (tests 2 and 3), and on one occasion the placebo-lithium test yielded better performance (test 6).

Self-ratings for memory functions during the lithium and placebo phases of the study (test 7) showed that the subjects responded somewhat differently to the various test items ($p<.05$), but on each of 18 items they rated their memories virtually the same when they were taking lithium as when they were taking placebo ($F<1.0$, $p>.10$).

Whereas lithium did not impair memory test scores for all 16 subjects together, it seemed possible that there might be a measurable relationship between serum levels of lithium and memory test scores. Accordingly, we computed correlations between serum levels and scores on each memory test. These correlations were significant for tests 1 and 4 ($r=-.61$, $p<.02$), indicating that subjects having higher lithium levels performed more poorly than subjects having lower lithium levels. For test 6, which measured detection in short-term memory, the correlation was positive ($r=.54$, $p<.05$), indicating that higher lithium levels were related to better performance. We also computed a composite memory score for tests 1–5 by ranking performance on each test and then summing the ranks for each subject across the five tests: a low composite

TABLE 1
Mean Cognitive and Perceptual Motor Test Scores of 11 Psychiatric Inpatients Receiving Lithium or Placebo

Test	Mean Score	
	Placebo	Lithium
1 (porteus maze)		
Time (seconds)	68.2	106.4
Cognitive errors	1.0	1.7
Motor errors	3.3	2.3
2 (serial sevens)		
Number completed	32.1	29.0[a]
Errors (%)	9.6	8.9
3 (number learning task)		
1st test	45.9	57.8
2nd test	51.0	43.9
4 (speed of closure)		
Number of letters scanned	334.6	320.6
Number of words correct per 100 scanned	5.2	5.1
Omission errors (%)	19.9	24.8[a]
Commission errors (%)	4.5	0.0[b]
5 (Minnesota clerical)		
Number completed	131.6	116.6[b]
Omission errors (%)	1.5	1.5
Commission errors (%)	2.4	2.1
6 (digit symbol substitution)		
Number correct	56.5	49.8[c]
7 (Trail-making)		
A	28.2	29.4
B	72.0	76.6

[a]p<.10.
[b]p<.05.
[c]p<.01.

memory score indicated good performance and a high score indicated poor performance. The correlation between lithium serum levels and the composite memory score was significant (r=.66, p<.01), indicating that higher serum levels were associated with poorer performance.

The results for the cognitive and perceptual motor battery are shown in table 1. Some drug order and practice effects appeared, but these factors were not significant on tests in which significant effects of drug treatment were found. The subjects performed somewhat worse during the lithium phase than during the placebo phase on nearly every test in the battery. On test 4 the subjects performed significantly better during lithium than during placebo as measured by one of the error scores (commission, p<.05). For two of the seven tests (tests 5 and 6) the subjects performed significantly worse during lithium than during placebo; the subjects taking lithium completed fewer items on the Minnesota clerical test (p<.05) and copied fewer symbols in the Digit symbol substitution test (p<.01) than when they were taking placebo. Since accuracy was not affected by lithium on any test, the effect of lithium appeared to reflect a slowing of performance rate.

We next computed correlations between serum levels of lithium and scores on each performance test. The relationship between serum levels and performance on test 7 (Trails B) was significant (r=.75, p<.01). We also computed a composite performance score for each subject by ranking performance on each test and then summing the ranks for each subject across the seven tests. There was no measurable relationship between lithium serum levels and the composite performance score (r=.19, p>.3).

Mood ratings as assessed by the Profile of Mood States (26) were available for 13 of the 16 subjects who took the memory tests and 9 of the 11 subjects who took the cognitive and perceptual motor battery. The POMS was administered on days 13 or 14 of each 2-week phase of the study. Correlations were computed between each of the eight scales of the POMS and the composite memory test score or performance score. None of these correlations was significant (p>.2).

DISCUSSION

Taken together, the results indicate that nearly 2 weeks of lithium treatment at an average dose of 0.94 mEq/liter did not significantly affect performance on formal tests of learning and memory. The results obtained with a newly developed self-rating instrument also indicated that lithium did not cause subjects to experience their memory as impaired. Nevertheless, the doses we studied exerted measurable effects on the central nervous system on certain tests requiring speed; this is consistent with results previously reported for such tests (12, 14–17). Thus, lithium appeared to cause slowing of performance in the absence of impairment of learning and memory. Since memory defects are commonly an early sign of diffuse cortical dysfunction in neurological disease, the finding that memory was not measurably affected by lithium suggests that in the doses we used lithium's effects on higher cognitive function are on the average rather unremarkable.

It is possible, of course, that measurable effects of lithium on memory would have appeared if treatment had been continued longer than 2 weeks. In one study (7), some memory complaints developed after 6 months to 2 years, in 20%–30% of subjects on maintenance lithium therapy. In another study (6), patients taking lithium for 1–4 years scored more poorly on a memory test than a group of patients not taking lithium; however, as these authors noted, it is possible that the findings reflected differences in group composition; a within-subjects experimental design is needed to settle this issue unambiguously. It also seems possible that effects on memory would have emerged in our study if lithium had been administered in higher doses, and our finding of a negative correlation between serum levels of lithium and memory test scores is consistent with this suggestion. Thus, the higher doses of lithium that we used may have tended to impair performance, although on the average the doses we used caused no measurable effect. The doses we used (0.5–1.2 mEq/liter) are those typically used for

maintenance lithium therapy but do not extend across the full range of doses that might be used in acute lithium treatment (up to 1.5 mEq/liter). Judd's informal clinical observations have also suggested that memory complaints are more frequent at higher doses of lithium (serum levels above 1.2 mEq/liter).

It is also important to note that our findings are based on a heterogeneous group of psychiatric patients and not on a group of patients with affective disorder selected because of their suitability for lithium therapy or on a group of healthy, nonhospitalized volunteers. In two previous studies of healthy volunteers (4, 5), 1–2 weeks of lithium treatment affected performance on a memory test that assessed recall of a word list after a delay. However, one of these studies (5) involved only 6 subjects, and it was not noted whether the difference between lithium and placebo was statistically significant. The other study (4) involved 12 subjects, and lithium's effect was to impair recall of words learned 2 weeks previously; lithium did not affect initial learning or recall after a 2-hour delay.

In summary, in this study we used six formal memory tests proven sensitive to the amnesic effects of ECT (19, 27), and lithium had no overall effect on memory; on a seventh testing instrument sensitive to the memory complaints associated with ECT (24), lithium did not cause a subjective sense of memory impairment. It therefore seems reasonable to suppose that lithium's effect on memory, at doses of 0.5–1.2 mEq/liter, is rather unremarkable. Since memory test performance tended to correlate inversely with serum lithium levels, higher doses of lithium (i.e., above 1.2 mEq/liter) might have produced greater effects on memory than we observed. Although further work is needed to evaluate fully the effects of lithium on cognitive function, our results, together with previous results obtained under the same experimental conditions (16–17), are consistent with the view that lithium affects the rate of performance on cognitive and perceptual motor tasks.

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Couples' Reactions to Male Infertility and Donor Insemination

BY DAVID M. BERGER, M.D.

The author interviewed 16 couples after the husband had been diagnosed as infertile. Eleven husbands experienced a period of impotency. Fourteen of the women experienced anger toward the husband, psychiatric symptoms, and/or dreams about their concern for the husband, a wish to be rid of him, and guilt over this wish. Ten couples had decided to pursue donor insemination; 6 had delayed 1¾ to 4 years after the diagnosis of infertility and had a better adjustment than 3 of the 4 couples who did not. The author believes that the total secrecy involved in donor insemination inhibits the working through of conflicts about infertility and donor insemination itself.

In order to study conflicts and behavior patterns in couples in whom the male partners had been discovered to be infertile, I interviewed 16 married couples after the diagnosis of azoospermia or severe oligospermia had been made in the reproductive biology clinic of a 600-bed urban general hospital. Excluded were couples who had been aware of the husband's infertility before marriage, couples in whom the woman had been found to have physical pathology that could have contributed to the infertility, and couples in whom one of the partners had severe psychopathology (e.g., schizophrenia or manic-depressive illness). The women's ages ranged from 21 to 34 years. The men's ages ranged from 21 to 38 years. At the time of the interview 10 of the couples had already decided to undertake donor insemination, 2 couples had decided against it, and 4 couples were undecided. Because the clinic is a special unit that receives referrals from other physicians, for only 2 couples was the diagnosis of male infertility a novel finding. The couples had known about the infertility for 8 months to 4 years before the interview.

The interview focused on a number of questions: What was each partner's reaction to the diagnosis of the husband's infertility? Could changes in feeling, thinking, or behavior be detected in the individual partners or in the relationship? What conflicts could be inferred from these changes? Was there any pattern to the couple's choosing donor insemination, and how did they feel about keeping it secret? The partners were encouraged to talk freely about whatever they felt to be important. I interposed questions to fit the subject at hand and requested dream reports of every individual.

FINDINGS

The Men

Only 3 of the 16 men were symptom free. Ten reported a period of impotence that had lasted 1 to 3 months (mean=1¼ months) after the discovery that they were azoospermic or oligospermic. Before this discovery, the frequency of their sexual activity had been 1 to 3 times per week. The onset was within 1 week of being informed of the diagnosis. Another subject was impotent for at least 4 months (the impotence had not improved at the time of the interview). This subject felt that the impotence was precipitated by "an attack of kidney stones" 3 years after the discovery of oligospermia. Three of the 11 men with impotence also admitted to depression, 1 had an exacerbation of peptic ulcer symptoms, 1 man began an affair (which he felt cured his impotence) within a month of discovering he was azoospermic, and 1 man suffered whiplash in a car accident 1 month after he was informed of his azoospermia and was incapacitated for 6 months. Two of the 16 men reported no change in sexual pattern or mood, but the level of their sexual activity was low to begin with (frequency of intercourse was less than once per month), and only 3 men, whose sexual activity level had been satisfactory, reported no change in sexual pattern or mood. These findings are striking when compared to the incidence of cessation of marital intercourse in a general population (1).

Only in the couple in whom the man had embarked on an affair shortly after the diagnosis was made did the couple report a marked change in attitude toward the wife. Some of the wives mentioned that their husbands appeared more withdrawn than in the past.

In addition to dreams that occurred after the discovery of infertility, I requested specifically dreams from

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the night before the interview, on the premise that anticipation of the interview might stir up conflicts related to the infertility. Only 5 of the 16 men were able to report dreams. All were from the night before the interview. The only unvarying finding was that heterosexual encounters were absent in all 5 dreams. This is in accord with the reported withdrawnness of the men after the discovery of infertility.

The Women

None of the 16 women reported a decrease in their frequency of sexual activity, and 3 reported an increase within 2 months of discovering their husband's infertility. Six of the 16 couples reported that the wives were significantly angrier toward the husbands shortly after the diagnosis was made. In 1 couple this led to a temporary separation. In 2 other couples this changed attitude led to the wife's having an affair in which no contraceptives were used. The other 3 couples stated that their marriage had been "on the rocks" for several months. Despite the fact that the marital difficulties occurred within 3 months of the husband's diagnosis, not one of the couples had linked the difficulties with the infertility.

One woman developed a fear that she would give birth to a defective child. The phobia was accompanied by rage and envy directed at others "who did have children but were terrible parents."

The dreams these women reported were remarkably consistent. One of the 16 women volunteered that within a week of first learning of her husband's infertility she had a dream in which she attempted to protect her husband from attack by criminals. Eight other women reported that the night before the interview they had had dreams in which the husband fell ill, committed suicide, or was physically or verbally assaulted. In only 1 of these 9 reports was the wife the aggressor; in the others her role was protector and comforter.

Another woman reported the following dream: "I was pregnant and very happy about it. Then I discovered that I had gained too much weight and would die." In the interview this woman stated that if the couple's infertility were to become known to others she would prefer people to believe that it was her problem, not her husband's. Moreover, since the discovery of the infertility she had become increasingly preoccupied with her husband's weight; her husband was obese and she worried that his health was declining.

Ten of the 16 women reported dreams with a similar content. None of them had experienced such dreams in the past. Three themes appeared to be incorporated in these dreams: 1) the woman felt bad (perhaps guilty) about the husband's infertility, 2) she wished to be rid of her husband, and 3) she felt guilty about her wish to get rid of him. Two women reported dreams in which these themes were not evident, and 4 women could not recall any dreams. The dream reports were compared to those of a control group of 10 women (ages 20 to 31 years) whose infertility workup revealed no evidence of physical pathology in either spouse: only 1 dream contained aggression directed at the husband. Compared to studies of dream reports gathered from the general population (2, 3), the dream reports of the women in this study contain a higher incidence of violence and a much higher incidence of violence directed at the spouse. The finding that more women than men report dreams is consistent with the findings of surveys of the general population.

In summary, after the revelation that their husbands were infertile 4 women developed symptoms (phobia, rage at the husband that led to marital difficulties) but no significant dream findings, 2 women had symptoms as well as significant dream findings, 8 women had significant dream findings but no symptoms, and 2 women had no symptoms and no significant dream findings.

The Decision to Pursue Donor Insemination

At the time of the interview 10 of the 16 couples had decided to undertake donor insemination, 2 had decided against it for religious reasons, and 4 were undecided or preoccupied with marital difficulties.

Six of the 10 couples who had decided in favor of pursuing donor insemination had delayed before arriving at their decision; they stated that they had been undecided for several months after donor insemination was first discussed with them. For these 6 couples the time between the discovery of the infertility and the interview ranged from 1¾ years to 4 years (mean=2¼ years). Two couples denied any conscious intent to delay their decision as long as they had (3 years) but for some time had been considered "problem patients" at the clinic because they were difficult to contact and would cancel appointments. Two couples felt that job difficulties contributed to the delay. Only 2 couples attributed the delay entirely to indecision and the husband's requiring time "to get over feeling bad." These 6 couples decided on donor insemination without further requests for psychiatric intervention. This pattern of delay is suggested in the report of Nijs and Rouffa (4) and supports Beck's contention (5) that deliberation (although it may be unconscious) precedes the decision to undertake donor insemination.

Four of the 10 couples who had decided to pursue donor insemination reported that they had been relatively certain about their decision from the start (although this certainty was voiced primarily by the wife). The time between their discovery of the husband's infertility and the interview was 18 months or less. To a large extent this time lapse could be accounted for by waiting periods entailed in referral to the clinic, consultations, and completion of the workup for both partners. How these 4 couples fared is revealing. One couple decided to adopt a child after an incomplete course of donor insemination. Another couple became disenchanted with our clinic, although before their departure the husband came to see me be-

cause he and his wife were finding intercourse painful. The third couple, in which the wife had developed a phobia as well as diffuse rage, was the only couple in this group to receive the recommendation that individual psychotherapy for the wife was necessary before proceeding with donor insemination. The fourth couple had known that the husband had had surgery as a child for an undescended testicle and that it had been crushed in the process, so that the man's infertility did not come as a surprise.

These findings suggest that a period of delay before deciding to pursue donor insemination is associated with a better adjustment to it. That another couple, who chose not to pursue donor insemination, had transferred to our clinic because they felt "rushed into donor insemination" by their previous physician supports this observation.

How did the couples feel about the concealment that accompanied donor insemination? Nine of the 10 couples who had chosen to pursue donor insemination felt that the secrecy presented no problem. One couple felt that it would be burdensome, that it would isolate them from others. This couple discontinued treatment shortly after its initiation and decided to adopt a child. When asked what benefits might derive from the secrecy, most couples responded with vague statements: "It's no one's business," "No one has to know." On further questioning 7 of the 10 couples stated that the secrecy would somehow benefit the child ("We don't want the child to feel different").

Three couples stated that "their" infertility would be kept from others. Repeated questioning was required before any of the 10 couples would admit that it was the husband's infertility that the secrecy might serve to hide. The tendency was to cling to phrases such as "our infertility." Interestingly, this specific admission was usually voiced by the women. It was also the women who seemed to react most critically to the possibility of sharing the secret with others as well as with the child who would result from donor insemination. These findings are impressionistic and do not lend themselves to clear-cut conclusions. However, they are consistent with the findings of Farris and Garrison (6) that, in frequency of response to the question of why couples chose donor insemination over adoption, keeping the husband's infertility hidden did not rank very highly. One might infer from these findings that the partners had resolved their conflictual feelings about the man's infertility. However, the apparent reluctance to admit that one obvious purpose of the secrecy was to conceal the male partner's infertility, coupled with evidence from the women's dream reports, suggests that there still remained unresolved feelings and that the concealment of donor insemination permitted displacement to take place, in most cases a shift in concern toward the child who would result from donor insemination.

DISCUSSION

The findings describe a specific common conflict pattern in couples after the diagnosis of male infertility: impotence, depressive mood and/or depressive equivalents in the husband, and hostility and guilt in the wife. In only 2 of the 16 couples did neither partner reveal signs or symptoms that would provide evidence for this pattern. The pattern is consistent with the findings of Gerstel (7) and Watters and Sousa-Poza (8) and suggests that the impotence deserves to be viewed as an interactional problem and not solely the man's problem.

The finding that a period of delay is associated with a better adjustment than is a rush to pursue donor insemination supports the views of Gerstel (7) and Watters and Sousa-Poza (8) that inability to mourn the infertility before turning to donor insemination contributed to their clients' later difficulties. The findings suggest that the decision to pursue donor insemination involves two problem-solving stages: 1) coming to terms with the husband's infertility, and 2) confronting the problems of donor insemination itself, and that the total secrecy that surrounds donor insemination may interfere with the successful progression through this two-stage process and may contribute to a blurring of the two separate tasks. Difficulties ensue, not because there is conflict (as Gerstel [7] and Watters and Sousa-Poza [8] suggest), but because the conflict has not been worked through and resolved. The implication for clinical management is that serious discussion relating to donor insemination might best be postponed for 3 to 4 months after completion of the infertility workup.

This study offers tentative support for the hypothesis that the anticipation of some openness, at least between parents and the future child, as opposed to total secrecy, might prove psychologically beneficial. Certainly this issue cannot be overlooked by physicians and deserves further study.

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The *DSM-III* Personality Disorders Section: A Commentary

BY ALLEN FRANCES, M.D.

The author reviews the DSM-III *section on personality disorders, discusses several of its more controversial diagnoses, and suggests some possible alternatives. He attributes the continued low reliability of personality diagnoses, compared with the other major sections of* DSM-III, *to two inherent obstacles: the lack of clear boundaries demarcating the personality disorders from normality and from one another, and the confounding influence of state and role factors. Nonetheless, the* DSM-III *multiaxial system highlights the importance of personality diagnosis and, together with the provision of clearly specified diagnostic criteria, achieves a considerably improved reliability compared with previous nomenclatures.*

The multiaxial diagnostic system introduced by *DSM-III* serves to highlight the importance of the personality disorders and to recognize their frequent coexistence with, and possible contribution to, the other psychiatric disorders. *DSM-III* provides separate axes—Axis I for all other clinical syndromes and Axis II for personality disorders—to ensure that the personality disorders not be overlooked "when attention is directed to the usually more florid Axis I disorder" (1). This concern arises from accumulating evidence that the quality and quantity of preexisting personality disturbance may indeed influence the predisposition, manifestation, course, and response to treatment of various Axis I conditions (2–7). The separation of Axes I and II has contributed to the increased diagnostic reliability achieved by the whole system. Evaluators are now encouraged to label both the clinical syndrome and the personality disorder (when both are present), rather than being forced to arbitrarily and unreliably make a choice between them.

This paper discusses some of the obstacles to the establishment of a reliable nomenclature of personality diagnosis. It also provides a critical review of the *DSM-III* section on personality disorders, particularly several of its more controversial diagnoses. As a member of the Advisory Committee on Personality Disorders to the APA Task Force on Nomenclature and Statistics, I acknowledge considerable bias in my presentation. Undoubtedly, I have blind spots that favor some of the choices made in *DSM-III* and may also be taking this last opportunity to argue against choices that I disliked. No one who worked on *DSM-III* had the quixotic goal of achieving any lasting or perfect solutions. It is my hope that a continued discussion of the controversies and decisions that resulted in *DSM-III* will encourage the research and debate required for an improved *DSM-IV*.

LIMITATIONS TO RELIABILITY

Previous clinical systems of personality diagnosis were plagued by very low reliability (8). Although *DSM-III* has improved on this considerably, the personality disorders as a group still attain the lowest reliability of any major category in the classification (9). There are at least two inherent reasons for this: 1) most of the personality disorders are probably no more than the severe variants of normally occurring personality traits that are distributed continuously and without clear boundaries to indicate pathology, and 2) personality assessment is inevitably confounded by intercurrent state and role factors. I will discuss these obstacles in turn.

In order to classify disorders into separate types, one ideally requires that they be distinct and mutually exclusive and that the number of cases falling at the boundary between disorders be relatively few (10). Although the organic, schizophrenic, and affective disorders all retain their fair share of fuzziness, these categories are to some extent discontinuous from one another and discrete from normality. In comparison, the various personality disorders are not at all clearly distinct from normal functioning or from each other (or even, as is discussed below, from Axis I conditions). *DSM-III* states, "It is only when personality traits are inflexible and maladaptive and cause either significant impairment in social or occupational functioning or subjective distress that they constitute personality disorders." Although in most instances *DSM-III* attempts to carefully and operationally define its criteria, it provides no definitions or quantifications for the

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terms "inflexible," "maladaptive," or "significant." It would indeed be difficult to make these concepts operational. Since each clinician is left to his or her own judgment in deciding whether a particular degree of personality impairment should be regarded as disorder or merely a normal trait, this decision is made with only fair reliability.

In addition, the various personality disorders are, to some extent, correlated and continuous with each other (11). Some of the diagnostic criteria appear, in more or less similar language, under more than one category. *DSM-III* does attempt to soften the potential rigidity and inapplicability of the category system to personality diagnosis by allowing for multiple and mixed diagnoses. Although this undoubtedly constitutes the only possible current compromise, there is another method of personality diagnosis that may receive increasing attention in the future. The alternative to the *DSM-III* categorical classification would be the use of a dimensional system. This method has been extensively studied by psychologists. Among the best-known factor scales are those of Eysenck (12), Cattell (13), Leary (14), and McLemore and Benjamin (15); the MMPI is a widely used nonfactorial scale.

A dimensional system avoids many problems of the categorical approach. It is, at least theoretically, the more applicable method for describing normally distributed traits that can be quantified and measured. As this applies to *DSM-III*, the patient might be rated (perhaps on a scale of 1 to 10) for each personality characteristic rather than being diagnosed within one or another distinct personality type. This procedure might greatly improve the description of patients who are on the boundary between normality and disorder or between one and another personality disorder. The dimensional system successfully renders continuities and saves information that is lost in the forced choice of just one or the other category. It also lessens the reification of categories and the accompanying halo effect on clinicians, who may perceive patients as neatly fitting into one or another category while perhaps ignoring those traits outside the anticipated cluster.

There are substantial reasons why *DSM-III* continues to apply the more traditional categorical approach in the face of these undeniable advantages to personality diagnosis of a dimensional system. Kendell (10) has observed that clinicians naturally think in terms of categories even if research scientists tend to prefer dimensions. Dimensions may provide more information than can be conveniently used and often seem too complicated for routine clinical discourse. The creation of categories serves to reduce information overload, abstract what is most crucial, and convey a more vivid, if less accurate, picture of the patient. It is good to realize that the evolution of diagnostic nosologies must be one of the most conservative of clinical endeavors; radical departures tend to be ignored. It would certainly be premature to demand widespread conversion to a dimensional system at this time. However, it is possible that with the future widespread availability of computers, psychiatry will prefer a dimensional system, at least for personality diagnosis.

The second inherent limitation to the reliability of the diagnosis of personality disorders results from the difficulty in distinguishing and untangling trait from state and role factors. The *DSM-III* definition of personality disorder does address and, in theory, solves these problems. Personality traits are defined as enduring patterns exhibited in a wide range of activities, recognizable by adolescence or earlier, and continuing throughout adult life. In actual practice, however, it is often quite impossible on cross-sectional evaluation to decide to what extent the current behavior and clinical picture represent an enduring and lifelong pattern or to what extent they are being strongly influenced by the patient's current state and/or role expectations. The interaction of trait and state is inherently confounding and difficult to analyze. It may be impossible to eliminate completely the possible influence of Axis I conditions in the assessment of personality disorders. Many studies have ignored the fact that the patient's current mood inevitably tends to contaminate the evaluation of his or her long-standing traits. The Eysenck neuroticism scale, supposedly a personality measure, has been found to be very sensitive to mood changes and may be more a measure of depression than of personality impairment (16). Only repeated evaluations and careful history taking (perhaps also using relatives) can minimize the contamination of current state factors. The multiaxial system is a useful advance in its recognition of the possible mutual influence of personality and Axis I disorders and its provision of independent ratings for each.

The trait-role overlap is almost as difficult to disentangle. Within an overall personality pattern, each individual is certainly capable of a range of behaviors that are differentially selected depending on the contingencies, expectations, or role assignments of the particular current social environment. Personality disorder is by definition a pervasive and inflexible set of maladaptive behaviors that occur widely and not exclusively in response to a specific set of circumstances. A pathological reaction that is elicited by only one particular and unusual stimulus (e.g., an Army recruit's disobedience in boot camp) would be defined as an adjustment, not a personality, disorder. In order to qualify for the often carelessly applied diagnosis of passive-aggressive personality disorder, the behavior in question would have to be general, long-standing, maladaptive, and not merely an isolated role response to one difficult situation. In any given evaluation of personality, the consultant must judge whether the patient's behavior is deeply ingrained or whether it is merely a particular reaction to the current environment, including, incidentally, the often unfamiliar and stressful environment of the psychiatric evaluation.

A CRITICAL REVIEW

The following remarks constitute a critical review of the *DSM-III* section on personality disorders. The criticisms must be understood in the general context of my overall enthusiastic approval of this section. For several years I have had opportunities to use it for clinical, teaching, and research purposes and have found it to be clinically and heuristically helpful and a vast improvement over *DSM-II*. The following discussion assumes and probably requires some familiarity with the *DSM-III* section on personality disorders, which might profitably be read simultaneously.

Additions and Deletions

Four of the *DSM-II* personality disorders have either been eliminated from the *DSM-III* classification or transferred to other of its sections. The diagnosis of asthenic personality had been rarely used anyway and was probably indistinguishable from chronic mild depression. The diagnosis of inadequate personality dealt with the patient's functional level rather than describing a distinct behavior pattern. These two diagnoses were retired, probably without mourners. The diagnosis of explosive disorder was transferred out of the personality disorders section because its *DSM-III* definition requires an intermittent rather than a continuous course. There was some support for including a diagnosis of impulsive personality for patients whose outbursts are consistent with deeply ingrained overall personality functioning. As a compromise, impulsive personality has been listed as one of the possible other personality disorders but without specific criteria. The transfer of cyclothymic disorder to the affective section will be discussed in more detail below. The rationale for most of the five new categories of personality disorders—the schizotypal, borderline, narcissistic, avoidant, and dependent—will also be discussed.

Clusters

There is a comment in the introduction to the section that the personality disorders are grouped into 3 clusters: 1) the paranoid, schizoid, and schizotypal disorders are characterized by odd or eccentric behavior; 2) the histrionic, narcissistic, antisocial, and borderline disorders are characterized by dramatic, emotional, or erratic behavior; and 3) the avoidant, dependent, compulsive, or passive-aggressive disorders are characterized by anxious or fearful behavior. The reader should understand that this clustering does not arise from any particular evidence. In fact, most of the criteria clusters that define the 11 defined personality disorders are based only on clinical intuition and impression that has yet to be confirmed by factor analysis or other forms of validation (i.e., follow-up, family, psychological test, or response to treatment studies). Allport and Odbert (17) gathered 4,500 personality adjectives that were listed in the dictionary. There is no particular reason to believe that the 11 personality types described in *DSM-III* are the most appropriate number or grouping of personality traits or that the criteria best cluster as they now stand. On a more optimistic note, there is some evidence that the factor analyses of items derived from self-report questionnaires turn up approximately the same syndromes as had been derived intuitively by clinicians (18, 19).

Handling of Spectrum Disorders

There is some inconsistency and an overcompensation for past historical errors in the classification of the schizophrenic and affective spectrum disorders. In order to keep psychotic conditions together and to narrow the diagnosis of schizophrenia, which has been applied overinclusively in the United States (20), *DSM-III* places the presumed nonpsychotic spectrum disorder (schizotypal personality) within the personality disorders section. This is in itself a useful advance, but, simultaneous to the taming of schizophrenia, *DSM-III* may have become paradoxically overinclusive with the affective disorders. The presumed affective spectrum disorders—cyclothymic and dysthymic—do not join the schizophrenia spectrum disorder in the personality section but instead are listed with the major affective disorders. This placement of cyclothymic disorder is agreeable enough because there is fairly compelling evidence of its close relationship to the major affective disorders (21). The inclusion of dysthymic disorder (chronic depressive disorder or depressive neurosis) is a much more controversial and worrisome decision. Dysthymic disorder probably includes an extremely heterogeneous group of patients. They may have vegetative symptoms indicative of an affective disorder; psychological conflicts that make them pessimistic, self-defeating, and unhappy; a chronically difficult life situation; or various mixtures of the above.

DSM-III would have been more consistent had it selected one way to handle both the schizophrenic and the affective spectrum disorders. If dysthymic disorder must be included in the affective section, it should have required as an essential criterion the presence of vegetative symptoms; this is not the case now. Nonvegetative chronic depression should have appeared separately under one or another title (I would have preferred depressive or masochistic personality) in the personality disorders section.

New Categories of Schizotypal and Borderline Personality Disorders

The introduction of two new categories, the schizotypal and borderline personality disorders, represents a major clarification of what has been a very confusing classification. The separation of these two different, but at times overlapping, types of "borderline" disorders should improve the level of clinical discourse and research. The manner in which the criteria

were derived and tested has been well described (11) and need not be reviewed here.

Perpetuation of the Term "Borderline Personality Disorder"

The perpetuation of this term caused considerable controversy and resulted only from the lack of wide acceptance of any substitute term. Many feel that the concept of borderline has been so variously and loosely applied that it has lost all precise meaning and shape. It is no longer clear what it "borders" on, especially since the border to schizophrenia is now occupied by the schizotypal personality disorder and the border to affective illness by the cyclothymic and dysthmic disorders. There are, in fact, three additional ways that the term "borderline personality" has been used: 1) descriptively, to define by symptomatic similarities a particular syndrome of unstable personality (this is the sense of the *DSM-III* usage), 2) psychodynamically, to define an inferred level of personality organization somewhere between the psychotic and neurotic levels of integration and with characteristic deficits in psychic structure (which may coexist with any of the descriptive personality types), and 3) as a rating of severity of impairment indicating the presence of any marked character disorder (regardless of its particular descriptive or structural characteristics).

It is yet to be determined to what extent these three uses correlate with one another in the groups they define. Since the diagnosis borderline personality has attained such an unusual popularity, it is probably helpful to have a definite set of criteria on which to base a more precise discussion and research investigation. The major dispute regarding the criteria that were chosen concerned the advisability of an additional essential feature—that the patient must have displayed transient, self-limited deficits in reality testing. This suggestion would have considerably narrowed the borderline personality group and rendered it more homogeneous. It was voted down pending the accumulation of data on the relationship between transient psychotic episodes and the rest of the syndrome of instability.

Antisocial Personality Disorder

Surprisingly, this was the most controversial of all the personality disorders. The *DSM-III* diagnostic criteria specifying antisocial personality are indeed clear and reliable and have been the most carefully studied (22), but they may have missed the most important clinical point. Using criteria comparable to those in *DSM-III*, approximately 80% of all criminals are diagnosed as antisocial (23). Many investigators have suggested the inclusion of additional items that might indicate the individual's capacity for loyalty to others, guilt, anticipatory anxiety, and learning from past experiences. Some distinction between antisocial and dyssocial patterns of behavior was included in both *DSM-I* and *DSM-II* and is reflected in the current definition of childhood conduct disorders in *DSM-III* (24). These are divided into the socialized and undersocialized subtypes—a separation that seems to have some usefulness in predicting adult behavior. It has also been shown that if criminals are evaluated with more traditional and less inclusive criteria, only 30% are diagnosed as having antisocial personalities (25). For clinicians who work in prisons, it would seem to be more useful to have criteria that distinguish those criminals who are capable of loyalty, anxiety, and guilt from those who are not, since the behavior and management of each group are likely to be quite different. There was also considerable concern that the *DSM-III* criteria would be too easily and universally attained by individuals growing up in rough and deprived areas.

Schizoid and Avoidant Personality Disorders

The distinction between these two disorders is a new one. Individuals with these disorders might closely resemble each other, although the individual with an avoidant personality is much more likely to have achieved at least one close relationship. By definition, the major difference between the categories resides in the patient's motivation for relative social isolation. The patient with an avoidant personality craves closeness but avoids it because of fears of criticism and rejection. The patient with a schizoid personality is beyond desire. Some people have argued that this judgment cannot be made reliably and depends on inference. They suggest that the two conditions be combined. This may become necessary, but my own clinical impression is that it is well worth trying to distinguish the two disorders. The avoidant and schizoid patients are likely to be markedly different in their family history, predisposition to schizophrenia, ability to form therapeutic or other relationships, and need for the response to treatment.

There was also a dispute involving the use of the word "schizoid." In earlier drafts of *DSM-III*, it was replaced by the term "introverted," but this was quite upsetting to Jungian analysts and factor psychologists, for whom "introverted" does not imply the presence of disorder and is, rather, a normal type. Some people felt that the motivation beneath the avoidant personality disorder should have been defined more broadly to include fears about shame, humiliation, losing control, and making a fool of oneself. The patient with an avoidant personality is often likely to meet the criteria for the dependent personality; once a relationship is formed it is clung to tenaciously.

Narcissistic Personality Disorder

This is a new category whose necessity was suggested by an increasing psychoanalytic literature and by the isolation of narcissism as a personality factor in a variety of psychological studies. The distinguishing

features in a psychoanalytic definition of narcissistic character might include 1) deficits in libidinal object constancy, 2) an incomplete internalization or maturation of psychic structures and regulatory mechanisms, particularly those having to do with self-esteem, and 3) aberrant or immature grandiosity. In making this diagnosis, the psychodynamic clinician might assess whether the patient experiences others as fully realized and separate objects or as self-objects and whether the transference that evolves in treatment takes on either a grandiose or idealizing configuration. Unfortunately, it is difficult to put these concepts into a descriptive and operational form that can be applied in one or two interviews by psychiatrists who may not have psychodynamic training or inclination. Instead, the *DSM-III* criteria for narcissistic personality disorder define descriptive and behavioral features that require only limited inferences about psychic structures, object constancy, and motivations. It is not clear to what extent the descriptive and psychodynamic definitions correlate and whether they will select the same patient groups.

CONCLUSIONS

The two major innovations of *DSM-III*—the multiaxial system and the provision of specific diagnostic criteria—are significant contributions to improved diagnosis of personality disorders. The next step in improving reliability is a clearer specification, even if an arbitrary one, of the precise quantity of impairment that defines a disorder. In addition, the individual personality disorders themselves need to be studied in various ways. The current *DSM-III* clustering of criteria is primarily based only on clinical intuition and should be validated with large studies to determine how the various criteria cluster in actual patients. It will be interesting to learn whether certain of the current disorders are highly correlated with each other and could perhaps be collapsed or whether new combinations might emerge. It is not clear whether the current criteria will successfully define relatively homogeneous groups of patients, e.g., the borderline personality disorder may include patients on an affective disorder spectrum, others with problems of impulse control, others with a masochistic need for punishment, and still others with a deficient self-concept. The validation of personality types as well as the investigation of their relationship to other *DSM-III* disorders are important and fascinating areas for future research.

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Self-Induced Water Intoxication in Schizophrenic Patients

BY WILLIAM O. SMITH, MD., AND MERVIN L. CLARK, M.D.

The authors report on 21 cases of self-induced water intoxication and cite 25 others in the literature. Three of their patients demonstrated at least temporary inappropriate antidiuretic hormone secretion, 3 were receiving a diuretic, and the other 15 appeared to have "pure" water intoxication. Fifteen patients were receiving a psychotropic medication. Self-induced water intoxication appears to be more common in schizophrenic patients than is generally realized and should be suspected in any schizophrenic patient who develops convulsions or coma. Hyperdopaminergic CNS activity may be involved, and the authors suggest a possible mechanism similar to that for tardive dyskinesia.

Since our initial report in 1962 (1) of self-induced oral water intoxication in 2 schizophrenic patients without other predisposing illness, 17 reports of this phenomenon involving 25 additional patients have appeared in the literature (2–18). Sixteen of these 27 patients suffered at least temporarily from the syndrome of inappropriate antidiuretic hormone secretion (SIADH); however, 11 of the patients were able to dilute urine in a satisfactory fashion and several of these 11 were shown to excrete a water load normally after recovery.

Since 1962 we have encountered 19 additional schizophrenic patients with self-induced water intoxication. Nine of these patients had suffered a total of 16 documented episodes of water intoxication. Hence, this problem is obviously more common than heretofore believed. Three of our 21 patients showed evidence of SIADH when first observed, 11 did not, and urinary data were not obtained on 4 patients with water intoxication. The other 3 patients were receiving a diuretic.

Normal individuals cannot induce water intoxication in themselves by maximal oral water intake. The mechanism that permits schizophrenic patients to do so remains unknown. Since most of our patients had been receiving antipsychotic drugs before or at the time of their episode of intoxication, the role of these agents (as well as the role of psychosis) must be considered in the etiology of this syndrome. However, some patients were receiving no medication at all before becoming water intoxicated.

PATIENT DESCRIPTION AND CLINICAL FINDINGS

Twenty-one patients with self-induced water intoxication form the basis for this report. We have observed 20 of these patients in our department since 1961, including the 2 patients reported in 1962. Eighteen of these patients were hospitalized on the medical service of Central State Griffin Memorial Hospital in Norman, Okla., 2 were at the Oklahoma City Veterans Administration Hospital, and 1 patient was made available to us courtesy of R. Corenblum, M.D., of the University of Calgary. Three patients had seizures when they were admitted to the hospital; the remainder developed symptoms of water intoxication as inpatients.

The clinical findings and laboratory results for these patients are summarized in table 1. The patients ranged in age from 31 to 63 years; 10 were women and 11 were men. The most frequent and important clinical findings were lethargy, disorientation, confusion, semicoma or coma, and seizures. The seizures were of the grand mal type. It should be stressed that a history of excessive water drinking could be obtained for only 10 of the patients; in the remaining 11 patients neither patient, relatives, ward attendants, nor nurses were able to determine whether the patient had a history of excessive water intake.

Fourteen patients were receiving an antipsychotic drug before the onset of water intoxication. One patient was receiving only an antidepressant, and 2 patients were receiving only a thiazide diuretic. Four of the patients were taking no medication that could be determined.

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TABLE 1
Clinical and Laboratory Findings for 21 Schizophrenic Patients with Self-Induced Water Intoxication

Patient Group and Number	Age (years)	Sex	Serum Na^+ Concentration (mEq/liter)	Serum Osmolality (mosmol/liter)	Urine		Medication	Clinical Findings	Number of Previous Episodes
					Osmolality (mosmol/liter)	Specific Gravity			
Pure water intoxication[a]									
1	49	F	119	231	79	—	Chlorpromazine, imipramine	Seizures, coma	4
2	44	M	111	214	100	—	Thioridazine	Seizures	—
3	35	F	110	—	50	—	Amitriptyline	Seizures, coma	—
4	47	F	118	238	90	—	Loxapine	Seizures, coma	3
Probable pure water intoxication									
5	59	M	105	—	—	1.001	Reserpine	Seizures, coma	—
6	33	M	115	—	—	1.004	Phenothiazines	Seizures, semicoma	1
7[b]	47	F	112	—	—	1.001	Reserpine, chlorpromazine	Seizures, semicoma	1
8	46	M	124	208	—	1.004	Trifluoperazine, chlorpromazine	Seizures, coma	2
9	60	M	112	—	—	1.002	None	Seizures, coma	—
10	54	M	108	—	—	1.003	Thioridazine	Seizures, disorientation	—
11	31	F	113	256	—	1.004	Thiothixene, chlorpromazine	Coma	—
SIADH									
12	39	M	117	229	243	—	None	Seizures	—
Probable SIADH									
13	51	F	114	—	—	1.006	Thioridazine, imipramine	Seizures, coma	—
14	32	M	118	—	—	1.008	Unknown	Seizures	—
Receiving diuretic									
15	63	F	122	244	66	—	Hydrochlorthiazide	Lethargy	—
16	45	F	100	191	287	—	Hydrochlorthiazide	Seizures, semicoma	2
17	49	M	101	217	260	—	Hydrochlorthiazide, haloperidol	Seizures, coma	—
Urine determinations not available									
18	31	M	120	—	—	—	Unknown	Coma	—
19	38	F	110	267	—	—	Chlorpromazine, haloperidol	Seizures	1
20	46	M	114	—	—	—	Trifluoperazine, thioridazine	Seizures, semicoma	1
21	44	F	113	—	—	—	Thioridazine	Seizures, disorientation	1

[a]No evidence of inappropriate secretion of antidiuretic hormone.
[b]Patient 7 had received a prefrontal lobotomy.

LABORATORY FINDINGS

On admission to the medical service, serum sodium concentration for the 21 patients ranged from 100 to 124 mEq/liter. In those patients in whom serum osmolality was measured, it ranged from 191 to 267 mosmol/liter. Urine specific gravity was below 1.005 in all but 2 of the 9 patients for whom it was recorded. In the 7 patients for whom both urine and serum osmolality were determined, 4 patients had a urine osmolality well below their serum osmolality. BUN was found to be 11 mg/100 ml or less in all patients. The white blood cell count was elevated in most patients and, in the absence of demonstrable infection, was considered a response to recent seizure activity. Four patients with symptoms, signs, and serum chemistries consistent with the diagnosis of water intoxication did not have urine concentration studies during the initial 12 hours.

In the 18 instances where water restriction was the sole method of therapy, the patients recovered clinically within 24 hours, after excreting large quantities of urine; 3 of these patients also received hypertonic saline. The following case reports illustrate the two most common types of water intoxication: "pure" water intoxication, which has an unexplained etiology, and water intoxication associated with SIADH.

CASE REPORTS

Case 1 ("pure" water intoxication). Patient 1, Ms. A, was a 49-year-old woman who had been diagnosed as having chronic paranoid schizophrenia and had had four previous admissions for water intoxication secondary to excess water drinking. She had been on a home pass for approximately 5 days and apparently had been doing reasonably well until the afternoon of admission, when she began having seizures. After 4–5 major motor-type seizures she was returned to the hospital bleeding from a large tongue laceration and was given 10 mg of diazepam in the emergency room. Her current medications were chlorpromazine, 200 mg t.i.d.; trihexyphenidyl, 2 mg b.i.d.; and imipramine, 25 mg t.i.d.

On admission Ms. A had a pulse of 100, a rectal temperature of 96°F, and a blood pressure of 108/83. She was semicomatose and responded to painful stimuli. Her head was normocephalic without evidence of trauma, and her optic discs were sharp. She had a large laceration on the left margin of her tongue. Her neck was supple, her lungs were clear, and she had a regular heart rhythm with a grade II/VI systolic murmur at the apex. Her abdomen was soft, and she had active bowel sounds with no palpable masses or organomegaly. Her extremities were free from edema or cyanosis. Her deep tendon reflexes were hypoactive but symmetrical; her Babinski's signs were normal on both sides, and she moved all extremities.

Serum electrolyte levels were as follows: sodium=119 mEq/liter, potassium=4.0 mEq/liter, CO_2=20 mEq/liter, chloride=88 mEq/liter, serum osmolality=231 mosmol/liter, BUN=13 mg/100 ml, and glucose=105 mg/100 ml. Her urine osmolality on admission was 79 mosmol/liter.

Ms. A was placed on fluid restriction of 500 cc per day. By the following morning, after passing 7500 ml of urine, she was alert, communicative, and taking oral feedings. That day her serum electrolytes were within normal limits: sodium=137 mEq/liter, potassium=4.5 mEq/liter, chloride=103 mEq/liter, CO_2=28 mEq/liter, and BUN=9 mg/100 ml. Her serum osmolality was 276 mosmol/liter. Her hematocrit was 42% and her white blood cell count was 12,300/mm^3.

Case 2 (SIADH). Patient 12, Mr. B, was a 39-year-old chronic schizophrenic man who was admitted to the medical service. He had been receiving no medication and had been noted to be drinking large amounts of water on the day before admission. He had vomited on the day before admission and several times early on the morning of admission. Mr. B had been found on the floor, unresponsive and incontinent of urine and feces.

Mr. B had a blood pressure of 120/80, a regular pulse of 60, a respiratory rate of 12, and rectal temperature of 98°F. He responded to painful stimuli and occasional voice commands. His pupils were dilated but equal, round, and reactive. He had multiple conjunctival petechiae. His fundi were normal and had sharp disc margins. His oropharynx and tympanic membranes were clear. His neck was supple. There was a red, papular eruption over his anterior chest and shoulders. His lungs were clear, except for occasional rhonchi. Cardiac examination revealed a normal sinus rhythm without murmur. His abdomen was soft without palpable masses, and his bowel sounds were active. Ecchymoses were noted on Mr. B's arms where he had been held. During the neurological examination Mr. B responded to painful stimuli; his deep tendon reflexes were 2+ and symmetrical without positive Babinski's or Hoffman's signs. Cranial nerve functions II–XII appeared to be grossly intact.

Laboratory studies on admission revealed the following: serum sodium=117 mEq/liter, serum potassium=3.6 mEq/liter, CO_2=24 mEq/liter, chloride=100 mEq/liter, BUN=8 mg/100 ml, and glucose=127 mg/100 ml. Serum osmolality was 229 mosmol/liter and urine osmolality was 243 mosmol/liter.

We felt that Mr. B was water intoxicated and restricted his fluids to 500 cc/day. Over the ensuing 48 hours he became responsive, and his serum sodium concentration rose to 130 mEq/liter and his serum osmolality to 258 mosmol/liter. Measurement of urinary output during this period could not be obtained.

DISCUSSION

The problem of self-induced water intoxication in mentally ill patients without other predisposing illness has now been reported in at least 27 patients in the American literature since the first case was reported by Barahal in 1938 (19). The cases of these patients are summarized in table 2. This is clearly not a rare phenomenon since we have now encountered an additional 19 such patients during the past 17 years. Indeed, water intoxication should be a major consideration in the differential diagnosis of seizure disorders that develop in any mentally ill patient, particularly one in a mental institution. None of our patients had any other illness, such as cardiac, hepatic, or renal disease, that could account for a dilutional state, and none received water other than orally. Three of the patients had been receiving a diuretic agent.

The experiences reported here and in the literature appear to identify at least three groups of patients:

1. Patients for whom the water intoxication has an unexplained etiology. Eleven of our 21 patients are in this category and 11 of the 27 patients cited in the literature would be so classified on the basis of serum and urine osmolalities. Urine osmolalities were not available in 7 of our cases in this category. We have chosen to accept a urine specific gravity of 1.004 or below as evidence of appropriately dilute urine (20–21) and have classified these 7 patients as having probable "pure" water intoxication (see table 1).

2. Patients who have at least a temporary in-

TABLE 2
Studies of Self-Induced Water Intoxication in Mentally Ill Patients

Author	Number of Patients	Diagnosis	Medication	Number of Patients with SIADH
Langgard and Smith (1)	2	Schizophrenia	1 patient, none; 1 patient, reserpine	0
Hobson and English (2)	1	Schizophrenia	Meratran	1
Dubovsky and associates (3)	1	Schizophrenia	None	1
Alexander and associates (4)	1	Schizophrenia	None	1
Chinn (5)	1	Schizophrenia	None	0
Ajlouni and associates (6)	1	Schizophrenia	Thiothixene	1
Olson (7)	1	Schizoid personality	None	1
Nielsen (8)	1	Schizophrenia	None	0
Luzecky and associates (9)	1	Depression	Amitriptyline	1
Raskind and associates (10)	3	Postmenopausal depression with schizophrenic symptoms	2 patients, none; 1 patient, conjugated estrogen	3
Rao and associates (11)	2	Schizophrenia	Thioridazine	0
Fischman (12)	1	Schizophrenia with recent head trauma	Thioridazine	1
DeRivera (13)	1	Schizophrenia	Fluphenazine	1
Mendelson and Deza (14)	4	Schizophrenia	4 patients, fluphenazine; 1 patient, chlorpromazine	1
Matuk and Kalyanaraman (15)	2	Schizophrenia	1 patient, thioridazine; 1 patient, haloperidol	2
Vincent and Emory (16)	1	Depression	Thioridazine	1
Cordoba and Chapel (17)	1	Schizophrenia	Fluphenazine	1
Rendell and associates (18)	2	Schizophrenia	None	0

appropriate secretion of ADH. Three patients in the present series demonstrated findings compatible with this syndrome, and 16 patients in the literature would be so classified. Urine osmolality was not determined in 2 of our 3 patients, and they are classified as having probable SIADH on the basis of urine specific gravity.

3. Occasional patients in whom the syndrome may be precipitated by diuretic therapy. Only 3 of our patients could be considered in this category, and only 3 patients of this type have been reported by other authors.

Urinary studies were not performed on our other 4 patients, but the clinical evidence for water intoxication, including serum osmolality, was compatible with the diagnosis (see table 1).

None of our patients manifested clinical evidence of overhydration (edema, papilledema, etc.) other than symptoms of cerebral edema. This has also been true of most patients reported in the literature, although a patient reported by Raskind and associates (10) died of cerebral edema with brain herniation, and Rendell and associates (18) recently reported 2 patients who died because of compulsive water drinking, 1 of whom was found at autopsy to have pulmonary edema.

In most of our patients it has not been possible to determine the cause of their high water intake. Some stated that they were attempting to wash out worms, other creatures, foul material, or cancer. In 1 reported case the patient stated that she was told by God to drink large quantities of "holy water." Other patients have stated they were cleansing themselves from sin.

All but 6 of our patients were receiving psychotropic medications before developing water intoxication. Little relevant information is available concerning the effect of these pharmacologic agents on salt and water metabolism. Ajlouni and associates (6) suggested that thiothixene is a possible stimulus for SIADH in a manner similar to other drugs such as chloropropamide, carbamazepine, and cyclophosphamide. This suggestion is based on well-documented observations of a case that they challenged and restudied with the use of water loading. Amitriptyline, thioridazine, fluphenazine, and haloperidol have also been linked with water intoxication associated with SIADH, but they have not been studied well enough to implicate them etiologically. They have also been associated with water intoxication without accompanying SIADH. On the other hand, reserpine and chlorpromazine, although associated with the development of water intoxication, have not been implicated in producing SIADH per se. Dyball (22) offered evidence from animal studies indicating effects of various phenothiazines on ADH that varied according to the structure of the side chain at position 10. He demonstrated that chlorpromazine with a three-carbon straight side chain inhibits ADH release, while promethazine with a branched two-carbon side chain stimulates ADH release.

How thiothixene fits into this concept with its three-carbon piperazine side chain structure is not clear. The fact that all of the neuroleptic drugs lower seizure threshold, are anticholinergic, and are dopamine blockers may be more important. Dubovsky and associates (3) suggested that the development of an acute psychotic episode per se, or some consequence thereof, may be the stimulus for ADH secretion. They pos-

tulated a possible autonomic pathway for this stimulation. Raskind and associates (10) hypothesized that the clinical triad observed in their 3 patients—psychotic depression, inappropriate secretion of ADH, and excessive water ingestion (thirst?)—represents a syndrome caused by or associated with disturbed CNS function, particularly dopaminergic hyperactivity. In other words, they suggest a hypothalamic mechanism rather than an autonomic one. To support this they point out the close anatomic proximity of the centers regulating thirst and ADH release and their interconnections with the limbic areas involved with emotions and primitive behavior; they cite evidence from animal studies that indicates the particular importance of dopamine as a neurotransmitter in regulating ADH release and extracellularly stimulated thirst. For example, destruction of hypothalamic dopaminergic pathways abolishes central receptor-induced ADH release (23) and produces adipsia (24); haloperidol, a potent dopamine receptor blocker, also inhibits thirst when injected centrally (preoptic area) (25). On the other hand, dopamine injected into the lateral cerebral ventricle effectively stimulates thirst (25) and when injected into the nucleus accumbens stimulates release of ADH (23). Considering the importance of the catecholamine neurotransmitters in regulating and modulating behavior, it is not difficult to visualize how hyperdopaminergic activity might explain the triad.

One could also speculate about an additional step. Schizophrenia is hypothesized to be associated with absolute or relative cortical hyperdopaminergic activity involving mesolimbic and mesocortical tracts (26). When a patient is treated with drugs that block dopaminergic activity, various aberrations of thirst, drinking behavior, ADH secretion, and other dopaminergic-dependent CNS functions could occur. Consistent with this speculation is the knowledge that nearly all the drugs used to treat schizophrenia have anticholinergic properties capable of producing a dry mouth and throat in man and consequent thirst. These drugs also lower the seizure threshold, thereby rendering patients more susceptible to seizures and perhaps rendering them more likely to have seizures as a result of minimal cerebral edema. However, by blocking dopamine receptors in the hypothalamic areas these drugs would tend to turn off or turn down thirst and to inhibit ADH release; therefore, the drugs, for the most part, by their primary central actions would not produce the water intoxication syndrome with or without inappropriate ADH secretion.

On the other hand, increased thirst and increased ADH release, depending on which receptors are most affected, are possible if a mechanism similar to that hypothesized for tardive dyskinesia is invoked for the dopamine-sensitive cells in the centers that regulate thirst and ADH release. Antipsychotic drugs, by blocking the postsynaptic dopamine receptors, may 1) pharmacologically and functionally denervate these postsynaptic neurons and render them supersensitive (denervation supersensitivity), while 2) simultaneously increasing presynaptic neuronal secretion of dopamine through feedback mechanisms around the block (27). With increased amounts of dopamine impinging on supersensitive cells, the resulting increased dopaminergic activity in the thirst and ADH-release centers could thereby account for the increased thirst and/or ADH release necessary to produce water intoxication. This could occur either as a breakthrough phenomenon while the patient is still taking drugs or a tardive phenomenon when drug treatment is stopped.

Whether ADH or thirst, or both, are affected in this process would depend on individual vulnerability and could thereby account for water intoxication without inappropriate ADH secretion for many of the subjects described in the literature and in this report. In addition, as in Raskind and associates' patients (10), the mechanisms (whatever they may be) that underlie the presumed hyperdopaminergic cortical activity and produce the signs and symptoms of schizophrenia might account for the episodes of water intoxication in the schizophrenic patients who have never had the benefit of treatment with neuroleptic drugs.

Three schizophrenic patients have also been reported to develop water intoxication when given a thiazide diuretic (28, 29). Three of our patients also developed the problem after thiazide administration. Since thiazide diuretics decrease free water clearance, it is quite likely that they contributed to and intensified the dilutional state in these patients during periods of massive water intake.

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Harvard Chair in Human Development

Harvard Medical School and its Department of Psychiatry at the Cambridge Hospital, Cambridge, Mass., are pleased to announce the beginning of a campaign to endow a Chair in Human Development in Honor of Erik H. and Joan M. Erikson. The purpose is to build on the importance of the contributions of the Eriksons to human development, health care, and social and community psychiatry.

For further information contact Daniel C. Tosteson, M.D., Dean, Harvard Medical School, 25 Shattuck St., Boston, MA 02115; Lee B. Macht, M.D., Chairman, Department of Psychiatry, the Cambridge Hospital, Harvard Medical School; or John E. Mack, M.D., Professor of Psychiatry, Harvard Medical School at the Cambridge Hospital, 1493 Cambridge St., Cambridge, MA 02139.

The Prediction of Dangerous Behavior in Emergency Civil Commitment

BY ETHAN S. ROFMAN, M.D., CLIFFORD ASKINAZI, M.D., AND EMILY FANT

The authors compared the records of 59 psychiatric patients involuntarily committed to a Veterans Administration hospital on an emergency basis with those of a control group of 59 psychiatric patients with respect to the number of assaults noted during the first 45 days of hospitalization. The committed group had a .41 probability and the control group a .08 probability of committing an assault. The difference between the two groups was mainly accounted for by assaults that occurred during the first 10 days of hospitalization. The occurrence of an actual act of battery before admission did not predict assault in the hospital to a greater degree than did a verbal threat. The authors conclude that short-term clinical predictions of dangerousness predict assaultiveness in the hospital to a significant degree.

The need and justification for emergency involuntary psychiatric hospitalization is currently a subject of concern for lawyers, psychiatrists, and social scientists. According to *Psychiatric News* (1), in *Mathew v Nelson* a three-judge panel of the federal appeals court in Illinois commented on the limited number of current statistical studies in the area of dangerousness prediction. They stated,

> No study has attempted to measure the extent to which the predictability of dangerousness is enhanced by a history of a recent overt act. . . . No study called to our attention attempts to measure the incidence of violent behavior in a sample population of persons civilly committed for dangerousness (1).

The judges in this case upheld the current Illinois standards for emergency commitment, which do not require an overt act or threat of dangerousness to self or others for involuntary civil commitment. Decisions by federal courts in other states, however, have held that for emergency commitment to be constitutional the threat of danger to others must be imminent and substantial, as evidenced by a recent overt act, attempt, or threat (2). At least one state now specifically provides that a threat alone does not constitute grounds for commitment (3). These legal decisions all question the psychiatrist's ability to forecast dangerousness and call for further study of those circumstances which might permit at least some predictive accuracy under special conditions.

Monahan (4, 5) has discussed the problems inherent in prediction research, pointing specifically to the need for data concerning the accuracy of forecasts made in emergency settings. Monahan hypothesized that in such settings the small temporal "gap" between initial and outcome behavior should theoretically allow for greater predictive accuracy than has been possible to date. He postulated that there is a qualitative difference between forecasts of violence made in the community for the purpose of emergency commitment and those reported in the current literature, which involve judgments made primarily on institutionalized patients or prisoners for the purpose of deciding eventual release.

In this study, we undertook a retrospective review of commitment papers and hospital records to determine the predictive accuracy of community psychiatrists' clinical judgment of potential dangerousness in an emergency setting. The frequency of recorded in-hospital assaultive episodes of psychiatric patients committed on an emergency basis for likelihood of harming others was compared with that of a sample of psychiatric patients admitted to the hospital for all other reasons. We also determined the probability of assaultive behavior occurring in the hospital given the absence or presence of a recent overt violent act in the community, the number of days following admission that assault was most likely to occur, and the relationship of paranoid schizophrenia as a diagnosis to assaultive behavior.

METHOD

We obtained the records of all psychiatric patients admitted to our hospital with emergency commitment status from January 1, 1977, to June 30, 1978, and re-

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viewed the Massachusetts Department of Mental Health Form 5A in each record. This form (the "temporary care paper") requires the applicant to state that in the opinion of the examiner (who is most often a psychiatrist in the community who was not chosen by the patient and whom we did not select for ability to predict dangerousness), the patient requires hospitalization to avoid the likelihood of serious harm by reason of mental illness. Written evidence must be presented supporting this opinion. The form includes the legal definition of "likelihood of serious harm," which is

> 1) substantial risk of physical harm to the person himself as manifested by evidence of threats of, or attempts at, suicide or serious bodily harm; 2) a substantial risk of physical harm to other persons as manifested by evidence of homicidal or other violent behavior or evidence that others are placed in reasonable fear of violent behavior and serious physical harm to them; or 3) a very substantial risk of physical impairment or injury to the person himself as manifested by evidence that such person's judgment is so affected that he is unable to protect himself in the community and that reasonable provision for his protection is not available in the community.

From 108 records initially obtained, we selected for further study 59 (54%) in which the reason for hospitalization given was risk of physical harm to others. We then searched the progress notes in these records for assaultive incidents noted during the 45 days following hospital admission. Such incidents were rated for severity on a 1+ to 4+ scale. The scale was divided as follows: 1+ was a serious assaultive threat that led to no action, 2+ was a threat that generated sufficient staff anxiety to lead to restraint, 3+ was an act that required immediate direct physical intervention by staff or patients to prevent a battery, and 4+ was an act involving a completed battery. We recorded the hospital day on which the incidents took place and the primary *DSM-II* admission diagnosis made by our hospital psychiatrists. The 59 records were further sorted into two groups on the basis of whether or not an actual act of violence was noted on the temporary care paper. The number of patients in each group was then compared to determine the probability of each group's being assaultive in the hospital.

Fifty-nine unmatched consecutive records of psychiatric patients admitted voluntarily or committed to the hospital in April and May of 1978 for reasons other than threat of harm to others were then reviewed; these patients constituted our control group. Fifty-four of these patients were voluntary and 5 were committed. Patients in both experimental and control groups were given psychiatric diagnoses and treated in the hospital by the same staff on the same two 30-bed units, which used a combination of milieu therapy, pharmacotherapy, group therapy, and individual psychotherapy. Both units are usually locked.

TABLE 1
Diagnoses of 59 Patients Committed for Dangerousness and 59 Control Patients

Diagnosis	Committed Group N	Committed Group %	Control Group N	Control Group %
Psychotic organic brain syndrome	3	5.1	0	
Schizophrenia, hebephrenic	0		1	1.7
Schizophrenia, catatonic	2	3.4	0	
Schizophrenia, paranoid	19	32.2	16	27.1
Schizophrenia, acute episode	1	1.7	5	8.5
Schizophrenia, latent	1	1.7	0	
Schizophrenia, schizoaffective	4	6.8	2	3.4
Schizophrenia, chronic undifferentiated	13	22.0	11	18.6
Manic-depressive illness	6	10.2	6	10.2
Psychotic depressive reaction	0		2	3.4
Neurosis	1	1.7	5	8.5
Personality disorder	1	1.7	4	6.8
Alcoholism	8	13.6	5	8.5
Transient situational disturbance	0		2	3.4

RESULTS

The average age of the patients in the experimental group was 38.5 years; in the control group the average age was 37.6. Both groups were composed of 57 men and 2 women. The mean length of stay for the experimental group was 45.5 days (median, 27 days); for the control group the mean stay was 29.9 days (median, 22 days). The diagnoses of the patients in both groups are presented in table 1.

Twenty-four (41%) of the 59 emergency committed patients carried out 52 separate incidents of assault in the hospital. Five (8%) of the 59 control group patients carried out 16 assaultive incidents. The difference between these numbers of patients was significant ($\chi^2=16.504$, $p=.00005$).

The actual number (and percent) of assaultive incidents that were scored 1+, 2+, 3+, and 4+ on our scale was 16 (30.8%), 14 (26.9%), 9 (17.3%), and 13 (25.0%), respectively, for the experimental group and 8 (50%), 2 (12.5%), 0, and 6 (37.5%), respectively, for the control group. Five of the 6 assaultive incidents that were scored 4+ in the control group were carried out by 1 patient.

For 30 of the 59 emergency committed patients, a specific violent act was noted on their emergency commitment form, for 23 no act was noted, and for 6 the data were insufficient to make a determination. Thirteen of the 30 (43%), 9 of the 23 (39%), and 2 of the 6 (33%) people went on to assault somebody after they were hospitalized. There was no significant difference between the patients who had actually acted violently and those who had not with respect to the number of patients in each group who committed assault in the hospital ($\chi^2=.09$, $p=.76$).

The greatest frequency of incidents of assault for both the emergency committed group and the control group occurred within the first 10 days of hospitalization. During this interval 24 committed patients car-

ried out 39 violent incidents and 5 control patients carried out 8. During the next 10 days the committed patients carried out 7 violent acts and the control patients carried out 2. From day 20 to day 40 each group was responsible for only 3 acts.

We also determined the relationship of the diagnosis of paranoid schizophrenia to assaultive behavior rated as 4+. The records of all paranoid schizophrenic patients in the experimental group and the control group were compared with those of all other patients in both groups with respect to the completion of assaultive acts noted 4+. The 6 patients with a diagnosis of paranoid schizophrenia who committed such violent acts represented 16.2% of the 37 patients with this diagnosis. The 4 patients with all other diagnoses who committed such acts represented 4.9% of the 81 patients with other diagnoses. The difference between the number of paranoid schizophrenic patients and those with all other diagnoses who committed acts with a rating of 4+ was significant (χ^2=4.1649, p=.0413).

DISCUSSION

This preliminary retrospective study indicates that the clinical determination of dangerousness by physicians who send people to a typical psychiatric Veterans Administration medical center for emergency psychiatric commitment predicts the patients at risk of in-hospital assaultive behavior to a statistically significant degree. In the group of patients who were committed to the hospital on an emergency basis the incidence of all assaultive episodes was 41% and for acts of battery alone was 13% in 45 days. This compared with an incidence of 8% and 3% in a control group of psychiatric patients. This finding supports the hypothesis that predictions of dangerous behavior have validity when the time of prediction and the time of validation are relatively close together.

Our results indicate that the history of a recent overt assaultive act as noted on an emergency commitment paper and the physician's clinical assessment of potential dangerousness in the absence of an overt act predict assaultiveness in the hospital to about the same degree. Therefore, an overt act of assault should not be a requirement for emergency commitment.

The ability to predict when assaultiveness in the hospital is most likely to occur is important from a clinical point of view. There is often staff pressure to keep a patient who commits an assault in a maximum security setting for an indefinite length of time. Information as to when patients who commit a single episode of assault are actually likely to stop being violent would help clinicians to know when such patients should be transferred to less restrictive settings. The data presented here indicate that patients who were thought harmful before admission are about as assaultive as all other patients after 20 days of hospitalization.

Patients with a diagnosis of paranoid schizophrenia in this study were responsible for roughly an equal number of all assaultive episodes and a statistically significant number of acts rated most violent during the 45 days after admission to the hospital. These paranoid patients were all men under the age of 45, and often they were also responsible for a series of acts rated most violent that continued to occur beyond the 45-day cutoff point of the study. These results support the conventionally held viewpoint that younger paranoid schizophrenic patients tend to be more dangerous than other patients in a large psychiatric hospital.

It should be noted that our control group was probably more dangerous than the general population: it contained paranoid schizophrenic patients. Therefore, any comparison with respect to risk ratios, such as the 41%:8% we found, considerably understates the dangerousness of the committed group. Furthermore, all of the assaults in our study took place in a structured hospital setting where attempts to understand feelings, opportunities for ventilation, and the ability to control aggressive behavior were apparent. All patients in both groups were receiving adequate doses of appropriate antipsychotic medication. It is likely, therefore, that the probability of the patients in our experimental group (who would be unmedicated outside the hospital) committing assaults in the community would have far exceeded 41% without emergency commitment.

Monahan and Wexler (6) pointed out that when a psychiatrist makes a prediction of dangerousness without further specifications, he or she is making at least three separable assertions:

1. The individual being examined has certain characteristics.
2. These characteristics are associated with a certain probability of violent behavior.
3. The probability of violent behavior is sufficiently great to justify preventive intervention.

The first two of these assertions are subject to various "standards of proof"; the third is essentially a social policy trade-off decided in the political process.

Our research basically addresses the second of these assertions and is an attempt to obtain empirical data concerning the probabilities involved in short-term prediction. We would agree with those who hold that many people ignore important differences between populations in degree of dangerousness and by so doing wrongly fail to consider respectable empirical correlations (7). This often occurs when there is confusion with respect to dangerousness as a probability statement, dangerousness as a social policy decision, and dangerousness as an individual outcome.

As a result of this confusion, critics of psychiatry have often been given a free hand in imposing on the public their own, often covert, social cost analysis. This operation has been characterized by an exclusive emphasis on implicit social cost comparisons in one direction only (false positives) and by a unilateral re-

definition of the prediction problem from one involving probabilities to one involving individual outcomes.

Given the social cost of a brief deprivation of liberty for those who are not fully dangerous (because dangerousness is not an all-or-nothing variable) versus the social cost of death or injury to victims of people not committed on an emergency basis, we believe our study demonstrates sufficient predictive accuracy to justify the use of brief emergency commitment. It further indicates that patients committed on an emergency basis are likely to harm staff and other patients until their psychoses have been diagnosed, treated, and brought under adequate control. These patients should, therefore, be treated with care, respect, and adequate staffing.

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Foundations' Fund Prize for Research in Psychiatry

The American Psychiatric Association takes pleasure in inviting submissions for the fourth annual Foundations' Fund Prize for Research in Psychiatry.

Candidates for this prize should be citizens of the United States or Canada and should be nominated by a sponsor. Sponsors should be members of the American Psychiatric Association. Members of the prize board are excluded from submitting nominations.

The *sponsor* should submit a supporting letter justifying the nomination in detail and summarizing the research accomplishments of the nominee.

The *nominee* should submit:

1) An up-to-date curriculum vitae;
2) An up-to-date bibliography;
3) A single paper or book or a group of representative, and thematically linked, papers or books published (or accepted for publication) in English and dated within ten years prior to the deadline of submission;
4) A summary statement written by the nominee, emphasizing the principal theme (or themes) of the work and its internal cohesiveness and consistency.

Six copies of each entry should be submitted to Joel J. Elkes, M.D., Chairman, Foundations' Fund Prize Board for Research in Psychiatry, American Psychiatric Association, 1700 Eighteenth Street, N.W., Washington, D.C. 20009. Entries will be acknowledged but cannot be returned. The award will be presented at the Convocation of Fellows at the 1981 APA annual meeting.

The deadline for submission is November 1, 1980.

The Mental and Nervous Disorder Utilization and Cost Survey: An Analysis of Insurance for Mental Disorders

BY O.B. TOWERY, M.D., STEVEN S. SHARFSTEIN, M.D., AND IRVING D. GOLDBERG, M.P.H.

The Mental and Nervous Disorder Utilization and Cost Survey in Washington, D.C., has yielded useful information about outpatient utilization within an insurance plan with broad mental health coverage, as well as evidence that a comprehensive benefit with a low deductible and copayment can be offered and reasonably utilized. From the results of this survey and a substudy of claims made during 1977, the authors conclude that psychiatric diagnostic information submitted on insurance claim forms may often be inaccurate, primarily because of providers' concerns about confidentiality; such information is of little use for peer review or claims review. The authors state that caution must be exercised in generalizing from the experience under this plan to speculate about outpatient utilization which might result from broad coverage on a national level.

The Mental and Nervous Disorder Utilization and Cost Survey (MANDUCS) is a statistical aggregation of all outpatient claims in one year for patients in the Washington, D.C., area who are covered by the Government-Wide Service Benefit Plan, usually referred to as the Blue Cross and Blue Shield Federal Employee Program (FEP). It represents the first large-scale survey of primarily office-based private practice using insurance claims forms as the input data. The utilization experience is especially important since FEP offers one of the most comprehensive mental health benefits in the nation. After a deductible of $100 or $200, depending on whether the coverage is high option or low option, 80% or 75% of usual and customary charges for all outpatient psychiatric visits is covered, the only limit being a lifetime maximum of $50,000. Services can be provided by either psychiatrists or psychologists as independent practitioners and by psychiatric nurses and social workers if they receive physician supervision.

As a body of data MANDUCS can be used to compare the mental health professions along a number of variables related to utilization and can be used to assess relationships among persons served, visits made, and costs (charges) for services. One of the original aims of the study was to permit the development of statistical patterns of utilization and costs to be used as a basis of comparison for selected or sampled individual cases by the insurance carrier for claims review or by local professional societies for peer review. However, because of evidence generated by the study (discussed below) that the diagnostic information submitted is of questionable accuracy, the information provided by the study is of only limited value for claims and peer review based on diagnosis. In this paper we will review the findings of the study and discuss implications of the data.

HISTORY OF MANDUCS

In 1975 there were cutbacks in psychiatric benefits in the two major plans participating in the Federal Employees Health Benefits Program; benefits in these two plans had been expanded in 1967 to be virtually unlimited and equivalent to general health care coverage. The second largest plan, the Government-Wide Indemnity Benefit Plan sponsored by the Aetna Insurance Company, which had experienced high psychiatric costs, cut outpatient coverage to an annual limit of 20 visits in a private office and 40 visits in a qualified community mental health center. In the "open" season a large number of Aetna enrollees with psychiatric claims switched to Blue Cross and Blue Shield (1). After initially proposing a similar major cutback in coverage, Blue Cross and Blue Shield instead introduced a lifetime maximum of $50,000 for mental disorders under its supplemental benefits. Further, they promised the Civil Service Commission that they would increase their efforts to review and account for the high expenses associated with psychiatric treatment.

In February 1975 the national office of Blue Cross and Blue Shield sent a new form, called the Mental

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Opinions expressed herein are those of the authors and do not necessarily reflect the official policy of the Alcohol, Drug Abuse, and Mental Health Administration.

Health Report Form, along with guidelines for nervous and mental disorders to the local Blue Cross and Blue Shield plans that administer the FEP. The instruction to the local plans stated, "It is imperative that we strengthen the review process in our efforts to administer the Federal Employee Program's nervous and mental benefits in a manner consistent with appropriate utilization. We believe it is necessary that Plans implement these guidelines in their claims review."[1] In the Washington, D.C., area the local plan, Group Hospitalization, Inc., delayed introducing the Mental Health Report Form to allow for provider input and to design the statistical survey known as MANDUCS.

Why was the Washington, D.C., area chosen for special study? In 1973 the Blue Cross and Blue Shield FEP-covered population in the Washington area represented 21% of the total FEP enrollees nationwide but included 32% of those persons treated for mental disorders and accounted for 36% of the total mental disorder benefit costs incurred. Under Aetna, residents in the Washington, D.C., area constituted 17% of total Aetna enrollees but accounted for 48% of the total mental illness benefits incurred. Whereas the combined Aetna and Blue Cross and Blue Shield mental health care costs comprised about 8% of the two plans' total health care costs nationwide, in the Washington, D.C., area the portion was almost 15% of the total. Further, the data indicated that the Washington, D.C., area accounted for a very high proportion of the larger claims—that is, 38% of all mental disorder claimants each of whom had total outpatient claims of over $7,000 in 1973. Due to these high expenses, a study of the Washington, D.C., area's patterns of utilization and cost, which could augment claims review and peer review, was of highest priority (1).

The provider community in Washington, including psychiatrists, psychologists, psychiatric nurses, and social workers, is large and well-organized. The introduction of the Mental Health Report Form, which asked for a diagnosis, symptom check, and estimate of degree of impairment, occasioned strong comment and protest. Many federal employees feared breaches of confidential information to employer or outside groups. It took 18 months for the provider organizations and Blue Cross and Blue Shield, meeting under the auspices of the Civil Service Commission and the National Institute of Mental Health, to agree to the introduction of the report form and the design of MANDUCS. After the form was introduced in the summer of 1976, many consumers and providers confused the Mental Health Report Form and augmented claims review with the statistical aggregations for MANDUCS. They protested to the Civil Service Commission, the Congress, and the Mental Health Law Project. Blue Cross and Blue Shield withdrew the form after one year of use and substituted a requirement for an itemized bill that included a diagnosis classifiable under *DSM-II*. The MANDUCS Advisory Committee, made up of representatives of the mental health professional associations, the Mental Health Association (a citizens' group), the insurance carriers, NIMH, and the Civil Service Commission, continued to meet and advise on the project. The experience with the Mental Health Report Form in Washington, D.C., is an important case study of the acceptability and usefulness of such a form for claims review.

DATA COLLECTION

MANDUCS comprises a statistical aggregation of six months (January–June) of report forms and six months (July–December) of itemized bills for individual and group psychotherapy collected for calendar year 1977. Insurance forms and itemized bills were abstracted and placed in an anonymous automated data file. The study included supplemental claims and excluded basic benefits. This meant that all outpatient care was included, but inpatient care was not. Because, with only minor exceptions, this survey was based on claims submitted for payment rather than on service actually rendered, there are several important points to bear in mind in interpreting the data. First, since there is a $100 or $200 deductible under supplemental claims, individuals who were seen only once or twice and did not submit claims are not included in the survey (other studies [2] have indicated that this group may comprise as much as one-third of all psychiatric outpatients). To some extent the amount claimed might have been influenced by the fact that the maximum amount payable for each visit varied by type of provider, with psychiatrists receiving a maximum of $45 per visit, psychologists $40, social workers $30, and nurses $20. Finally, patients who never submitted claims for services received because of concerns about confidentiality or other reasons would of course not be included in this survey.

Throughout the next section, in which the findings of the survey are presented, it is important to keep in mind that some individuals were seen by different types of therapists or received different diagnoses at different visits. Thus, the summation of patients across provider types or across diagnostic groupings results in a duplicated count of individuals.

A final caveat about the data collected in this survey relates to the fact that some bills for treatment are not submitted until several months after the treatment is rendered. The cutoff date for collection of these data was extended to February 1978, but even so more bills came in throughout the rest of the year. Although there is no way to determine exactly what percent of the total claims for service in 1977 the MANDUCS

[1] R. Leuhrs: Guidelines for Claims Review for Nervous and Mental Disorders, Blue Cross/Blue Shield Federal Employee Program, Feb. 1975.

TABLE 1
MANDUC Survey Data on the Utilization and Cost of Outpatient Services for Mental Disorders for 15,484 Individuals in 1977, by Type of Provider

Measures of Utilization and Cost	Board-Eligible Psychiatrists	Clinical Psychologists	Psychiatric Social Workers	Psychiatric Nurses	Nonpsychiatric Physicians	All Providers
Individuals						
Number	11,232	3,760	2,459	126	91	15,484[a]
Percent	72.5	24.3	15.9	0.8	0.6	100[a]
Total visits	352,303	93,155	58,713	1,376	904	506,451
Average number of visits per individual	31.4	24.8	23.9	10.9	9.9	32.7
Median number of visits per individual	15.9	15.4	14.7	6.8	6.2	19.5
Total dollars claimed	14,788,492	3,504,803	1,760,327	27,861	33,826	20,115,309
Percent of total dollars claimed	73.5	17.4	8.8	0.1	0.2	100
Average dollars per individual claimed	1,317	932	716	221	372	1,299
Average dollars per visit claimed	42	38	30	20	37	40

[a]Not a row sum because some individuals were seen by more than one type of provider.

data represent, the Blue Cross and Blue Shield claims department estimates they account for approximately 80% of all claims for treatment rendered in 1977.

FINDINGS

Tables 1-5 present some of the major findings from the MANDUC survey. During the 1977 calendar year there were approximately 758,771 participants in the Washington, D.C., area Blue Cross and Blue Shield FEP. As can be seen from table 1, mental disorder claims were submitted during 1977 for 15,484 individuals (2% of the enrolled population), for a total of 506,451 outpatient visits. The total charges for these visits were $20,115,309, for an average of $1,299.10 per individual and $40.00 per visit. The cost of outpatient psychotherapy per enrollee was approximately $26.50 per year.

As can be seen in table 1, most patients were seen by psychiatrists, followed by psychologists and psychiatric social workers. Only a small proportion of patients were seen by psychiatric nurses and nonpsychiatric physicians. The proportion of visits claimed by each type of provider was similar, with psychiatrists making claims for 69.6% of the visits, psychologists 18.4%, social workers 11.6%, psychiatric nurses 0.3%, and nonpsychiatric physicians 0.2%.

Table 2 presents data on the percent of individuals with paid claims who made a specified number of visits to the indicated type of provider. Similarly, table 3 presents data on the percent of all visits to that provider type accounted for by those individuals. Table 3 shows that approximately one-half of all visits paid for were those of individuals who made 60 visits or less, and table 2 reveals that these individuals represented 84% of all subjects in the study. Stated conversely, nearly one-half of all visits paid for were for the 16% of individuals who made 60 visits or more in 1977. This situation, in which a small number of recipients use a large portion of services, is comparable to other medical benefits, especially those in the "catastrophic" category. In table 4 we see that these individuals accounted for more than one-half of all money claimed for the 1977 outpatient mental benefit.

It is important to note that although we may be able to conclude from these data that a small proportion of patients account for the majority of visits in any one year, we must be cautious about any inferences related to the extent to which intensive psychotherapy or psychoanalysis accounts for this. Because the only claims tabulated were those made for services received between January 1, 1977, and December 31, 1977, many individuals who either ended treatment early in 1977 or began treatment late in 1977 would have only a small number of visits claimed for 1977. For example, an individual beginning treatment on November 1 and receiving treatment 4 times each week would have only 32 visits tabulated for 1977 and would appear as one of the individuals receiving 32 visits or fewer in this study.

It can also be seen from table 3 that psychiatrists tended to have more claims for individuals with a large number of visits in 1977 than did the other types of providers. Although about 60% of the visits claimed by both the psychologists and social workers were for individuals with 50 visits or less, only about 40% of the visits claimed by psychiatrists were for such individuals. It is possible that some of this difference is attributable to the great number of psychoanalysts in the area.

For discussion purposes we have included in table 5 the distribution of diagnostic categories that were recorded on the claims which were paid. Although these

TABLE 2
MANDUC Survey Data on the Cumulative Percent of 15,484 Individuals with a Specified Number of Visits to Various Types of Providers in 1977

Number of Visits	Percent of Individuals					
	Board-Eligible Psychiatrists	Clinical Psychologists	Psychiatric Social Workers	Psychiatric Nurses	Nonpsychiatric Physicians	All Providers
1-5	24.7	21.9	20.0	42.9	44.0	17.0
1-10	39.0	37.2	37.1	66.7	72.5	31.5
1-20	55.9	57.8	60.3	87.3	86.8	50.8
1-30	66.2	70.9	75.1	92.1	92.3	63.4
1-40	74.9	80.3	84.3	96.8	96.7	73.4
1-50	80.5	87.4	89.0	98.4	97.8	80.2
1-60	83.9	90.5	91.5	98.4	100.0	84.0
1-80	89.6	95.1	95.4	99.2	100.0	90.2
1-100	93.5	98.1	98.0	100.0	100.0	94.2
1-150	97.4	99.7	99.3	100.0	100.0	97.8
1-200	99.5	99.9	99.7	100.0	100.0	99.6
1-200+	100.0	100.0	100.0	100.0	100.0	100.0

TABLE 3
MANDUC Survey Data on Cumulative Percent of Visits Accounted for by 15,484 Individuals with a Specified Number of Visits to Various Types of Providers in 1977

Number of Visits	Percent of Visits					
	Board-Eligible Psychiatrists	Clinical Psychologists	Psychiatric Social Workers	Psychiatric Nurses	Nonpsychiatric Physicians	All Providers
1-5	2.0	2.6	2.4	11.3	12.7	1.6
1-10	5.6	7.4	8.1	29.4	35.0	5.1
1-20	13.7	20.0	22.7	56.2	55.6	14.0
1-30	21.9	33.3	38.3	66.4	68.8	23.7
1-40	31.8	46.7	51.9	82.8	84.0	34.5
1-50	39.8	59.5	60.6	89.2	88.5	43.8
1-60	45.8	66.3	66.4	89.2	100.0	50.3
1-80	58.6	79.5	77.7	93.8	100.0	63.5
1-100	69.6	90.4	87.5	100.0	100.0	74.6
1-150	85.0	97.7	93.8	100.0	100.0	88.1
1-200	96.4	99.5	97.0	100.0	100.0	97.0
1-200+	100.0	100.0	100.0	100.0	100.0	100.0

TABLE 4
MANDUC Survey Data on the Cumulative Percent of Dollars Claimed by 15,484 Individuals with a Specified Number of Visits to Various Types of Providers in 1977

Number of Visits	Percent of Dollars Claimed					
	Board-Eligible Psychiatrists	Clinical Psychologists	Psychiatric Social Workers	Psychiatric Nurses	Nonpsychiatric Physicians	All Providers
1-5	2.1	3.5	2.5	12.4	13.7	1.7
1-10	5.8	8.5	8.3	29.9	36.2	5.4
1-20	14.0	21.5	23.1	57.3	57.1	14.4
1-30	22.3	34.6	38.8	67.9	68.3	23.9
1-40	31.9	48.3	52.1	86.3	82.6	34.4
1-50	39.9	60.7	60.9	93.3	88.1	43.6
1-60	45.6	67.4	66.7	93.3	100.0	49.9
1-80	58.3	80.2	77.7	96.1	100.0	62.7
1-100	68.6	90.6	87.4	100.0	100.0	73.3
1-150	84.3	97.7	93.8	100.0	100.0	87.3
1-200	96.3	99.5	97.1	100.0	100.0	96.9
1-200+	100.0	100.0	100.0	100.0	100.0	100.0

data are valid from the standpoint that they represent what was submitted on the insurance claims, we do not believe that they accurately reflect actual practice in the area. A substudy conducted by the Washington Psychiatric Society provided evidence that providers have submitted less "severe" diagnoses (i.e., more diagnoses of neurosis and transient situational adjustment reaction) because of legitimate concerns about confidentiality. For the substudy, area psychiatrists were asked to anonymously submit aggregate data on the numbers of FEP patients they saw in 1977 and their diagnoses, with no identifying patient information.

TABLE 5
MANDUC Survey Data on the Number and Percent of 15,484 Individuals with a Specified Diagnosis of Mental Disorder on Claims by All Providers in 1977

	Individuals with Diagnosis	
DSM-II Diagnosis	Number[a]	Percent
Schizophrenia	821	5.3
Affective disorders	474	3.1
Psychotic organic brain syndrome	32	0.2
Neuroses	12,742	82.3
Personality disorders	1,585	10.2
Alcoholism	145	0.9
Drug dependence	17	0.1
Psychophysiologic disorders	182	1.2
Transient situational disorders	1,892	12.2
Nonpsychotic organic brain syndrome	36	0.2
Mental retardation	11	0.1
All other diagnoses	858	5.5

[a]Numbers total more than 15,484 because some individuals received more than one diagnosis.

Diagnoses seen in the Washington Psychiatric Society study were much more evenly distributed across diagnostic categories then those in the MANDUC survey, and the distribution more closely resembles that found in other studies of private-practice psychiatry, such as the Joint Information Service study of 1975 (3). Because of this finding, we feel that tabulations such as numbers of visits and dollars claimed based on each diagnostic category are of little value for peer and claims review purposes unless mechanisms can be developed to collect these data separately from personal identifying information. We have therefore omitted these data from our presentation.

DISCUSSION

There is a paucity of knowledge about the utilization of outpatient mental health services in the private sector. Nonetheless, health insurers and others must make difficult decisions related to the types of services and service providers, as well as length and intensity of treatment, that should be covered. The MANDUCS data can be very informative when used alongside data from other studies of different populations in different treatment settings. However, we would like to caution about the extent to which these data can be used as a basis for speculation about the degree of utilization nationwide if the entire population were covered by an outpatient nervous and mental benefit as comprehensive as the Blue Cross and Blue Shield FEP. The Washington area is in many ways not typical of the rest of the country. There is a very dense concentration of mental health providers in the Washington area, probably as dense as or denser than any other area in the country. In general, the insured population is more highly educated and of a higher socioeconomic class than is found in most areas. Employment and other demographic characteristics of the insured, such as average age and family size, no doubt also affect the utilization patterns in this area.[2]

Given these considerations, it is probably safe to conclude that utilization under the FEP represents the most intensive utilization that might occur under such a comprehensive mental health benefit. Certainly the utilization is greater than has been reported in other studies of outpatient psychiatric practice (4).

Is the 2% of the enrolled population who use mental health services too high? Is the $26.50 charged per enrollee to pay for these services excessive? Questions such as these can only be addressed in the context of information not provided by a survey such as MANDUCS. In order to meaningfully discuss them, we must consider such factors as the effectiveness of the treatment in alleviating distress and improving the level of social and occupational functioning and the costs that would result if such treatment were not available. Ultimately, we must make decisions based on the value and importance that we attribute to providing psychotherapy as a means of treating mental disorders.

A final issue that merits discussion relates to any attempt to analyze utilization patterns of a health insurance benefit based on the data submitted on the claims form. This is the question of the accuracy of the data that are submitted. Here, the most important questions do not relate to fraudulent and abusive practices. Although there may be instances of such practices as upgrading (billing for services more extensive than those actually provided) and billing for treatment sessions not actually rendered (5), it is doubtful that any of these practices occur on a wide enough scale to greatly alter the utilization patterns that are inferred from the data collected. A study such as MANDUCS is not designed to detect such practices, for to do so would require the development of individual profiles of providers and patients.

The most questionable item submitted on FEP mental and nervous disorder insurance claims is the diagnosis. Even though many have questioned the usefulness of a *DSM-II* diagnosis in determining the appropriate length and intensity of psychotherapy, insurers have generally insisted that a *DSM-II* diagnosis be submitted as part of the routine claims procedure. Before the MANDUC survey was begun, two hypotheses were entertained relating to the accuracy of diagnoses submitted. The first was that mental health professionals, because of their desire to make sure that a patient perceived as needing therapy should not be denied coverage by the insurance carrier for some arbitrary reason related to diagnosis, might submit a more severe diagnosis such as schizophrenia in an attempt to assure continued treatment coverage. The second hypothesis was that fears about confidentiality might lead

[2]S.S. Sharfstein, B. Dean: Washington psychiatry—boom or bust? Presented at a meeting of the Washington Psychiatric Society, Sept. 1977.

providers to submit diagnoses perceived to be less severe because of the social stigma and negative connotations of many diagnoses.

From the results of the MANDUC survey it would appear that the former practice occurred rarely, if at all, simply because few diagnoses such as schizophrenia, manic-depressive psychosis, or even the personality disorders were submitted. Ninety-four percent of all diagnoses presented were for either neurosis or transient situational adjustment reaction. If these diagnoses are inaccurate, we feel that it is probably due to providers' concerns about confidentiality. Such diagnostic information is of little use for peer review or claims review unless mechanisms are developed to collect it without personal identifying data. Such a system, developed by the Washington Psychiatric Society, has been described elsewhere (6).

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OPINION AND COMMENT

"Medical Responsibility" in Institutional Settings

BY BEN BURSTEN, M.D.

The economics of institutional delivery of mental health care sometimes makes it impossible for psychiatrists to meet the demands of medical ethics. At times the psychiatrist's signature is used to imply a degree of medical responsibility that is not justified or a degree of supervision of less highly trained personnel that has not been given. The continuing variance between psychiatry's ethical precepts and its practice in some institutions creates an undesirable situation. The author examines several options and concludes that the ethics should be rewritten. Dr. Frederick J. Stoddard comments on the author's presentation.

Henry Tucker, a prisoner in the state of Virginia, recently won a lawsuit charging that antipsychotic drugs were administered to him improperly. Although the suit centered around extensive neurological damage, pertinent to the complaint were the facts that the medications were ordered by a nonphysician and the orders were countersigned by physicians. The countersigning physicians had not performed a complete examination of Mr. Tucker. According to *Psychiatric News* (1), the physicians' direct contacts with Mr. Tucker were confined to rounds, during which "he (Tucker) would be seen but insofar as hands-on procedure, no. We would be seeing, ordinarily, pass the amenities, 'How are you today?' . . . and then go on to the next." The expert witnesses for the plaintiff stressed the impropriety of delegating responsibility to people who were not qualified. Decisions such as the Tucker case should arouse the greatest concern throughout our profession.

DIFFERENCES BETWEEN PRINCIPLES AND PRACTICE

Consider the *Principles of Medical Ethics* promulgated by the American Medical Association in 1957 and annotated and revised with reference to psychiatry by the American Psychiatric Association in 1978 (2). Section 6 of the *Principles* states, "A physician should not dispose of his services under terms or conditions which tend to interfere with or impair the free and complete exercise of his medical judgment and skill or tend to cause a deterioration of the quality of medical care" (2, p. 3). The annotations for section 6 continue,

> In relationships between psychiatrists and practicing licensed psychologists, the physician should not delegate to the psychologist or, in fact, to any nonmedical person any matter requiring the exercise of professional medical judgment. . . . When the psychiatrist assumes a collaborative or supervisory role with another mental health worker, he/she must expend sufficient time to assure that proper care is given. It is contrary to the interests of the patient and to patient care if he/she allows himself/herself to be used as a figurehead. . . . In referring patients for treatment, counseling, or rehabilitation to any of these practitioners, the psychiatrist should ensure that the allied professional or paraprofessional with whom he/she is dealing is . . . competent to carry out the therapeutic task required. The psychiatrist should have the same attitude toward members of the medical profession to whom he/she refers patients. (1, p. 7)

I submit that in many institutional settings this precept is largely ignored.

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Often in institutional settings patients theoretically under our care are seen primarily by personnel other than psychiatrists. It is not uncommon that the treatment plans we sign are carried out in significant part by master's level psychologists, bachelor's level social workers, and nurses who may or may not have a four-year college degree. Does our signature indicate that we have spent sufficient time to understand in detail what other team members will do? Does it indicate that we approve of or endorse these efforts, or is it sometimes a matter of compromise with the status aspirations of other personnel or a necessary medical-appearing validation that allows them to do whatever it is they do? I suggest that often the psychiatrist's involvement amounts to little more than providing a legalizing countersignature. Indeed, the sheer number of patients for whom some psychiatrists assume responsibility, especially in outpatient settings, often precludes either direct contact or meaningful supervision.

In institutional settings we work with whoever is on board. Sometimes our colleagues are superb; sometimes they are merely holding down a job—and this goes for medical as well as nonmedical colleagues. The psychiatrist is not in a position to select the colleague to whom he or she will refer. In most hospitals, each discipline has the autonomy to select its own staff, often without consultation with the other disciplines. This system makes a mockery of our ethical standard of assuming the competence of colleagues to whom we refer patients within an institution.

Section 7 of the *Principles* states, "In the practice of medicine, a physician should limit the source of his professional income to medical services actually rendered by him, or under his supervision, to his patients" (1, p. 3). Carrying this precept over to institutional settings, we are not talking about the source of the *physician*'s income but, rather, the source of the *institution*'s income billed in the name of the physician. It seems to me that the principle would be the same, and I submit that in many institutional settings this precept is largely ignored.

The criteria of adequacy of supervision have not been spelled out; however, much of what goes on in some large institutions is virtually unsupervised. The concept of physician-extenders is rapidly gaining acceptance as a viable dimension of health care delivery. However, "physician-extender" does not mean "physician-replacer," and at times what is billed as the former proves to be the latter. There may be instances in the delivery of health care when it is appropriate actually to replace the physician by a less highly trained person or a differently trained person. However, it is not always clear that third-party payers desire to reimburse for the services of unsupervised less highly trained personnel at the same rate that they would reimburse for physicians' services. When we ambiguously bill under the physician's name as if the services were provided or supervised by the physician, we may be violating section 7 of the *Principles of Medical Ethics*. That such practice occurs has been documented by Towery and Sharfstein (3). According to them, such practice may be more than violation of an ethical principle; it may be fraud. Even if it is not fraud (which implies intentionality), there may still be negligent misrepresentation.

When we consider billing practices we must be careful to distinguish the psychiatrist in his or her administrative role from the psychiatrist in his or her treatment role. As an administrator, the psychiatrist who signs a bill may be indicating only a certification that, to the best of his or her knowledge, the work was performed. As a treater, however, the psychiatrist who signs a bill seems to imply that the physician-signer has assumed medical responsibility and that whatever delegation may have occurred has been well supervised.

The annotation to section 10 of the *Principles* states that "it is unethical for a psychiatrist to offer a professional opinion unless he/she has conducted an examination" (1, p. 11). Although this guideline has reference to diagnosing public figures, it would seem all the more relevant as a guideline to diagnosis leading to treatment implications in terms of actual patients. I submit that in many institutional settings this precept is largely ignored.

At times psychiatrists countersign intake reports of patients whom they have not personally seen. They may sign treatment plans for patients whom they have not personally interviewed. Sound medical practice requires that a plan of treatment be initiated only after adequate diagnosis. Therefore, it follows that a psychiatrist who lends his or her name and weight of authority to a treatment plan implies that he or she is satisfied with and is endorsing the diagnosis. According to the precept of section 10 as annotated, this cannot be done at long distance.

Another practice that is relevant to "long distance" diagnosis is the use of routine and p.r.n. orders. Some so-called doctor's orders are routine to the point of being preprinted. When we order an injectable neuroleptic or seclusion "p.r.n. agitation," a diagnosis is required when the decision to employ the order is made. The nursing staff, at whatever level is allowed to give injections or place people in seclusion, diagnoses the existence of sufficient agitation and diagnoses the quality of the agitation in relation to its social surround to judge whether it requires this medical intervention. The decision of medical appropriateness (as contrasted with, for example, punitive appropriateness or inappropriateness) is not made by the doctor who signed the order. One might argue that the doctor made the initial assessment of the patient's volatility or lack of impulse control, but I have seen many admission work-ups with no evidence of volatility followed by routine orders for p.r.n. medications or restraints.

In large institutions, then, this diagnostic appraisal

is often delegated to less highly trained personnel with the doctor's signature given well in advance of the incident. In cases of acute emergency, few would quarrel with the use of restraint by anyone on the scene. However, the p.r.n. order is often carried out under the psychiatrist's authority in a somewhat less than emergency situation. It is difficult to see how the psychiatrist can have made an adequate diagnostic assessment before the agitation even occurred.

There are more appropriate p.r.n. orders in which the decision making is given to the patient himself or herself. Not infrequently we will give the patient sleeping medications and say, "Take one of these pills if you have trouble going to sleep." When the decision lies with the patient, the implication is that the psychiatrist has decided that the patient is capable, within limits, of regulating his or her medication. However, when the decision lies with someone other than the patient, there is a delegation of diagnostic authority that needs to be well thought through.

CAUSES FOR THE DIFFERENCES

Why are some of our institutional practices at variance with our ethical precepts? I do not believe that we psychiatrists are generally motivated by either avarice or malice. More often, institutional psychiatrists are forced into these practices by the politics and economics of the delivery system. *Psychiatric News* (4) quoted the late Browning Hoffman's definition of institutional psychiatry as "that mental health care delivery system in which massive numbers of patients are assigned to minimal numbers of often poorly trained professionals and are 'treated' by techniques and practices that further institutional as well as, and perhaps instead of, patient interests." Institutional psychiatrists simply do not have the time to adhere strictly to the ethical precepts.

I doubt that this numbers-bind will be resolved by deinstitutionalization programs. There is evidence (5, 6) that large numbers of people who are perhaps prematurely discharged from hospitals find their way into nursing homes, emergency rooms, underfunded and understaffed community mental health centers, and boarding homes subsidized by the welfare system. It should be clear by now that the so-called deinstitutionalization program is merely a transitionalization program: the patient frequently bounces from one doctor's delegated line of authority and responsibility to another's. The basic problem of medical responsibility in institutions persists; only the setting has changed.

Viewed in the transinstitutional context, then, it is probable that there has been no decrease in the institutionalized patient population. On the other hand, there is a serious person-power problem with regard to psychiatric providers. There may or may not actually be a decreasing average number of psychiatrists in community mental health centers (7, 8). More important than the actual number of psychiatrists per institution, however, is the question of what these psychiatrists do while there and the nature of their contact with patients. Often they are squeezed into rapid-fire drug dispensing roles. Other times they or their signatures are used to "back up" the activities of other mental health professionals. Frequently they must interrupt what they are doing to handle emergencies that arise. Although the number of psychiatrists per center remains low, the number of other mental health workers for whom they are providing "backup" has skyrocketed. Because of budgetary considerations, the educational training and professional standards of these workers can be expected to settle lower and lower, and the need for even more careful supervision should increase.

In actuality, "backup" often means providing signatures and being relegated to the position of medicator while other personnel are designated "primary therapists." However, when the patient becomes disturbed, the so-called primary therapist requests or demands that the psychiatrist quell the disturbance with drugs and then retreat into the background. These same "primary therapists" sometimes view us with contempt, seeing us as mindless pill-pushers while they are doing the therapy. Because we have been pushed into "backup" positions with medical and medication responsibility for a large number of patients, we may not have time to be more than pill-pushers. It is distressing to see psychiatrists having to sacrifice quality for quantity in institutions in which other professionals can control their caseloads to protect the quality of their practices.

This situation has led to a vicious circle: fewer psychiatrists are attracted to work in these institutions, and those who are attracted quickly "burn out" (9). In my opinion, this phenomenon is not due to lack of exposure or training during residency years but, rather, to the type of psychiatry one is compelled to practice in many large institutions and the discrepancy between the responsibilities heaped on the psychiatrist and his or her ability to meet these responsibilities. Thus there are fewer and fewer available psychiatrists in institutions, which makes psychiatric positions even more unattractive. As funding becomes more scarce, we can expect the psychiatrist's role to be even further circumscribed and even less attractive.

The Psychiatric Education Branch of NIMH is attempting to meet this situation by targeting service to minority communities as one of the priority training areas to be funded. I predict that this strategy, however well-intended, will not work. Psychiatric residents, channeled into mental health centers by residency programs dependent on NIMH funding, will quickly learn the lessons to which I refer in this paper. Exposure to situations in which true medical responsibility is not feasible is a form of aversive conditioning.

THE TWO-TIER HEALTH DELIVERY SYSTEM

What we see in large psychiatric institutions is an example of a two-tier delivery system, with one set of practices and ethics for the wealthy and another for the poor who use institutionalized settings (5). This is nothing new; we have always had a two-tier delivery system in the United States, and in a tight-money economy, the gap between the two tiers may widen. I doubt that national health plans or other third-party payment systems will raise the quality of service for institutional patients to the level available to the wealthy. Already we see the tendency to limit the mental health benefits covered by third-party payers. Third-party payment systems, however valuable, are governed by institutional rather than personal points of view.

What, then, are the options available in such a situation? For one thing, we might do nothing. As a profession we might continue on this dismal course. Such a course is fraught with some danger because as long as our practice deviates from our stated ethical principles we lay ourselves open to further litigation.

A second possibility is that we might promote such litigation in the hope of forcing the hand of the state or federal government to fund mental health. I do not think this course will be productive. Patients' rights will come squarely up against Proposition 13, and I doubt that legislators will resolve this dilemma in favor of patients' rights. On the contrary, alternate and cheaper delivery methods will be devised, often by those who have no real knowledge of the service to be delivered. We may enter an era of even further transinstitutionalization.

As a profession we might lobby very actively for adequate funding. I am sure that we are doing this already, but, for some of the reasons I have just enumerated, I am not optimistic that this will be productive.

A fourth possibility is that we might drop out of this losing game. As a profession we might urge psychiatrists not to work under the unethical conditions required in many institutions. This will certainly keep us pure and help us avoid being sued; however, society will not respond by increasing funds for mental health delivery. Society, particularly in a tight-money economy, has shown time and again that mental health is not its highest priority. Our role in institutions will be taken over by doctors of pharmacy, nurse practitioners, and physician assistants without our being given even a supervisory role that would use these personnel as physician-extenders.

A fifth possibility, which I am thinking very seriously about, is that we should change the ethics. Ethics that speak to the ideal but are impractical are counterproductive. It may be that the guidelines of our profession represent an ethic of plenty while we are in an arena of scarcity. I suggest that if we attempted to rewrite our ethical precepts in line with what is feasible in large institutional settings, we might accomplish two things. In the first place, we would drop an unwitting pretense of a standard of practice and call attention to the fact that laws for the healing arts must be revised to conform with realities. This will make us less vulnerable to lawsuits. In the second place, as we attend to the details of this rewriting we will be forced to develop an alternate method of delivery of services in institutions that will probably place more reliance on physician-extenders.

Rewriting the ethics is not so absurd as it may sound. In July 1979 the AMA House of Delegates approved for distribution a new set of ethical principles, which, I am told, is more in tune with the social and legal tenor of the times. These were to be discussed in July 1980; at the time this paper was written they were not yet official.

Although they stress our social responsibilities, the new ethics contain none of the specific prohibitions I cited above. So far as I can see, the only principle of the new code relevant to those prohibitions states that we must provide "medically competent service." This need to be on safer legal grounds is tragic testimony to the litigious society that has evolved, but these are not the guidelines I seek. I suggest guidelines that spell out the two-tier method of practice.

It might be well argued that my suggestion merely ratifies the two-tier delivery system. I would agree with that assessment, but we have in fact a two-tier system, and there is every indication that it will continue. I do not personally defend the morality of a two-tier system or a two-tier ethic, but (to borrow a phrase from commitment law) when we deal with the medically poor we may have to resort to the least immoral alternative. A high-standard ethic that cannot be adhered to and that leaves actual practice without guidelines makes less moral sense to me than a fallback position that defines reasonably well-thought-out guidelines for second-class treatment.

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Comment

BY FREDERICK J. STODDARD, M.D.

I disagree with Dr. Bursten's two conclusions: 1) that the *Principles of Medical Ethics with Annotations Especially Applicable to Psychiatry* (1) should be revised to allow for the impossibility of ever giving the best care in some settings and 2) that we have no other effective means to improve psychiatric care for the poor. I agree with him that we are faced with serious ethical and clinical dilemmas. He deserves our gratitude for bringing this topic, which plagues us all, out in the open and for presenting his unusual and radical options. The evidence for my contrasting viewpoint is drawn from my clinical and peer review experience, pertinent articles, and documents that present the emerging position in psychiatry as I understand it. The following vignette helps illustrate my position:

Ms. A, a nurse, sought a child psychiatric evaluation for her 12-year-old son, Jimmy, who appeared hyperactive with questionable neurological impairment. She was referred to Dr. Z, who informed her that he did not have the time to examine the boy but referred Jimmy to a qualified social worker with whom Dr. Z worked. Dr. Z never saw Jimmy. The social worker evaluated the boy and his mother and recommended child psychotherapy and parent counseling. Ms. A refused the recommendation and refused to pay the psychiatrist when he billed her for his services. She appealed the case to a psychiatric peer review committee, which had no criteria by which to rule.

This type of supervision question has arisen often. In 1978 Blue Shield of Massachusetts asked the Massachusetts Psychiatric Society to prepare guidelines for supervision of "physician's assistants" (2); these guidelines were passed after two other psychiatrists and I labored over them for months. They were later revoked on the advice of Dr. Alan Stone due to risks of litigation. Although no doubt the safest legal course, our revocation of responsibility in this area has left Massachusetts psychiatrists without standards of care for supervision of nonmedical therapists, which encourages confusion or misrepresentation, as might apply in the case of Jimmy, and abuse and fraud in other cases.

Many emerging trends within psychiatry converge on the responsibility of psychiatrists as physicians. In caring for our patients, to what degree can we ethically delegate—or not delegate—responsibility for psychotherapy, medications, consultations, seclusion, etc.? Where does the role of the psychiatrist begin and that of the family physician, internist, pediatrician, or surgeon end? Where does the psychiatrist's role properly end and that of the psychiatric nurse, social worker, psychologist, or other nonmedical therapist begin? How can the psychiatrist best integrate and use new psychobiological data from psychopharmacology, developmental psychology, and psychoanalysis? What legal responsibilities and risks are involved in each area? What professional standards are to be expected of all psychiatrists as physicians? Are our medical diagnostic skills particularly needed for *DSM-III*? If we delegate too much, or vaguely, how does that affect our patients' care and weaken our position with third parties and with the drafters of national health insurance legislation? If we do not become more skilled in administration and delegation of responsibility, are we not leaving the mental health administration of health care systems to nonphysicians? These are a few of the wider issues that pertain to the responsibility of psychiatrists, in addition to the limited but major problem in institutions.

In his paper Dr. Bursten concludes by suggesting an apparently sensible option that psychiatric medical ethics be revised to relieve us of unrealistic ethical, medical, and legal responsibilities. He suggests a two-tier method of practice that would require us to "rewrite our ethical precepts in line with what is feasible in large institutional settings" to make them "conform with realities," "make us less vulnerable to suits," and force us "to develop an alternate method of delivery of services . . . that will probably place more reliance on physician-extenders." This view is opposed to the existing *Principles* (1), to established professional standards of care, and to new guidelines being written by APA. Although rewriting our medical ethics might relieve us of onerous responsibilities, it seems to me that it could be devastating to psychiatry and to our patients at a time when there is promise for improved quality and standards of care in all settings. Rather than retreating from medical responsibilities or rewriting our ethics, I think a better option is for us to understand and adhere to them more clearly than before, whether in private practice, academic institutions, or federal or state mental health systems.

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The assertion that we must restrict our medical responsibility is heard from many quarters. Other mental health professionals and even other physicians voice such views. There are increasing demands for certification and for extensive documentation that psychiatric and other medical services meet certain standards within certain cost limits. These demands come from our own professional associations, Professional Standards Review Organizations (PSROs), governmental agencies, third parties, and the legislative and judicial systems. We must renew our licenses annually both to practice and to prescribe, be certified by the American Board of Psychiatry and Neurology, earn certificates in continuing education for licensure and Association membership, and have our fees and services reviewed by peers, the public, and third parties. The fact of "absolute confidentiality" is extinct except for those patients who pay cash for their own outpatient care. What procedures we perform and how and for what fee are now influenced by executives, legislators, judges, and even the Federal Trade Commission (3). Under such regulations, it is hardly surprising that many physicians have sought less medical responsibility—either to acknowledge new realities or in reaction to lawsuits.

However, this is not the predominant direction our profession is taking today, nor, in my view, should it be. I will cite examples in addition to the *Principles of Medical Ethics* to illustrate my sense of the direction psychiatry is taking today regarding medical responsibility.

NEW ETHICAL AND LEGAL GUIDELINES FOR PRACTICE

APA is revising its position statement on relationships with nonmedical therapists (4). At its meeting in June 1980 the Board of Trustees accepted a document titled "Guidelines for Psychiatrists in Consultative, Supervisory, or Collaborative Relationships with Nonmedical Therapists" with the additional prefatory statement that the guidelines were not an official policy of APA but "a living document to be adapted to local custom and practice." These guidelines merit a place in this dialogue and are the most current statement on this important topic. They represent the results of extensive work by the Washington Psychiatric Society, the late Dr. Warren Williams, and many others. They are specific about the relationship referred to in Dr. Bursten's paper, supervisory relationships between a psychiatrist and a nonmedical therapist:

> In a supervisory relationship the psychiatrist retains direct responsibility for patient care and provides professional direction and active guidance to the therapist. In this relationship the nonmedical therapist may be an employee of an organized health care setting or of the psychiatrist. The psychiatrist is clinically responsible for the initial workup, diagnosis, and prescription of a treatment plan, as well as assuring that adequate and timely attention is paid to the patient's physical status and that such information is integrated into the overall evaluation, diagnosis, and planning. The psychiatrist remains ethically and medically responsible for the patient's care as long as the treatment continues under supervision. The patient should be fully informed of the existence and the nature of any changes in the supervisory relationship.

The APA guidelines are especially clear about the psychiatrist's ethical responsibility under the heading "Appropriateness of Care":

> It is unethical for the psychiatrist to continue an administrative supervisory, consultative, or collaborative relationship if it is ascertained that his or her role is being misrepresented, or when convinced that the care being provided is either inappropriate or inadequate. An exception may be made when the psychiatrist is specifically accepting a responsibility with the goal of improving the quality of care so that it does become adequate. The psychiatrist with an ongoing administrative supervisory, consultative, or collaborative relationship should undertake such relationships with a therapist only if he or she is able to keep himself/herself appropriately informed of the nature of treatment and the progress of those patients about whom he/she is supervising, consulting, or collaborating, and can assure himself/herself that the treatment provided is being carried out competently and adequately.

Although these are general guidelines for the psychiatrist's use rather than ironclad rules for ferreting out fraud and abuse, they represent progress within the profession. They are consistent with other steps being taken in defining psychiatry's role in the 1980s, and some psychiatrists advocate even clearer guidelines, including such details as how many nonmedical therapists one psychiatrist can supervise and improved documentation by psychiatrists in the records of all patients for whom nonmedical therapists are participating in care. It is noteworthy that no mention of the credentials of the nonmedical therapists is made—this is due to risks of litigation for restraint of trade, but it also makes it more difficult for psychiatrists advising hospitals, third parties, and agencies to make recommendations regarding standards of care.

Two relatively recent court cases are also illustrative of psychiatry's position. The first is the *Virginia Academy of Clinical Psychologists v. Blue Cross of Virginia and Blue Shield of Virginia* (5). APA's amicus curiae testimony, given by then President-Elect Dr. Donald Langsley, clearly defined his conception of the medical training and responsibilities of psychiatrists for medical treatment of nervous and mental disorders as distinct from nonmedical therapists:

> Langsley: I might refer a patient to a psychologist for psychological therapy or psychotherapy once a diagnosis has been made and a treatment plan devised.

Question: Would you continue to supervise a clinical psychologist in the treatment of one of your patients?

Langsley: I would continue to consult with the psychologist. Forgive me if I use the euphemism of consultation. I think it is often more acceptable. But in essence, I could continue to feel that I was taking ongoing medical responsibility and that the psychologist would be taking responsibility for doing the psychotherapy in that kind of situation. Though he and I might confer regularly and periodically, I personally would want to see the patient face to face to assure myself that there was no change in the condition of the patient. In some cases I might work with the psychologist. I would see the patient regularly for chemotherapy while the psychologist was doing individual or group or family therapy.

Question: Is it in the best interests of a patient who has exhibited a nervous or mental symptom to be examined by a physician before commencing psychotherapy?

Langsley: In my opinion, it is. The way to guarantee the best possible evaluation and treatment of a patient with a mental disorder or with a nervous or mental symptom is to have a thorough evaluation by a psychiatrist or by a group of people with a psychiatrist as part of the team. The reason for the psychiatrist's involvement is first of all, to be sure that any physical disease or related physical disorder is diagnosed, and, also, to be able to make a plan of treatment which would provide the best possible treatment of the patient. That might include psychotherapy, but it might also include medication, physical treatment such as electroshock therapy in a small number of cases, and also might include hospitalization if indicated. In addition, in my opinion, the best type of treatment requires having the psychiatrist available not only for the initial evaluation but for ongoing consultation if the therapist is a nonmedical person. There is a much broader scope of treatment psychiatrists can give in the field of nervous and mental disorders than is permitted by psychologists, clinical or otherwise. The psychiatrist is a physician who is trained in all of the skills of medicine, and who in the specialty of psychiatry is trained in a number of areas that are not part of the training or skill of the clinical psychologist.

Dr. Langsley and APA supported no two-tier method of practice but, on the contrary, supported a system in which outpatient psychological services could be rendered only when these services were ordered by, supervised by, and billed through a physician. The Virginia situation represents an extreme and unusual example, but it is more consistent with psychiatry's position today.

A different situation is the *Rennie v. Klein* case in New Jersey (6), in which several physicians and the state mental health department were sued for violation of the patients' civil rights in the ways in which orders were written for forcible administration of medications by nonphysician staff. In this case psychiatry's position has been that inadequate state funding led to the inadequate conditions but that the psychiatrists were working there to improve the conditions and had the right to medicate committed patients involuntarily. Nevertheless, as Dr. Bursten suggests, one result of that type of suit has been to force psychiatrists out of state mental health systems because of legal liability, which has the effect of lowering the quality of care.

A third situation has also placed the issue of supervision of nonmedical therapists squarely in the limelight: peer review and consultation to third parties, such as the Massachusetts Blue Shield situation referred to above. There are repeated questions from various third parties as to what supervision means, especially such specifics as hours of supervision per week, the number of nonmedical therapists a psychiatrist can supervise, the credentials of those therapists, and billing practices. The answers are critical to provision of adequate and competent psychiatric treatment; yet, because of the threat of lawsuits, no answers are provided. These questions will not disappear so long as psychiatrists supervise nonmedical therapists *and* sign their names on bills for their services or supervise them in institutions. One possible option is to develop more specific guidelines for supervision. Our colleagues in such specialties as surgery, internal medicine, pediatrics, anesthesiology, and ophthalmology all have "assistants" and specific guidelines for supervision but not for a double standard of care. If we are to supervise nonmedical therapists and be requested by them to do so, shouldn't we have specific supervision guidelines as well?

TOWARD CONSISTENCY OF PRINCIPLES AND PRACTICE

Although I disagree with the means Dr. Bursten suggests, I agree on the end: improved psychiatric services for the chronically ill in institutions and for others too poor to afford them. Dr. John Talbott (7) has studied these problems in depth and has clearly and concisely described the possibile outcomes, some of which are grim. Indeed, there are no easy solutions. Community mental health is out of fashion as a federally funded priority, and it is mainly through such programs that the poor have had access to psychiatric care of good quality in the last 15 years.

I see several options for improved services, and I am cautiously optimistic about them. The first is eventual universal psychiatric coverage through flexibly designed private and public national health insurance plans. The second is continued advocacy of criteria and standards of high quality. These have been described by Nelson (8) as clinical (e.g., APA criteria sets and PSROs), practitioner (e.g., certification), program or facility (e.g., the Joint Commission on Accreditation of Hospitals), and payment (e.g. CHAMPUS, Blue Cross and Blue Shield, and Medicaid) standards.

A third option is advocacy on behalf of our patients through the judiciary for funding of appropriate serv-

ies. A fourth is limited efforts in Congress to extend services for those most in need, such as children and the elderly. Finally, we have the option of continued careful work within the federal and state bureaucracies for improved standards for the wide range of federally funded programs (e.g., military, NIMH, Medicare, Medicaid, Federal Employees' Health Benefits Program, CHAMPUS, and maternal and child health programs). It appears to me that we have more, not fewer options for improving treatment for our patients than we have had in the past. The result of our efforts may well be a downgrading of quality of care in our excellent treatment settings due to intrusions into patient care from without but an upgrading of care in our poorest treatment settings due to advocacy and oversight from within and without.

In conclusion, I find Dr. Bursten's examples and deep concerns about inappropriate delegation of responsibility down-to-earth and forthright. However, the direction toward which I feel such sound observations should lead is not an endorsement of a double standard of care but positive steps toward one equitable standard.

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EDITORIAL

Are Books and Readers Facing Extinction?

> For books are not absolutely dead things, but do contain a potency of life in them to be as active as that soul was whose progeny they are. . . .
>
> Where there is much desire to learn, there of necessity will be much arguing, much writing, many opinions; for opinion in good men is but knowledge in the making.
>
> —John Milton
> *Areopagitica*

Pessimistic Cassandras have been warning for years that the art of reading books may be dying. The Common Reader of 50 years ago may be becoming as rare as the dodo, malnourished by a diet of intellectual fast food in the form of TV programs and glossy magazine articles, his taste and discrimination atrophied through contact with the emptiness of popular culture, made mentally sterile by the flood of polluted prose dumped in his environment, depressed and confused when intellectual effort is required and instant gratification must be delayed, wheezing misspelled clichés as his farewell benediction.

A readership survey conducted recently confirms this concern: the book reviews were the least read regular section of the *Journal*. Of course, low interest in the book section may indicate that the respondents were more interested in reading books outside the field of psychiatry and were doing so. But it may also reflect a feeling that books in general are not very important.

Although psychiatrists have generally prided themselves on being among the most literate and intellectual of the medical specialists, the pressures to turn our attention away from books are strong. As psychiatry becomes progressively more scientific, books may seem to convey less important or significant information than articles in journals. Books take longer to write and produce, and they therefore necessarily embody knowledge that accumulates more slowly and is less timely. Books cannot be used to communicate recent scientific advances because of this lag time. The clinician or researcher who wants to stay in touch with these advances can usually learn of them only through reading articles, attending meetings, or discussing them with his or her colleagues.

So why read books? Has the scientific emphasis in psychiatry made books outdated? The answer must be a categorical no. Books will continue to have many values and purposes in psychiatry and other scientific disciplines. Books are a large, rich, and varied medium for communication and expression. Textbooks and surveys are used to summarize composite knowledge. Books can present larger masses of data than can a single article and are used to report the results of large research projects. Sometimes the medium of the book is used to present many facets of a single problem in the case of the multi-authored monograph. Perhaps most important of all, books are used to present ideas or theories on a large scale. To ignore books is to limit one's perceptions to the microcosm and to miss the existence of the macrocosm.

This issue of the *Journal* begins some changes that we hope will highlight the importance of books and draw our readers' attention to them again. The book review section has been renamed *Book Forum* to emphasize the importance of books as a means for expressing and debating ideas. We hope to make it truly a forum, a place where there is much arguing and many opinions, a place to which readers will wander to find new ideas discussed. We will try to include both books and reviewers that reflect a diversity of points of view.

The new *Book Forum* will also include some changes in the types of books reviewed. The old staples such as textbooks and scientific monographs will of course continue to be covered. Sometimes these will be reviewed at length, and at other times briefly, depending on the complexity of their contents, the breadth of their appeal, their size and potential impact on the field, and their overall quality and merit. A new heading, Brief Mention, will be used for shorter reviews to call the readers' attention to worthwhile books that may not require a detailed review. From time to time, we will also try to have reviews of books that represent less standard intellectual fare and will appeal to epicurean tastes. We will reach into such fields as intellectual history or art or literature and try to identify exciting new books of interest to psychiatrists that they might otherwise miss. Carl Schorske's *Fin de Siècle Vienna,* reviewed in this issue, is an example of such a book. Sometimes we will also review books written about issues related to psychiatry but directed to more general readers, in order to explore how our field is perceived and described in popular culture.

Books are the major repository of our civilization's ideas. Psychiatry has by tradition been a profession "of the book," devoted to examining these and other artifacts produced by the object of its science, the human mind. As psychiatry's methods become more quantitative and objective, psychiatrists may feel tempted to ignore the larger and perhaps more blurry view of the mind conveyed by books and to value only a microscopic, precise, detailed, and factual view. As it struggles to become more scientific, psychiatry should not permit its ties to a tradition of learning and culture to become extinct. Even the microscope gains its power by enlarging what we see, not by reducing it.

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BRIEF COMMUNICATIONS

Use of a Self-Report Symptom Scale to Detect Depression in a Community Sample

BY JEROME K. MYERS, PH.D., AND MYRNA M. WEISSMAN, PH.D.

The authors gave the CES-D, a self-report depression symptom scale, to 515 people drawn from a longitudinal community survey. The subjects were also interviewed using the Schedule for Affective Disorders and Schizophrenia (SADS). From the information collected on the SADS, the subjects were given diagnoses based on Research Diagnostic Criteria. The results indicate a modest relationship between self-reported symptoms of depression and the diagnosis of a major or minor depression. However, the groups defined as "cases" by such reports also include many people with other diagnoses or with no diagnoses at all. Thus, symptom scales are useful for the screening of depressed persons in research studies but are only rough indicators of clinical depression in the community.

In recent years self-administered questionnaires have been used with increasing frequency to determine the incidence and severity of depressive symptoms in the community (1–11). Self-reports are economical, do not require extensive rater training, and avoid the problem of observer bias. However, the relationship between self-report symptom scales and psychiatric diagnosis is unclear, and it is uncertain how useful self-report depression symptom scales are as screening instruments for depression.

Most symptom scales treat psychiatric illness as a unitary phenomenon of varying severity without distinguishing among discrete syndromes. This concept differs from that of the clinical diagnosis, in which symptoms are not necessarily linear and additive but are instead classified into dimensions or categorical groups. This difference holds whether the symptom scale measures all psychiatric illness or a particular set of symptoms, such as depression.

Differences in determining what constitutes a case have led to a gap between epidemiologic and clinic studies. Research based on a diagnostic approach has relied heavily on patient populations and ignored community populations. Community studies have used quantitative measures, usually self-report scales, and have reported severity of symptoms but not diagnosed illness. Consequently, little is known about the relationship betwen symptom scale ratings and clinical diagnosis in community populations.

A major reason for this lack of information has been the unavailability of diagnostic instruments suitable for large-scale community studies. However, techniques to improve the reliability and validity of psychiatric diagnoses have now been developed and applied to community populations. This permits comparisons of the results of diagnostic evaluation and symptom scale rating within a community. The present paper presents data on such a comparison for depression in a community study in New Haven, Conn.

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METHOD

Rating and Diagnostic Procedures

The CES-D. In 1971 the Center for Epidemiologic Studies (CES), National Institute of Mental Health, began a series of studies to develop techniques for the continuous measurement of psychiatric impairment, particularly depressive symptomatology, in the community (1). As part of this research, a 20-item self-report rating scale, the CES-D, was developed to measure depressive mood (2, 7). All items were selected from previously developed scales to represent major symptoms in the clinical syndrome of depression, as identified by clinical judgment, frequency of use in other questionnaires for depression, and factor analytic studies (12). Major contributors to the CES-D scale were selected from the Minnesota Multiphasic Inventory (13) and from depression scales developed by Zung (14), Beck and associates (15), Raskin and associates (16), and Gardner (unpublished). Scores on the CES-D were found to have a correlation of .90 with scores on the Zung scale and .81 with those on the Beck scale in a study of recovered depressed patients (17).

The SADS-RDC. A technique for making psychiatric diagnoses in a community as well as a patient population has been developed by Spitzer and associates (18) as part of a collaborative project in the psychobiology of the depressive disorders sponsored by the Clinical Research Branch, National Institute of Mental Health. Their techniques attempt to correct for unreliability due to criterion variance (the most serious problem in such research) as well as to correct for unreliability due to subject, occasion, and information variance.

Criterion variance is handled by the use of Research Diagnostic Criteria (19) to establish formal diagnostic definitions. Subject, occasion, and information variance have been approached by the development of a structured interview guide and accompanying inventory of rating scales and specific items, the Schedule for Affective Disorders and Schizophrenia (SADS) (20). This instrument is used to record data on the subject's functioning and manifest symptomatology and contains all the information necessary for making the RDC diagnosis.

Diagnostic assessment. There are several versions of the SADS based on the time period assessed—current, past five years, or lifetime. We used the lifetime version, which also includes an assessment of the subject's current status.

Based on the information collected on the SADS, the subjects were classified by the RDC. Raters underwent a three-month period of training on the SADS and RDC, and interrater reliability following training was excellent. (Information on training, reliability, and agreement with clinical records has appeared in detail elsewhere [21].)

TABLE 1
Relationship Between RDC Diagnosis of a Community Sample of 482 Individuals and Their Rating on the CES-D[a]

	CES-D Rating			
	Depressed		Not Depressed	
RDC Diagnosis	N	%	N	%
Depressed (N=22)	14	63.6	8	36.4
Not depressed (N=460)	28	6.1	432	93.9

[a]Data were missing for 33 individuals.

Subjects

The sample consisted of 515 respondents interviewed in 1975–1976 as part of a longitudinal survey begun in 1967 of a community mental health center catchment area in New Haven, Conn. (22, 23). The area has a population of approximately 72,000, which includes a changing inner-city section of 22,000 and a more stable industrial town of 50,000. It represents an ethnic, racial, and socioeconomic cross-section of the community's population.

The original sample consisted of 938 adults 18 years of age or over chosen at random from a systematic sample of 1,095 households. This number dropped to 720 in the second wave of interviews in 1969 and to 515 in 1975–1976 because of death, refusal to participate, and movement out of the area. Full methodologic details have been described elsewhere (21).

RESULTS

The CES-D and Major Depression

Table 1 shows relationships between scores on the CES-D and a diagnosis of major depression based on the SADS-RDC. Use of the traditional CES-D cut-off point of 16 and above as defining a case led to a false positive rate of 6.1% and a false negative rate of 36.4% (see table 1). Recently, Husaini and associates suggested that cut-offs of 17 and 23 provide better estimates of "possible" and "probable" cases (24). The cut-off of 17 for a probable case produced a very similar false positive rate of 5.6% and the same false negative rate (36.4%). The cut-off point of 21 (a definite case) produced corresponding rates of 4.1% and 45.5%.

The CES-D and Minor Depression and Depressive Personality

The false positive rate for current minor depression of 8% at the 16-point CES-D cut-off was similar to that for major depression. However, the false negative rate, 77%, was twice as high. For depressive personality, the false positive rate was 7% and the false negative, 57%.

TABLE 2
Distribution of Current RDC Diagnosis of a Community Sample of 482 Individuals by Their CES-D Rating

	CES-D Rating			
	Depressed (N=42)[a]		Not Depressed (N=440)[a]	
Current RDC Diagnosis	N	%[b]	N	%
Major depression	14	33	8	2
Minor depression	3	7	10	2
Depressive personality	3	7	11	3
Bipolar disease	0	—	1	—
Schizophrenia	0	—	0	—
Anxiety	4	10	7	1
Briquet's syndrome	1	2	1	—
Alcohol or drug abuse	1	2	9	2
Phobia or panic	1	2	4	1
Personality disorder	1	2		
Other	0	—	4	1
No diagnosis (not currently ill)	14	33	385	88

[a]N here refers to subjects. For subjects who had more than one current diagnosis (N=22) depression was counted if it was one of the diagnoses. If one of the multiple diagnoses was not depression the most severe diagnosis was counted.
[b]Percentages do not add up to 100 because of rounding error.

Persistent Symptoms and Major Depression

Craig and Van Natta (25), who also used CES-D, have suggested that the persistence of symptoms may be more important than their prevalence for screening devices. They suggested that a scale based only on persistence of symptoms for 5 to 7 days before interview was the most efficient way to identify individuals at high risk for the depressive syndrome, while minimizing the likelihood of contamination by individuals reporting a large number of transient symptoms.

We examined our data by determining whether one or more symptoms occurred for 5 to 7 days. The false positive rate of 16.3% and false negative rate of 31.8% we found were very similar to those obtained when the 2-week prevalence rates were used for the CES-D total score.

Diagnosis of False Positive and False Negative Cases

Table 2 shows the distribution of diagnoses on the RDC by the respondent's CES-D status (where scores of 6 and above indicate a case). Although depression was the most frequent RDC diagnosis for subjects with high CES-D scores, 18% of such respondents had other diagnoses and 33% had no psychiatric diagnosis.

DISCUSSION

The results of this study demonstrate that a self-report symptom scale can be used as a screening device to identify cases of major depression for research purposes. For example, only 6% of the cases not diagnosed as major depression by RDC were so classified by the CES-D (false positives). However, the instruments examined are not efficient in identifying cases of minor depression or depressive personality in the community.

These instruments are far less effective for clinical purposes; about one-third of the cases of RDC-diagnosed major depression were missed by the symptom scales (false negatives). If we change the cut-off point so that the false negative rate declines significantly, the false positive rate increases substantially. Consequently, a large percentage of the population identified in this way would not actually be "cases." If screening scales are to be used in clinical settings such as primary care or medical clinics to detect persons who may require treatment for depression, much lower cut-off scores would be required to reduce the false negative rate. For example, a cut-off score of 5 would give a false negative rate of 0 for major depression. However, there would then be a 30% false positive rate.

It is clear that self-report screening scales can identify many persons with a diagnosis of depression in the community. However, the group defined as "cases" by such instruments also includes many people with other diagnoses or with no diagnoses at all. Thus the screening devices are rough indicators of clinical depression in the community, but diagnostic instruments should be used if one is interested in determining the rates of diagnosed illness. Nonetheless, these instruments are extremely useful for screening depressed persons in research studies.

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A Comparison of Unipolar and Bipolar Depressive Illness

BY RICHARD ABRAMS, M.D., AND MICHAEL ALAN TAYLOR, M.D.

In a study of 40 consecutively hospitalized patients with research diagnoses of endogenous depression, the authors found no difference between unipolar and bipolar depressive patients in the risk for affective disorder in first-degree relatives, proportion of EEG or neuropsychological abnormalities, clinical evidence of the depressive syndrome, or response to doctor's choice of treatment. Bipolar patients had an earlier age of onset and displayed more manic symptoms than did unipolar patients. The authors conclude that the two forms of depressive illness are clinically and genetically homogeneous, are without identifying EEG or cognitive differences, and have an equally good response to somatic treatments.

In an earlier study (1) of unipolar and bipolar endogenous depressed patients referred for ECT, we found no differences between diagnostic groups in the severity of illness, clinical psychopathology of the depressive syndrome, or response to ECT. The group of bipolar patients had onset of illness at an earlier age, a larger number of women, a greater incidence of cyclothymia and endomorphy, and a greater genetic loading for affective illness (but not for alcoholism and sociopathy). We concluded that although our data were consistent with the concept that unipolar and bipolar depressive illnesses were separate biological entities, they were a homogeneous depressive syndrome and had a uniform response to ECT.

The present study again compares unipolar and bipolar depressive patients, but the current sample was drawn from consecutively admitted patients and was studied on a much larger range of variables.

METHOD

The present study was done on an acute treatment, university psychiatric inpatient unit serving a suburban-rural population in New York state. All consecutively admitted patients were included for the 22 months ending May 1976. Each patient was interviewed by one or the other of us with a psychiatric resident who was specifically trained in principles of phenomenology and data collection and who followed

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a semistructured outline to collect the necessary clinical and demographic data. At the time of the patient's discharge, a summary was prepared that included all information and that was meticulously reviewed to ensure accuracy and completeness of data.

After all data collection was completed, research diagnoses were made by one of us (R.A.), who was given the patient's chief complaint, history of present illness, and mental status portions of the research records and who was blind to all identifying information and all demographic, family history, and treatment response data.

The criteria for endogenous depression (1 through 3 required) were 1) sad, anxious, or dysphoric mood; 2) three of the following: early morning waking, diurnal mood swing (worse in the morning), weight loss of more than 2.3 kg in three weeks, retardation or agitation, suicidal thoughts and/or behavior, and feelings of guilt, hopelessness, or worthlessness; and 3) no diagnosable coarse brain disease, no use of steroids or reserpine in the past month, and no medical illness known to cause depressive symptoms.

The severity of the patient's illness on admission and at discharge was determined separately in the same manner as the diagnoses, using a scale with half-step increments in severity ranging from "not ill" (rating of 0) to "most severely ill" (rating of 4).

Family Illness

We obtained complete family history information from each proband and at least one first-degree relative (if available). If these two sources agreed that the family history was entirely negative for mental illness, no further interviews were generally obtained. If either source indicated the presence of an ill relative, an attempt was made to interview all available first-degree relatives, who were defined as those living in the United States and available for personal or telephone interview. All interviews were conducted by a research fellow without knowledge of the proband's research diagnosis. The semistructured form that was used included assessment of present and past psychopathology.

We collated the family interview protocols and all family history and interview data from the research summary, removed all identifying information, and then presented these data to a "blind" examiner who made research diagnoses in the first-degree family members. Diagnostic criteria for present psychopathology were the same as for proband diagnoses.

Diagnostic criteria for past depressive illness in relatives (1 through 4 required) were 1) an illness of at least two weeks' duration; 2) sad, anxious, or dysphoric mood; 3) at least one of the following for probable diagnosis and two for definite diagnosis: insomnia, anorexia, weight loss of more than 2.3 kg, feelings of hopelessness, feelings of guilt and/or self-blame, suicidal ideas (consummated suicide plus items 1, 2, and 4 were considered definite evidence of depressive illness), loss of interest in daily activities, and feeling slowed down; and 4) treatment by a physician with medication or ECT, with much improvement or recovery afterward.

TABLE 1
Clinical, Demographic, and Laboratory Variables for Unipolar and Bipolar Depressed Patients

Variable	Unipolar Patients (N=26)	Bipolar Patients (N=14)
Index age (years)	42.5	37.3
Onset age (years)[a]	34.0	25.4
Sex		
Male	2	4
Female	24	10
Race		
White	24	13
Black	2	1
Percent with abnormal EEG	16.7	7.7
Percent with abnormal rating on aphasia screening test	46.2	33.3
Severity of illness at admission	2.6	2.8
Duration of present illness (months)[b]	2.9	1.0
Duration of index admission (days)	48.7	30.3
Percent improved at discharge	88.3	88.1
Depression score	5.8	5.2
Mania score[c]	0.0	0.43
Catatonia score	0.34	0.43

[a]The difference between the two groups was significant ($\chi^2=3.34$, $p=.002$).
[b]$\chi^2=2.19$, $p=.035$.
[c]$\chi^2=2.48$, $p=.028$.

Diagnostic criteria for past manic illness in relatives (1 through 4 required) were 1) an illness of at least two weeks' duration; 2) euphoric, expansive, labile, or irritable mood; 3) at least one of the following for probable diagnosis and two for definite diagnosis: excitement and/or overactivity, overtalkativeness and/or rapid speech, excessive spending, excessive optimism, overestimation of own capacities, and racing thoughts; and 4) treatment by a physician with medication or ECT, with much improvement or recovery afterward.

The criteria for antisocial personality and alcoholism were those of Feighner and associates (2). For the present study, definite and probable diagnoses in relatives were combined for analysis.

Laboratory Studies

EEGs were routinely obtained for all cooperative patients and read by a "blind" examiner (R.A.) who classified them as normal, borderline, or abnormal according to the criteria of Gibbs and Gibbs (3). The aphasia screening test, a neuropsychological test of cortical dysfunction (4), also was routinely administered and read by a "blind" examiner (M.A.T.) who interpreted the results according to Heimburger and Reitan (4) and the principles of Luria (5). We have reported the detailed methodology of these procedures elsewhere (6).

TABLE 2
Patterns of Illness in First-Degree Relatives of Unipolar and Bipolar Depressed Patients[a]

Illness	First-Degree Relatives of Unipolar Patients			First-Degree Relatives of Bipolar Patients		
	Number Ill	Number at Risk	Morbidity Risk (%)	Number Ill	Number at Risk	Morbidity Risk (%)
Affective illness	13	106.5	12.21	7	47	14.89
Unipolar	8	106.5	7.51	3	47	6.38
Bipolar	5	106.5	4.69	4	47	8.51
Alcoholism	17	106.5	15.96	7	47	14.89
Antisocial personality	2	177.5	1.13	3	82.5	3.64

[a]None of the differences between the two groups was significant.

We defined unipolar depressive illness as the presence of at least one depressive episode in the absence of any history of mania or hypomania, and bipolar depressive illness as the presence of at least one manic or hypomanic episode.

Assessment of Clinical Variables

A total of 40 individual clinical signs and symptoms were coded as present (1) or absent (0); from these, three clinical scores were computed for each patient as follows:

1. Depression score (range, 0–9): the sum of depressed mood, retardation, guilt, insomnia, suicidal ideation, hopelessness, weight loss, diurnal variation, and depressive delusions.
2. Mania score (range, 0–6): the sum of hyperactivity, rapid or pressured speech, euphoria, grandiosity, intrusiveness, and flight of ideas.
3. Catatonia score (range, 0–8): the sum of mutism, stupor, negativism, posturing, catalepsy, mannerisms, echolalia or echopraxia, and automatic cooperation.

Statistical Analysis of Data

Differences between means were analyzed by independent t tests, correlations by the Pearson product-moment correlation coefficient (r), and contingency tables by raw chi-square or Fisher's exact probability test where appropriate. To test the relationship between individual clinical signs and symptoms (scored as 0 or 1) and the depressive diagnostic group (scored as 0 or 1 for unipolar or bipolar), we used the phi coefficient, a form of the product-moment correlation coefficient.

RESULTS

Of the 465 patients admitted during the study period, 40 satisfied our research criteria for endogenous depression; 26 were unipolar and 14 bipolar. Table 1 displays a variety of demographic, clinical, and laboratory variables for the two diagnostic groups. Bipolar patients were significantly younger than unipolar patients at the time of their first illness episode, had a shorter duration of their present illness just before the index admission, and had a significantly higher mania score than unipolar patients.

We examined the correlations between 26 individual clinical features and polarity of illness. Consistent with a higher mania score, bipolarity was significantly correlated with agitation and aggression (phi=.272, p=.045) and euphoria (phi=.311, p=.027) and tended to be correlated with intrusiveness, hyperactivity, rapid or pressured speech, and grandiosity ($p<.10$). Unipolar patients lost more weight than bipolar patients (phi=.340, p=.017) and tended to be more depressed ($p<.10$).

We compared the two groups on number of total hospitalizations; episodes of illness per year; and history of drug abuse, alcohol abuse, childhood hyperactivity, seizures, head trauma with unconsciousness, unexplained high fever, gestational or perinatal complications, delayed landmarks, and serious medical illness. There were no significant differences between the unipolar and bipolar patients on any of these variables.

Treatments administered to the two groups were doctor's choice and included ECT (alone or with drugs), antidepressants, lithium alone, neuroleptics alone, or lithium plus neuroleptics. The groups differed significantly in treatments received ($\chi^2=11.1$, df=5, p=.049) due primarily to the fact that more bipolar patients were treated with lithium alone (p=.01, Fisher exact probability test). The response to the various treatments did not differ for the two groups; with specific regard to ECT, the degree of improvement was 94.7% and 95.8% for the unipolar and bipolar patients, respectively.

Table 2 displays patterns of family illness of affective disorder, alcoholism, and antisocial personality for the two groups; there were no significant intergroup differences in the risk for any of the diagnoses.

DISCUSSION

We confirmed our prior finding (1) of an earlier age at onset for bipolar patients as well as a homogeneous

clinical presentation and response to doctor's choice of somatic treatments for the two groups. However, in the present study, we found no difference between groups for the risk for affective disorder in first-degree relatives. The proportion (simple prevalence) of affective illness in the relatives of the present sample was 7.7% for bipolar patients and 6.6% for unipolar patients, figures that can be compared with our prior findings of 14% and 1%, respectively. This difference may be accounted for by the improved methodology of the present study, which employed blind diagnoses of both proband and relative, as well as blind family interviews.

The higher mania scores for bipolar patients and the fact that they received more treatment with lithium suggests that manic symptoms are at times manifest in the depressed phase of bipolar illness (it was never observed in the unipolar sample), a finding that has been noted by others (7).

The shorter duration of the present illness in bipolar patients suggests that their clinical symptoms demanded earlier medical attention, perhaps due to the mixture of manic symptoms.

Our selection of the unipolar sample on the basis of a single endogenous depressive episode might bias the data in favor of homogeneity of unipolar and bipolar depression if a significant proportion of first-episode unipolar patients ultimately developed a manic attack. However, this likelihood is quite small; in a long-term follow-up study Winokur and Morrison (8) found that fewer than 5% of depressive patients followed such a course.

The present study, with its improved methodology and more extensive selection of variables, including EEG and neuropsychological testing, showed fewer important differences between unipolar and bipolar depressive patients than did our earlier report. Moreover, the differences found in the present study may be interpreted as simply a function of severity of illness (e.g., earlier age at onset, more manic symptoms) rather than as specific biological markers.

We conclude on the basis of these new data that unipolar and bipolar depressive illnesses are clinically and genetically homogeneous, are without EEG or neuropsychological differences, and have an equally good prognosis.

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Predicting Outcome of Antipsychotic Drug Treatment from Early Response

BY PHILIP R.A. MAY, M.D., THEODORE VAN PUTTEN, M.D., AND CORALEE YALE, M.S.

The authors gave 48 newly admitted schizophrenic patients an initial test dose (2.2 mg/kg) of chlorpromazine hydrochloride and additional doses at 24 and 36 hours. Clinical response was measured at 48 hours and at the end of 28 days of subsequent treatment with a fixed dose (6.6 mg/kg) of chlorpromazine. There was a consistent correlation between 48-hour change and the eventual amount of improvement by the end of drug treatment for all 10 outcome criteria. The authors conclude that change early on in drug treatment is a useful predictor of outcome.

It has been established that antipsychotic drugs generally work. We now turn to the problem of how to predict outcome for the individual patient. Although most schizophrenic patients improve if given medication, a substantial number do just as well without it and some get worse or develop adverse effects. Regrettably, controlled research on prediction of response has been meager (1), and our current state of knowledge is not impressive—in a personal communication in 1976 Hollister observed that drug treatment is unlikely to make a patient play the violin if he has never played before.

Over and above statements of the obvious, can one predict whether drug treatment is likely to help a particular person? Having failed in 7 years of painstaking research to develop methods that predict the outcome for a specific patient from pretreatment information, we concluded that we may have been trying to predict from the wrong set of variables. Our rationale that we might learn something relevant to the eventual clinical outcome by studying the patient's immediate response to the drug led to procedures for monitoring the immediate response to a test dose (2, 3). Our preliminary findings that follow are most encouraging: they indicate that measuring early clinical response may be a simple and practical aid to predicting the outcome of a course of drug treatment.

METHOD

We gave an initial test dose of chlorpromazine hydrochloride and additional doses at 24 and 36 hours to 48 newly admitted patients suffering from a functional psychosis with definite schizophrenic features but not from significant physical disease, drug addiction, or alcoholism. Clinical response was measured at 48 hours and at the end of 28 days of subsequent treatment with a controlled standardized course of drug therapy. The subjects had given informed consent in a manner approved by a human studies review committee.

As described elsewhere (4), the diagnosis was substantiated by using four different sets of diagnostic criteria for schizophrenia: the diagnostic criteria used in the National Institute of Mental Health collaborative studies (5), the Diagnostic Certainty rating scale (6), the New Haven Schizophrenia Index checklist (7), and the World Health Organization criteria for the diagnosis of schizophrenia (8). Our objective was to select patients for whom treatment with antischizophrenic antipsychotic drugs was clearly indicated, an issue that we suspect may be of greater concern to the clinician than achieving complete certainty about a diagnosis of schizophrenia. Although there is no substantive evidence that drug response is predicted better by any particular set of diagnostic criteria, whether stringent or relaxed, all 48 patients met at least two of the sets of criteria and 92% met all four.

The initial dose was 2.2 mg/kg of chlorpromazine, given as oral concentrate (100 mg/ml), diluted in approximately 22 ml of orange syrup. The same dose was repeated at 24 hours, and 4.4 mg/kg was given at 36 hours (just before bedtime). After this, a fixed daily dose (liquid form) was given once daily (between 8:30 p.m. and 9 p.m.) for 28 days, toward the lower end of

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TABLE 1
Pearson Product-Moment Correlations Between Clinical Measurements of Early (48-Hour) Change and Amount of Improvement After 28 Days for Schizophrenic Patients Treated with Chlorpromazine

Measure	r
BPRS (N=31)	.669
Thought disorder	.631
Anxiety-depression	.669
Paranoia	.537
Withdrawal	.513
MACC (N=29)	.573
Mobility	.597
Affect	.558
Cooperation	.511
Communication	.507

the generally accepted therapeutic range—450 mg/day for a 67.5-kg person (6.6 mg/kg). If there had been a severe toxic reaction to the first dose, it was reduced. We chose the lower end of the therapeutic range for ethical reasons.

Clinical status was rated by the treating psychiatrist on the Brief Psychiatric Rating Scale (BPRS) (9) (total score and four factors) and by nursing staff on the MACC Behavioral Adjustment Scale (MACC) (10) (total score and four subscales). The posttreatment ratings were independent; i.e., the raters were blind to the 48-hour values.

RESULTS

There was a consistent correlation between 48-hour change and the eventual amount of improvement by the end of drug treatment for all 10 outcome criteria. The Pearson correlations ranged between .507 and .669, all statistically significant at $p<.01$ or .001 (see table 1).

These correlations indicate that the more a patient's condition is improved at 48 hours, the better the eventual outcome of treatment with a fixed dose and vice versa. Put another way, change early on in drug treatment is a useful predictor of what will eventually be. In practice, when the patient improves rapidly, all is well and is likely to stay well; e.g., all 9 patients whose BPRS thought disorder scores improved 3 scale points or more in the first 48 hours did reasonably well at outcome. When no rapid change occurs, the clinician can wait or raise the dose. If he or she chooses to wait, our data indicate that he or she should do so with reduced expectations of improvement. The clinician is likely to worry about what to do with the patient who is worse after only 48 hours of drug treatment. We would not expect much benefit from continuing the same dose; for example, none of the 5 patients who were rated worse on the BPRS thought disorder scale after 48 hours of drug treatment went on to a very good outcome. It is difficult from our data to generalize about the effect of raising the dose of patients who are worse after 48 hours. At the end of 28 days on a fixed dose, we gave 4 of 5 such patients higher doses: 1 became even worse, 2 improved, and 1 remained unchanged.

COMMENT

We conclude that when patients get worse early on in drug treatment, this should be taken as a clear warning and a definite indication that something may need to be changed. For example, the dose could be raised, although the patient's condition must be carefully monitored and the possibility for eventual benefit is slight, or another drug could be prescribed instead. It is possible that the patient might do just as well, or even better, without drug treatment. Our data contradict the widespread belief that drugs do not affect thought disorder or, alternatively, that there is necessarily a lag period before the onset of drug effect.

Although our findings are subject to the usual caveats about preliminary findings from a relatively small number of patients, the consistency and magnitude of the correlations are impressive. It is easy in clinical practice to monitor 48-hour improvement, and we, therefore, suggest that this simple prognostic indicator be given further clinical trial. The underlying principle is very simple—if you want to know what is likely to happen on drug treatment, try a test dose.

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MHPG as a Predictor of Antidepressant Response to Imipramine and Maprotiline

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To explore the hypothesis that depressed patients with low pretreatment levels of urinary 3-methoxy-4-hydroxyphenylglycol (MHPG) respond more favorably to antidepressant drugs which act on noradrenergic neuronal systems than do patients with high MHPG levels, the authors administered 150–200 mg/day of imipramine or maprotiline to 13 depressed patients. All of the 5 patients with low pretreatment MHPG levels responded to treatment compared with 1 of the 14 patients with high MHPG levels; 4 patients dropped out of the study.

Research from a number of laboratories has indicated that pretreatment levels of urinary 3-methoxy-4-hydroxyphenylglycol (MHPG), the major metabolite of norepinephrine which originates in the brain (1–3), might aid in predicting antidepressant response to specific tricyclic antidepressant drugs. In two studies (4, 5) depressed patients with low pretreatment levels of urinary MHPG were found to respond more favorably to treatment with imipramine or desmethylimipramine than patients with higher urinary MHPG levels. In contrast, three studies (5–7) have shown that patients with high pretreatment levels of urinary MHPG responded more favorably to treatment with amitriptyline than did patients with lower MHPG levels, but this has not been clearly demonstrated in other studies (8, 9).

Basic neuropharmacological studies (10) have shown both similarities and differences between the effects of imipramine and its demethylated metabolite desmethylimipramine and those of amitriptyline and its demethylated metabolite nortriptyline on various central neurotransmitters—including norepinephrine and serotonin. This literature, which has recently been reviewed by Maas (11), suggests that imipramine produces more potent effects on noradrenergic neuronal systems than does amitriptyline.

To further explore the hypothesis that patients with low pretreatment levels of urinary MHPG will show favorable responses to antidepressant drugs which act on noradrenergic neuronal systems, in this preliminary study we examined pretreatment urinary MHPG levels as a possible predictor of response to imipramine and maprotiline, a new tetracyclic antidepressant drug that exerts potent effects on norepinephrine uptake but has little effect on serotonin uptake (12–14).

METHOD

The subjects in the study were 13 depressed patients who met the Feighner criteria (15) for primary affective disorder, unipolar type, and the Research Diagnostic Criteria (16) for major depressive disorder. All patients were free of significant medical or neurological disorders and scored at least 28 on the Hamilton Depression Rating Scale (17). The patients ranged in age from 38 to 62 years; 8 were men and 5 were women. Informed consent was obtained from all patients participating in this study.

During the pretreatment baseline period, 24-hour urine specimens were collected for measurement of MHPG, which was determined by a modification of the method of Dekirmenjian and Maas (18). Patients received no psychoactive drugs for at least 10 days before and during the collection of 24-hour urine specimens with the exception of occasional doses of oxazepam, 15–45 mg/day, which were administered to 6 of the patients for anxiety or sleeplessness. All patients

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were hospitalized during the period of urine collection and received a standardized tyramine-free diet for at least 24 hours before and during the entire period in which urine was collected. We used only complete urine collections, as determined by nurses' observations and by creatinine concentration and total volume.

After the pretreatment baseline urine collections the patients were randomly assigned to double-blind treatment with maprotiline or imipramine. These drugs were initially administered at a dose of 150 mg/day, and most patients continued to receive this dose for the first two weeks of the study (although a few patients required transient decreases in dosage because of side effects). During weeks two, three, and four of the study the protocol allowed drug dosages to be increased by 50 mg every three days, and all but 1 of the 9 patients who completed the four-week protocol and who failed to respond to treatment at a dose of 150 mg/day received a therapeutic trial of 200 mg/day during this study.

The Hamilton scale scores were obtained during the pretreatment baseline period and weekly throughout the study. Patients were considered responders if the Hamilton scale score at four weeks was less than 16 and had fallen at least 60% from the baseline value. Although patients who failed to complete the four-week protocol (dropouts) were regarded as nonresponders, wherever possible data were analyzed in two ways, including these patients as nonresponders and excluding these patients from data analysis. All clinical diagnoses and Hamilton depression ratings were made by clinicians who were blind to the biochemical data, and all biochemical determinations were performed by laboratory personnel who were blind to the clinical data.

RESULTS

Pretreatment urinary MHPG levels ranged from 1370 to 3640 μg/24 hours. This range is similar to that observed in a previous series of patients (19) with unipolar endogenous depression, in which the mean MHPG level was 1950 μg/day. In the present study 6 of the 13 patients had pretreatment MHPG levels of 1950 μg/day or less, which we shall refer to as "low" levels, and 7 of the 13 patients had pretreatment MHPG levels over 1950 μg/day, which we shall refer to as "high" levels.[1] The mean age (±SE) of the low-MHPG group was 47±8 years; the mean age of the patients with high MHPG levels was 48±8 years.

In an analysis of the data in which dropouts were considered nonresponders, 5 of the 6 patients with low pretreatment MHPG levels had a favorable response to maprotiline or imipramine. In contrast, 6 of the 7 patients with high pretreatment MHPG levels did not respond to treatment with these drugs (p=.05, Fisher exact probability test). The 1 patient with high MHPG levels who was classified as a responder relapsed after 31 days of treatment even though treatment with imipramine was continued.

Excluding patients who dropped out, all of the 5 patients with low pretreatment MHPG excretion who completed the four-week protocol showed a favorable response to treatment with maprotiline or imipramine, while only 1 of the 4 patients with high pretreatment MHPG levels showed a favorable response (p=.05, Fisher exact probability test). The mean (±SE) Hamilton scale score at baseline was 32±1 for the patients with low pretreatment MHPG levels and 36±3 for the patients with high MHPG levels. At the end of the four-week study, the patients with low MHPG levels had a mean Hamilton scale score of 10±1, and the patients with high MHPG levels had a mean score of 20±4 ($p<.05$, one-tailed t test). The mean percentage of reduction in Hamilton scale scores was 69±4% for patients with low urinary MHPG levels and 45±13% for the patients with high MHPG levels ($p<.05$). The decrease in mean four-week Hamilton scores from baseline values was greater in patients with low urinary MHPG levels (22±1) than in patients with high MHPG levels (16±4), although this difference did not attain statistical significance.

DISCUSSION

In this study low pretreatment urinary MHPG levels (1950 μg/day or less) predicted a favorable response to treatment with either imipramine or maprotiline. The incidence of and reasons for dropping out also support a differential responsivity based on pretreatment urinary MHPG levels; only 1 of the 6 patients with low pretreatment urinary MHPG levels dropped out of the four-week study, and this patient dropped out because of an allergic dermatitis. In contrast, 3 of the 7 patients with high pretreatment urinary MHPG levels (above 1950 μg/day) dropped out, and all of these patients dropped out due to the occurrence of troublesome side effects (other than allergic dermatitis) in conjunction with a lack of clinical response.

Thus, our results support previous findings which suggest that patients with low urinary MHPG levels are more responsive to treatment with drugs that inhibit norepinephrine uptake than are patients with high pretreatment urinary MHPG levels. However, since no patients in this study received more than 200 mg/day of either antidepressant drug, we are unable to rule out the possibility that patients with high pretreatment levels of urinary MHPG might be responsive to more vigorous treatment. Further studies to explore this possibility are currently in progress.

[1] Of the 6 patients who received oxazepam, 3 had low pretreatment MHPG levels and 3 had high pretreatment MHPG levels.

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Drug-Related Admissions to an Inpatient Psychiatric Unit

BY RONALD B. STEWART, M.S., PHILIP K. SPRINGER, M.D., AND JOHN E. ADAMS, M.D.

The authors prospectively monitored 60 consecutive admissions to an inpatient psychiatry service to determine the role of drugs in psychiatric hospitalization. They categorized drug-related problems as 1) drug intoxication, 2) drug abuse, 3) side effects of therapeutic drugs, and 4) noncompliance with prescribed medications. During the 6-month study 25 (41.7%) of the admissions were related to drug use; in 16 (26.7%) admissions a drug-related problem was identified as the primary cause of hospitalization. Alcohol was involved in 8 admissions, and Fiorinal, cocaine, and flurazepam were each related to 2 admissions.

The number and use of drugs that influence behavior have increased tremendously during the past 25 years. The availability of tricyclic antidepressants, neuroleptics, and anxiolytic drugs in the physician's armamentarium has dramatically affected the nature and course of mental illness in this country. Phenothiazines (1), for example, have been given credit for the dramatic decline in the number of hospitalized schizophrenic patients. Stolley and associates (2) documented the popularity of these agents in a study of drug use in an American community showing that 17% of all prescriptions were for psychotherapeutic drugs.

Advances in research on and the development of psychoactive drugs have not been without liabilities since no drug is completely safe, particularly if used improperly. The same drugs that have reduced the number of hospitalized schizophrenic patients have been associated with major side effects, including tardive dyskinesia (3). Problems related to the abuse of psychoactive drugs have in some cases reached epidemic proportions; they are perhaps most obvious in psychiatric patients, in whom alcoholism, drug overdose, and extrapyramidal side effects occur frequently.

Recently, an enhanced awareness of drug-induced illness has led to systematic investigation of this problem. Studies of hospitalized medical patients have shown that 1.7%–4.5% of admissions are due to drug-induced disease (4–8). McKenny and Harrison (9) expanded the definition of drug-induced disease to include all drug-related problems and found that 27.3% of medical patients had a drug-related problem on admission. To our knowledge similar studies of psychiatric patients have not been conducted. We conducted a prospective epidemiologic study of patients admitted to an adult inpatient psychiatric unit to delineate the role of drugs in psychiatric admissions.

METHOD

The 60 patients in our study were consecutive admissions to an inpatient adult psychiatry service over a 6-month period who were primarily referred by psychiatrists.

One of us (R.B.S.) prospectively monitored patients by attending daily staff rounds, reviewing medical records, and interviewing newly admitted patients to obtain a drug history. Whenever possible, family members and/or friends of the patients were questioned concerning the patient's drug use and drug compliance habits. When indicated, a toxicology screen on blood and urine was performed to confirm the presence of drugs. Final assessment of the effect of drugs on admission was made after the patient's discharge from the hospital.

We developed the following criteria and definitions for drug-related admissions before initiation of the study:

Drug-related admission. Patient was admitted as a direct result of drug use or because drug use contributed to the need for hospitalization.

Drug intoxication. Patient intentionally took a drug overdose to attempt suicide.

Drug abuse. Hospital admission was related to the patient's abuse of a drug or drugs not being used for therapeutic purposes. We evaluated each patient by interviewing the patient and his or her relatives, by chart review, and by information obtained from the referring psychiatrist to determine if the use of drugs such as alcohol, cocaine, or propoxyphene was related

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to the admission. We did not use strict drug abuse criteria such as physical dependence, because a patient could be abusing drugs at the time of admission and not show evidence of true dependence. Many patients had acknowledged using drugs of abuse, but if in our opinion the drug or drugs had no direct bearing on hospital admission, we did not consider the patient as a drug abuse admission.

Side effects. Hospital admission was related to adverse effects of therapeutic agents used in normal therapeutic doses (e.g., neurotoxicity from lithium or ataxia from anticonvulsants).

Noncompliance. Hospital admission was related to the patient's not following directions for prescribed medications (e.g., a patient with manic-depressive illness refusing to self-medicate with lithium or a patient abusing a prescribed narcotic).

A structured monitoring protocol was developed to collect information concerning therapeutic and social drugs used immediately before admission and to assess the severity of detected drug problems in the patient. We categorized drug-related problems into four major groups: 1) drug intoxication (overdose), 2) drug abuse, 3) side effects of therapeutic drugs, and 4) noncompliance with prescribed medications. We also noted the drugs causing the problem and specific effects of the problem. We reviewed all patient evaluation forms at the time of discharge to assess to what degree the hospital admission was related to drug use.

RESULTS

During the 6-month period of study, there were 60 admissions to the psychiatry service, 25 (41.7%) of which were related to drug use. In 16 (26.7%) admissions we identified a drug-related problem as the primary reason for hospitalization. The average age of the 60 patients admitted to the unit was 33.9 years; 24 (40%) were men and 36 (60%) were women. The average age of the 25 patients admitted with a drug-related problem was 34.3 years; 10 (40%) were men and 15 (60%) were women.

Of the drug-related hospitalizations 13 (52%) resulted from drug abuse, 4 (6.6%) resulted from drug intoxication (suicide attempts), 3 (12%) were because of drug side effects, and 5 (20%) were related to noncompliance with prescribed medication. The largest number of drug-related admissions involved abuse of alcohol (N=8). Cocaine, flurazepam, and Fiorinal were each implicated in 2 drug-related admissions. One patient each was addicted or habituated to meperidine, propoxyphene, or butorphanol; admission of 2 patients was related to Fiorinal. Five patients were admitted with problems related to noncompliance with prescribed medications. One patient had been noncompliant with carbamazepine, which resulted in exacerbation of a seizure disorder, and 3 patients had been abusing Fiorinal, chlorpromazine, or butorphanol. One diabetic patient was admitted because she had factitiously induced numerous hypoglycemic episodes with her prescribed insulin.

COMMENT

Our study indicates that the incidence of drug-related admissions to an inpatient psychiatric unit is significantly higher than suggested in previously reported studies for medical wards (5–8). In this study we focused on a broad range of drug issues that present patient management problems for the psychiatrist. It has been estimated (10) that 16%–28% of all medical admissions in the United States are directly related to alcohol abuse. Since our data suggest that drug-related hospital admissions represent a major problem for the psychiatrist, one could question whether psychiatrists and mental health nurses are adequately prepared to deal with the nature and scope of these drug problems. It may be desirable to increase the emphasis on pharmacology, toxicology, and therapeutics in their training programs.

Most physicians feel that patients will follow their advice when medications are prescribed, although there is solid evidence to the contrary (11, 12). Because problems of noncompliance are prevalent among psychiatric patients, the psychiatrist must be especially vigilant to problems of medication misuse.

We do not know whether the fact that this study was conducted in a university teaching hospital which serves as a referral hospital and is clearly not representative of the average inpatient psychiatric unit resulted in an increased or decreased number of drug-related admissions. We believe that similar studies should be conducted in other inpatient and ambulatory care settings to more clearly define this problem.

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Depression as Expressed in Pre-Columbian Mexican Art

BY JUAN-RAMON DE LA FUENTE, M.D., AND DONATO ALARCÓN-SEGOVIA, M.D.

While undertaking a larger study dealing with representations of disease in pre-Columbian ceramic figures, the authors found four figures in which depression was clearly depicted. Their findings prove that psychiatric disorders did not go unnoticed by the people who inhabited the American continent before the arrival of the Spanish.

Depression, an unwelcome companion of man for thousands of years, has been traced back to ancient Egyptian manuscripts, to the Old Testament, and to the teachings of Aristotle and Hippocrates. These documents have been described and dealt with by various authors (1–3). Conversely, there are few accounts of depression in pre-Columbian times. However, pre-Columbian sculptors were keen observers and recorders of states of mind, and we and others have previously referred to the acumen of their empiric clinical observations (references 4 and 5 and an unpublished paper by Alarcón-Segovia and de la Fuente). We therefore sought evidence of depression in pre-Columbian Mexican art. Although depression may not always have been specifically identified as such by the pre-Columbian people of Mexico, in this report we present evidence that they recognized it, attempted to prevent it, and probably had some form of treatment for it (6, 7).

METHOD

We reviewed the entire collection of the Museo Nacional de Antropología in Mexico City, including that portion in its warehouses. We also reviewed various large private collections in Mexico City and the collection of the Regional Museum of Jalapa, Veracruz. Technical support was amply provided by the Section of Archeology of the Museo Nacional de Antropología. It was not always feasible to distinguish between pathological features and stylistic traits. However, we believed that some stylistic traits afforded actual representations of disease, albeit involuntarily. From the initial survey we selected about 200 pieces, both in ceramic and in stone, for further study and to be photographed. One hundred fifty pieces were finally considered to represent pathological features or states of mind. Among these were realistic representations of cirrhosis of the liver, brain tumors, paraplegia, goiter, Pott's disease, congenital syphilis, and skin disorders.

ARCHEOLOGICAL BACKGROUND

The region of America that encompasses the southern two-thirds of Mexico and northern Central America to Nicaragua is known as Mesoamerica. It is particularly important anthropologically and archeologically because here the Indian population reached, by anyone's definition, the stage of true civilization (8). The main cultures that inhabited this geographic area were

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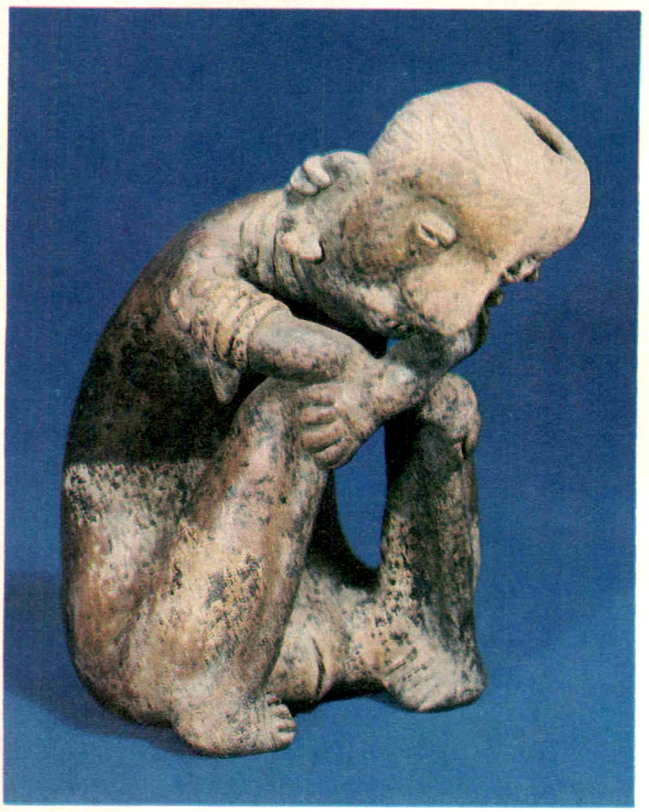
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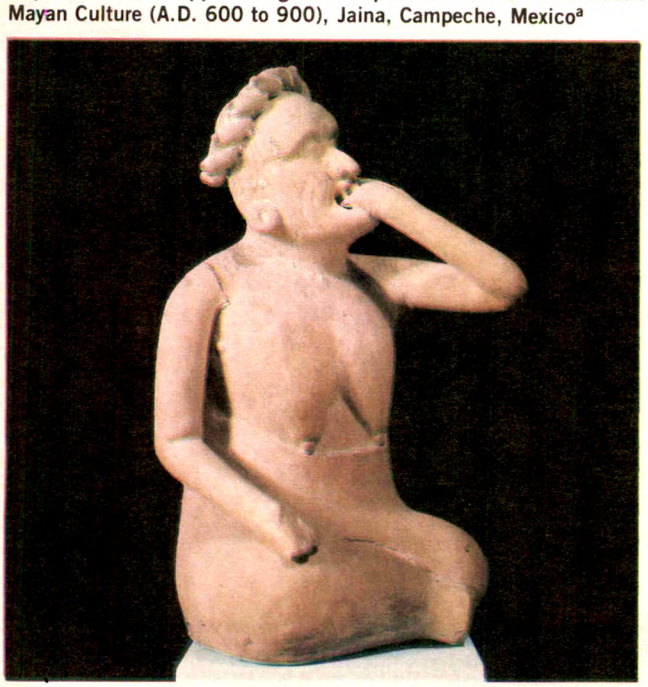
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FIGURE 1
Illustration of Postpartum Depression from the Shaft-Tomb Complex Culture (100 B.C. to A.D. 250), Nayarit, Western Mexico[a]



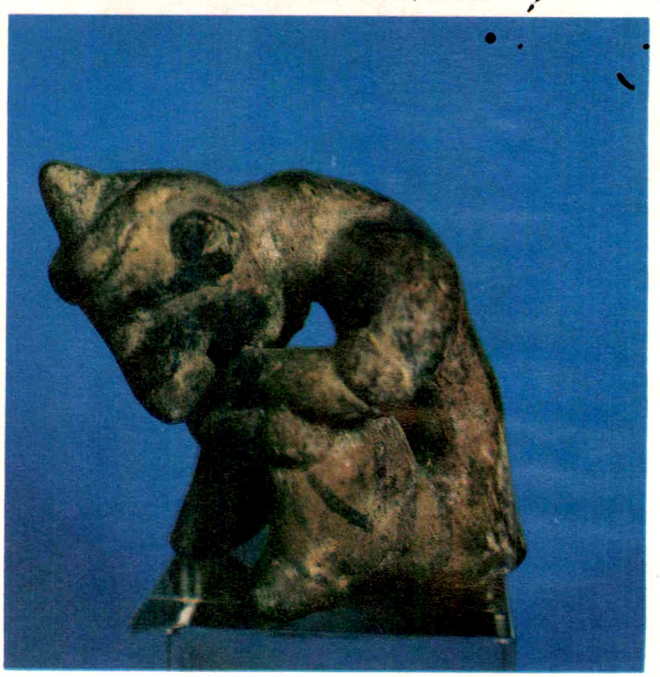
[a]The hole in the head, usually present in figures from this area, which were cooked at very high temperatures, allowed hot air to escape. Height=19.8 cm. Kurt Stavenhagen Collection.

FIGURE 2
Representation of Apparent Agitated Depression from the Late Classic Mayan Culture (A.D. 600 to 900), Jaina, Campeche, Mexico[a]



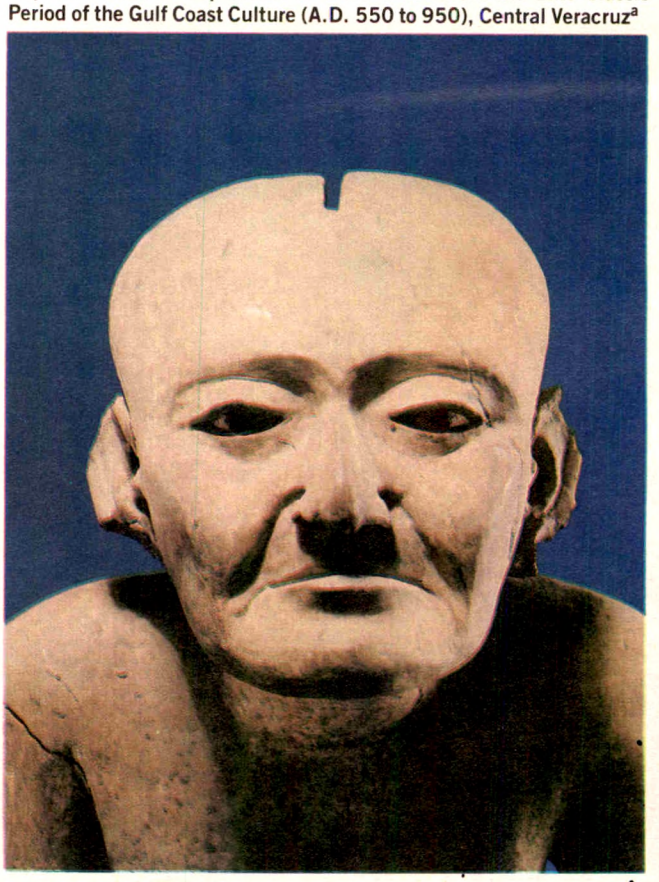
[a]Height=16.5 cm. Collection of the Museo Nacional de Antropología, Mexico City.

FIGURE 3
Illustration of Apparent Retarded Depression from the Shaft-Tomb Complex Culture (about A.D. 100), Colima, Mexico[a]



[a]Height=8.8 cm. Collection of the Museo Nacional de Antropología, Mexico City.

FIGURE 4
Representation of Depression of Senescence from the Late Classic Period of the Gulf Coast Culture (A.D. 550 to 950), Central Veracruz[a]



[a]Height=16 cm. Kurt Stavenhagen Collection.

those of central Mexico, western Mexico, Oaxaca, the Gulf coast, and the Maya civilization. In turn, these cultures can be divided into three periods: the preclassic, from 2300 B.C. to 100 B.C.; the classic, from 100 B.C. to A.D. 950; and the postclassic, from A.D. 950 to the time of the Spanish conquest in 1521 (9, 10).

FINDINGS

We found four human ceramic figures in which depression was clearly depicted. Figures in which grief or sorrow was apparent were also found but are not presented here.

Postpartum depression is well illustrated by the piece shown in figure 1. The postpartum stage is evidenced by the open vulva and hanging breasts. The former is a feature we have found only in figures representing immediate puerperium. The subject is in deep sorrow, appears greatly depressed, with a sad face, and gives the impression of extreme hopelessness. The figure belongs to the western Mexican early classic culture known as the shaft-tomb complex because the figures usually were found in deep ground burial chambers connected to the surface by shafts. These figures usually were placed in corners of the chamber or at the head of the body remains (11).

The old woman biting her fingers (figure 2) would seem to represent agitated depression (involutional melancholia). Features of this disturbance include the involutional age, psychomotor agitation, and melancholia—literally, black bile—as described by Hippocrates, who also described epilepsy, mania, and dementia (12). The old age and the biting of the hand with evident anxiety illustrate well the saying of Plutarch (A.D. 46–120), "Every little evil is magnified by the scaring spectres of his anxiety" (3, p. 67). This figure belongs to the late classic period of the Mayan culture (A.D. 600–900) and was found in 1957 on the island of Jaina off the coast of Campeche. Ceramics from this island excel in depicting naturalistic features of daily life.

The gloomy, taciturn, despondent man shown in figure 3 appears to depict the image of a retarded depression. One can reason that he is also akinetic and anhedonic. The deepness of his depression brings to mind the words of Celsus (A.D. first century): "There are forms of madness that go no further than sadness" (13). This figure also belongs to the shaft-tomb culture of western Mexico and was found in Colima, close to the Pacific coast.

Figure 4 presents another example of depression in the elderly. This figure from central Veracruz on the Gulf coast of Mexico illustrates the bland affect, the silence, the lowered glance, and the drooping lip characteristic of depression. His stare recalls Galen's description of the melancholic state: "dread and desire of death at the same time" (14).

DISCUSSION

The ceramic sculptures presented here provide evidence that the pre-Columbian inhabitants of Mesoamerica recognized the profound changes that people undergo when they are depressed. They give us insight into the fact that the unknown artists who made them were able to convey the expression of states of mind, a skill that appears late in art.

The pre-Columbian people apparently recognized dementia (15) and other psychoses (16) and, according to Vargas-Castelazo (6), who reviewed the chronicles of the Spanish missionaries of the 16th century, they distinguished two forms of depression: *tlahuiloca-yotl*, or agitated, and *xolopiyotl*, or retarded. They seem to have been particularly concerned with the prevention of "postpartum blues," since, according to Sahagún in his *Historia General de las Cosas de Nueva España* ("General History of the Matter of New Spain") (7), the midwife practiced what could be interpreted as supportive psychotherapy. She would tell the woman who had just given birth how brave and resourceful she had been, "alike the eagle and the tiger, having won an actual battle equivalent to that fought by the bravest soldiers." Sahagún's information was gathered firsthand from the Indians. Arriving only a few years after Cortés' conquest of Mexico, Sahagún trained young Indians to write Nahuatl, the language of the Aztecs, into an alphabet and had them interview the Aztec elders with prepared questionnaires. In this way he obtained direct information, which he compiled in several volumes and codices.

The herbal medicine of the Aztecs, as described also by Sahagún, had a preparation consisting of two herbs, the *iztauhyatl* and the *quauhyayual*, that was meant for one who had "anguish in the heart by reason of some bad humor that oppresses him" (17, p. 311). These herbs, said to produce good results, were boiled in water and drunk.

It is apparent, therefore, that manifestations of disease, including those of disorders of the mind, did not go unnoticed by the people who toiled in Mesoamerica long before the arrival of the Spaniards. It is possible that their concern for the recognition and management of psychiatric illness later resulted in the foundation, by the Spanish missionary Fray Bernardino de Alvarez in 1566, of the first hospital in the Americas for the study and care of mental disorders (18).

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Treatment of Persons Found Incompetent to Stand Trial

BY LINDA PENDLETON, PH.D.

Persons found incompetent to stand trial comprise the largest group of psychiatric patients committed to mental hospitals through the criminal justice system in the United States. The law dictates that these patients receive short-term treatment aimed specifically at enabling them to stand trial with as little delay as possible. The author describes the treatment and assessment aspects of a program that admits approximately 200 trial-incompetent persons each year. During 1978, 90% of the 205 patients discharged from this program were certified by the treatment staff as competent to stand trial; 97.5% of that group subsequently completed the trial process.

Persons found incompetent to stand trial comprise the largest single group of criminal commitments to mental hospitals in the United States. It has been estimated that as many as 15,000 persons are committed on this basis at any one time, approximately 52% of all criminal commitments in mental hospitals (1). As a result of a 1972 Supreme Court decision (2), restoration to competency within a reasonable period is now required in order to retain a patient on an incompetency to stand trial commitment. Much has been written about incompetency but surprisingly little about its specific treatment. In this report, I describe an innovative treatment program for incompetency at Atascadero State Hospital, California's primary treatment facility for the mentally disordered criminal offender.

CHARACTERISTICS OF PATIENTS

Patients in this treatment program have all been adjudged, according to California Penal Code statutes, unable by reason of mental disorder to understand the nature of the proceedings taken against them and/or to cooperate with counsel in the preparation of a defense. Criminal charges against these patients range from misdemeanors, such as defrauding an innkeeper, to felonies, such as homicide. Thus far, all of the patients have been men, and the most common diagnosis among them has been schizophrenia, paranoid type; approximately 90% of the 205 patients who were dis-

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charged from this program during the calendar year 1978 had this diagnosis. Generally patients are acutely psychotic upon admission, with delusions that are frequently related to their offense. Described below is a typical patient found incompetent to stand trial who was admitted to the treatment program at Atascadero State Hospital.

Mr. A, a 32-year-old man, was diagnosed schizophrenic, paranoid type, upon admission. He had been charged with assault with a deadly weapon after striking a man with a two-by-four board. The circumstances of the offense were as follows. Mr. A was obtaining gasoline for his vehicle at a service station when another individual, unknown to him, pulled up for gas. That individual had a CB radio in his automobile, and Mr. A reacted with some alarm because he believed CB radio owners were involved in a conspiracy to broadcast information about his sex life. Mr. A promptly grabbed the nearest available weapon, a board from an adjacent construction site, and assaulted the CB owner. Upon Mr. A's admission to the hospital he was able to coherently report the events leading up to his arrest, but he maintained that his offense was justified given his belief that a gross violation of his personal privacy had been perpetrated by owners of CB radios.

TREATMENT PROCEDURES

Upon admission to our hospital each patient's competency is assessed by a psychiatric technician specially trained in the administration of the Competency to Stand Trial Assessment Instrument (3). This instrument was developed as part of a project undertaken by a multidisciplinary team of lawyers, psychiatrists, and psychologists from the Laboratory of Community Psychiatry at the Harvard Medical School who sought to demonstrate quantifiable clinical criteria for competency to stand trial. Their purpose was to design instruments that would accurately assess competency, conceptualized and expressed, in language with sufficient familiarity to both the law and psychiatry to provide a basis for effective communication and assessment of the issue. The resulting Competency Assessment Instrument is a structured interview and rating system that yields an assessment of the accused's capabilities on 13 separate dimensions of competency to stand trial. The 13 dimensions of competency are as follows: appraisal of available legal defenses, unmanageable behavior, quality of relating to attorney, planning of legal strategy, appraisal of roles of courtroom personnel, understanding of court procedures, appreciation of charges, appreciation of range and nature of possible penalties, appraisal of likely outcome, capacity to disclose pertinent facts to attorney, capacity to realistically challenge prosecution witnesses, capacity to testify relevantly, and self-defeating versus self-serving motivation. We have used this assessment of capability, which is conducted under the supervision of the program's psychologist, to identify specific deficits in the patient's ability to cope with his legal situation.

Following the admission assessment, each patient participates in an individual admission team conference in which an interdisciplinary treatment team, comprised of psychiatric technicians, registered nurses, a social worker, rehabilitation therapist, psychologist, and psychiatrist, interviews him and formulates a treatment plan. The treatment plan is designed to eliminate or reduce those symptoms that interfere with standing trial, more commonly delusional beliefs about the alleged offense or the defense attorney. Although traditional methods of treatment are employed, including psychotropic medication and structured activities such as occupational therapy and physical recreation, we also use specialized individual and group therapy. These focus on orienting the patient to the nature of the proceedings being taken against him and on building relationship skills essential to cooperating with the defense counsel.

Once the patient's deficiencies that were identified in the admission assessment have been remedied or sufficiently reduced, he is referred by his treatment team to a competency class. General information is presented in class regarding pleas that may be entered in court, roles of courtroom personnel, courtroom procedure, and appropriate courtroom behavior. After completing the competency class, which includes a written examination on the information that was presented or an oral examination in the case of illiterate patients, the patient next participates in a videotaped mock trial.

The mock trial, developed with the assistance of judges and attorneys, is a treatment and assessment procedure whereby the patient is exposed to a simulated courtroom situation tailored to his own individual case. This is designed to provide the patient with a behavioral rehearsal with feedback, as well as to desensitize him to the stressful situation of standing trial on criminal charges. In addition, it provides an assessment of the patient's capability of withstanding the stress of a trial before he is actually referred back to the court.

ASSESSMENT PROCEDURES

Assessment of the patient's competency to stand trial involves a sequence of procedures that begin with the baseline data collected at the time of the patient's admission through interviewing him with the Competency to Stand Trial Assessment Instrument. The next assessment step, which is less formal, involves a judgment by the patient's treatment team that his problems in competency noted on the admission assessment have been adequately treated; this judgment results in his referral to the competency class. Next is the examination at the end of the class; a score of 70%

is necessary to pass. The patient's performance in the mock trial is also scored pass or fail. A failure in either the class or the mock trial results in the patient's being referred back to the treatment team for more individual work in the remaining areas in which he or she is deficient. After additional training in these areas the patient repeats the failed activity, and his performance is reassessed.

When the patient successfully demonstrates his competency in the class and the mock trial, he is referred to the disposition staff interview, the final assessment procedure. The Program Director, or his assistant, conducts a formal interview with input from the interdisciplinary treatment team. The interview consists of an amalgam of prior assessment procedures, including an abbreviated version of the Competency to Stand Trial Assessment Instrument and questions on information covered in class, with particular attention paid to areas in which the patient demonstrated deficiencies earlier in the assessment sequence. In addition, a modified mental status examination is conducted to check for the presence of symptoms formerly identified as interfering with the patient's competency to stand trial. If the patient successfully completes this procedure, he is referred back to the courts as competent to stand trial; if the patient fails, which is rare at this stage, he is referred back to the treatment team for additional treatment in deficient areas.

RESULTS

The effectiveness of this treatment program is best demonstrated by the fact that, in 1978, 97.5% of those patients certified competent by the program staff were able to successfully complete the trial process upon their return to court. During 1978, 184 patients (90%) of the 205 patients discharged were certified competent. The remaining 10% of the patients either failed to attain competency within the time limits specified by state law (patients must achieve competency within 3 years or the maximum time they could have served if convicted of the offense, whichever is less) or they made so little progress that they were deemed unlikely to attain competency. During the 15 months preceding April 1979, the average duration of treatment before competency certification was given was 104 days.

It is our hope that eventually controlled studies may be undertaken to determine the relative impact of various facets of our treatment program.

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Interpersonal Factors in the Psychiatry Clerkship: New Findings

BY CAROLE S. ORLEANS, PH.D., JEFFREY L. HOUPT, M.D., AND DAVID B. LARSON, M.D.

The authors took an empirical approach to determining ingredients of a successful psychiatry clerkship. They collected measures of clinical, didactic, and interpersonal experiences throughout the clerkship and correlated these with a range of outcomes. Correlations showed that positive relationships with supervisors, fellow students, and patients were strongly related to favorable attitudes and opinions at the end of the clerkship and to improvements in self-rated clinical skills. Only outcomes related to formal knowledge were independent of these factors. These findings highlight the importance of structuring a positive interpersonal environment for the psychiatry clerkship.

Which experiences contribute most to positive outcomes in a psychiatry clerkship? The data to answer this question are currently limited to studies asking students to rank different activities and experiences according to their relative value. From these studies (1-5), one finds that high levels of patient involvement and responsibility are ranked most highly and that experiences involving positive interpersonal relationships with supervisors, peers, and patients are also frequently singled out as particularly valuable.

How well do students' rankings correspond to actual positive changes in their knowledge, skills, and attitudes? In this study we took an empirical approach to identifying ingredients of a successful clerkship. Our main objective was to determine correlates of positive outcome in four domains—knowledge, skill, attitude, and opinion. We incorporated several measures of the interpersonal aspects of clerkship training in addition to measuring a broad range of actual didactic and patient care experiences.

METHOD

Subjects. Our subjects were 92 of 110 second-year Duke medical students completing their required 8-week psychiatry clerkship during the 1977 calendar year who agreed to participate in the study. The Duke psychiatry clerkship program has been described in detail elsewhere (6).

Training measures.[1] To measure training experiences, students were asked to complete four biweekly summaries of their clerkship activities. These periodic summaries avoided reliance on a single, less accurate retrospective report. Students estimated the actual number of hours they had spent during the previous 2 weeks in 6 different modes of training (supervision, demonstration, formal instruction, study, training-related staff interaction and training-related student interactions) and in 7 different types of supervised clinical experience (evaluation, crisis intervention, pharmacotherapy, psychotherapy, family therapy, referral for further mental health care, and liaison-consulting). Students also documented various aspects of their patient care experience. We determined the number of psychiatric and nonpsychiatric patients for whom the students assumed a substantial amount of responsibility and derived a single score representing the percent of psychiatry patients. We also collected bipolar numerical ratings of severity of disorders, duration of treatment needs, degree of overall patient care responsibility, and adequacy of supervision. Mean scores based on all four biweekly summaries were calculated for each training measure and used in subsequent correlations.

Outcome measures. Attitude and perceived skill ratings were obtained before and after the clerkship, and change scores were derived by subtracting preclerkship from postclerkship scores. Seven highly correlated attitude scales described in detail elsewhere (7) were combined to yield a single score reflecting attitudes toward psychiatry, psychiatric patients, and psychiatric techniques. Students used numerical bipo-

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[1]Further information about measures and scoring procedures is available from Dr. Orleans.

TABLE 1
Significant Correlates Between Outcome and Training Measures of a Psychiatric Clerkship

Outcome	Significant Correlate[a]	r
Positive attitude change[b]	Satisfaction with house staff	.28[c]
	Satisfaction with senior staff	.39[d]
	Satisfaction with patients	.39[d]
	Adequacy of supervision	.25[c]
Overall clerkship satisfaction	Satisfaction with house staff	.65[d]
	Satisfaction with senior staff	.42[d]
	Satisfaction with fellow students	.27[c]
	Satisfaction with patients	.56[d]
	Adequacy of supervision	.56[d]
	Hours of supervision	.33[d]
	Hours of demonstration	.30[c]
	Hours with fellow students	.32[c]
	Hours with staff	.31[c]
	Degree of responsibility for patients	.39[d]
Area of perceived skill improvements[b]		
Evaluation	No correlates	
Crisis intervention	Hours of crisis intervention	.34[d]
	Hours of referral	.28[c]
	Satisfaction with house staff	.38[d]
Pharmacotherpy	Hours of pharmacotherapy	.29[c]
	Hours of psychotherapy	.29[c]
	Satisfaction with house staff	.39[d]
Psychotherapy	Hours of psychotherapy	.25[c]
	Hours with fellow students	.33[d]
	Satisfaction with house staff	.39[d]
Family therapy	Hours of psychotherapy	.26[c]
	Hours with fellow students	.27[c]
	Satisfaction with house staff	.38[d]
Referral	No correlates	
Liaison-consulting	Hours of liaison-consulting	.40[d]
	Adequacy of supervision	.35[d]
	Percent of psychiatry patients	−.25[e,f]
Treating mild disorder	Satisfaction with house staff	.38[c]
Treating severe disorder	Hours of pharmacotherapy	.25[c]
	Hours with staff	.25[c]
	Hours with fellow students	.25[c]
	Satisfaction with house staff	.46[d]
	Adequacy of supervision	.30[c]
National Board of Medical Examiners scores	Hours of crisis intervention	−.25[c]

[a]Only correlations significant at $p<.02$ or higher are included.
[b]Pre- to postclerkship improvements were significant at $p<.05$ or higher.
[c]$p<.01$, df=90, two-tailed.
[d]$p<.001$, df=90, two-tailed.
[e]$p<.001$, df=90, two-tailed.
[c]$p<.02$, df=90, two-tailed.
[f]Percent of nonpsychiatry patients correlated +.25.

lar rating scales to rate their level of skill in each of 9 areas: evaluation, crisis intervention, pharmacotherapy, psychotherapy, family therapy, referral, liaison-consulting, treating mild disorder, and treating severe disorder. After completing their clerkship, students rated their overall satisfaction with the clerkship using a bipolar numerical rating scale. Positive response bias was minimized by collecting attitude, skill, and satisfaction ratings in addition to the biweekly summaries, apart from usual clerkship evaluation procedures, using special coding to preserve students' anonymity.

RESULTS

Preliminary analyses showed that all pre- and postclerkship changes in attitude and perceived skill were positive and significant at the $p<.05$ level or higher.[2] Overall satisfaction ratings were also favorable; the mean rating was +6.4 on a scale ranging from −10 (extremely dissatisfying) to +10 (extremely satisfying). The mean National Board of Medical Examiners (NBME) score was 551, placing overall performance at the 68th–69th percentile.

To identify clerkship experiences associated with positive outcomes, we computed Pearson product-moment correlations between outcome and training measures. Because of the large number of correlations performed, a conservative significance level of $p<.02$ was chosen (two-tailed, df=90). Table 1 presents only the significant correlates for all outcome measures.

[2]Results of analyses not reported are available on request from Dr. Orleans.

Looking first at the correlates of attitude change, we find that table 1 suggests that interpersonal training experiences play a crucial role in shaping medical students' attitudes toward psychiatry, psychiatric patients, and techniques. Highly rated relationships with house staff (primary clerkship supervisors), senior staff, and patients as well as ratings of the quality of overall supervision correlated positively with a favorable attitude shift and with a rating of overall clerkship satisfaction. Measures of the amount of time spent in interpersonal teaching experiences (supervision, demonstration, contact with fellow students, and contact with house and senior staff), the degree of responsibility for patient care, and the quality of peer relationships during the clerkship were also correlated positively with overall satisfaction with the clerkship. Attitude change and overall satisfaction measures thus seemed to tap similar outcomes and were themselves positively correlated ($r=.48$, $df=90$, $p<.001$).

Turning next to perceived skill outcomes, table 1 shows that the amount of time students spent in a given clinical activity correlated with improvements in self-rated skill in that and related activities. Table 1 correlations also suggest that positive didactic and supportive relationships with supervisors and peers are needed to maximize benefits of high levels of patient contact and responsibility. Satisfaction with house staff relationships correlated positively with 6 of 9 skill improvements, and quality of peer interactions was associated with improvements in psychotherapy and family therapy skills and in treating severe disorder. Overall, positive clerkship relationships seemed most important for teaching complex interpersonal treatment skills and clinical responsibilities that typically generate a high level of anxiety in novice therapists (e.g., individual and group psychotherapy and treating severe disorder).

Finally, table 1 shows only one significant correlate for NBME scores—time spent in crisis intervention activities was inversely related. Surprisingly, NBME performance did not even correlate with measures of time spent studying and reading. It is possible that students prepared for these exams outside of their usual clerkship activities. Additional correlations[3] showed that NBME scores failed to correlate significantly with any of the other 11 outcome measures, indicating that the cognitive outcomes it taps are fairly independent of the perceived skill, attitudinal, and affective outcomes tapped by other measures.

COMMENT

The interpersonal context of training emerged as the single most important factor influencing clerkship outcomes. Correlations suggest that positive relationships with supervisors and fellow students during the clerkship play an extremely important role in shaping positive attitudes toward psychiatry and psychiatric patients and techniques and in determining satisfaction with training experiences. Moreover, the amount and quality of clerkship-related peer and supervisor contact was strongly related to skill improvements, particularly for the most demanding interpersonal treatment skills. Given the crucial role of interpersonal training experiences, curricula relying in large part on impersonal and self-instructional teaching aids may not have the best outcomes (8).

Future research should clarify determinants of a positive interpersonal experience during the psychiatry clerkship. Along these lines, positive relationships probably combine didactic, modeling, and supportive functions: relaying concrete knowledge and helping students manage the anxieties commonly elicited by clerkship responsibilities (9). Modeling and support functions may give house staff an especially critical role. Because students see house staff as more similar to themselves than senior staff, they may be better able to identify with house staff and to receive more helpful modeling and support. Other studies of the psychiatry clerkship (1, 3) have found that ease of identifying with supervisors contributes to positive attitude and opinion outcomes. In general, similar models are often more helpful than dissimilar expert models in teaching a new skill or in overcoming skill-related anxieties (10).

[3]Results of analyses not reported are available on request from Dr. Orleans.

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Psychiatric Residents' Attitudes Toward Teaching

BY KENNETH E. CALLEN, M.D., AND JOHN M. ROBERTS, M.D.

The authors surveyed psychiatry residents in academic training programs to determine the amount of time they spent in teaching activities and to assess their attitudes toward teaching. They found that most residents spent from 1 to 2 hours per day teaching and felt they should do more. They tended to see themselves as capable teachers, and they valued traditional medical skills. They did not seem to value the use of audiovisual materials, written handouts, or self-instructional materials. Most of the residents had received no formal instruction designed to make them better teachers or direct feedback from faculty supervisors about their teaching skills.

The teaching of psychiatry has become a popular subject at national meetings and in journal articles. Interest in this topic is clearly shown by the increasing attendance at meetings of organizations devoted to education, such as the Association for Academic Psychiatry, the American Association of Directors of Psychiatry Residency Training, and the Association of Directors of Medical Student Education in Psychiatry. At least some of the concern about psychiatric education comes from studies showing that medical students and practicing physicians tend to rate their training in psychiatry as mediocre to poor (1–4). The tendency for medical students to be quite outspoken in their criticism of poor teaching has also precipitated reexamination of many educational programs (5). Other factors contributing to the rising interest in teaching are the need to attract more medical students into psychiatry residency programs, the appointment of many full-time directors of medical student education, and the popularity of liaison psychiatry, with its associated teaching commitment.

This new wave of interest in educational issues has generated a large number of papers dealing with curriculum organization and design as well as content (6–9). However, very little has been written about the most important part of any program, the teacher. The house officer, as noted by Kaufman (5), serves as the main teacher and the student's closest role model during the clinical clerkship. This important role was recognized in 1978, when APA's Office of Education sponsored a combined meeting of the Association for Academic Psychiatry, the American Association of Directors of Psychiatry Residency Training, and the Association of Directors of Medical Student Education in Psychiatry. The meeting was titled "Teaching the Resident How to Teach." Although this meeting was very useful, it was apparent that little was known of the role of residents as teachers.

This survey was designed to gather information about the teaching activities of residents in psychiatry with specific emphasis on the time involved, their attitudes toward teaching, and their opinions on a variety of other educational topics.

METHOD

We sent questionnaires that we designed to each of the 110 university-based psychiatry programs listed in the 1979 edition of the Association of American Medical Colleges *Directory of American Medical Education*. Each director of residency training was asked to randomly select a first-year resident and a third-year resident to complete and return the questionnaire. The questionnaires were returned anonymously except for inclusion of the first three digits of the zip code, which was done so we could compare the results from different geographical areas.

The questionnaire was designed to elicit information in the following areas: 1) sex, age, year in training, and future career plans of the respondents, 2) attitudes of the residents toward themselves as teachers and toward the role of teaching in a residency program, 3) estimates of the actual amount of time spent teaching and opinions about the "ideal" amount of time one should devote to teaching in a good training program, and 4) ratings of the importance of including certain topics and skills in a program designed to improve the effectiveness of teaching by residents in psychiatry.

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TABLE 1
Psychiatry Residents' Responses to Questionnaire Statements About Teaching

Questionnaire Statement	Percent of 136 Responses		
	Agree	Disagree	Not Certain
Training of nonpsychiatric physicians to deal with emotional illness is one of the most urgent needs in medical education	80	7	13
In my role as a resident, I am here to learn psychiatry, not to teach it	6	89	5
First-year residents really don't know enough to teach someone else	13	78	9
The main reason residents are required to teach medical students is to free up time for faculty to do research and other things	6	78	16
Residents should be required to attend courses designed to make them better teachers	32	47	21
Third-year residents don't know enough to teach someone else	0	99	1
Questions pertaining to education should be included in Board examinations	22	48	30

RESULTS

One hundred sixty-four (75%) of the questionnaires were returned. However, 28 of these were discarded because of incompleteness or misinterpretation of instructions, leaving 136 complete questionnaires. The data were divided into the following groups for analysis: total sample, male and female respondents, first-year and third-year respondents, eastern United States (zip code digits 000–299), middle United States (zip code digits 300–799), and western United States (zip code digits 800–999). In general, the responses of each of these groups were quite similar; there were only a few significant differences, which will be discussed below. The mean age of all respondents was 29.40 years; first-year residents averaged 28.13 years of age, and third-year residents averaged 30.27 years of age. There were no significant differences in age between male and female respondents or residents from different parts of the country.

Table 1 shows the agreement and disagreement of the total sample with each of seven statements about attitudes of residents toward teaching. The only significant departure from the overall responses occurred with residents from the western states. Fifty-seven percent disagreed with the statement that first-year residents really do not know enough to teach others, in contrast to the overall 78% who disagreed ($p<.001$, chi-square). Only 14% from the west felt that residents should be required to attend courses designed to make them better teachers, whereas 32% of the total sample agreed with this statement ($p<.01$, chi-square).

TABLE 2
Mean Estimates of Actual and Ideal Teaching Time of 136 Psychiatry Residents

Group Taught	Estimate of Actual Time Spent Teaching per Week (hours)	"Ideal" Amount of Time to Spend Teaching per Week (hours)
Medical students	3.67	4.48
Nurses, occupational therapists, social workers	2.04	2.59
Psychiatry residents	1.45	3.01
Nonpsychiatric physicians	1.61	3.22
Total	8.77[a]	13.30[a]

[a]The difference between the means was significant at the .01 level (chi-square).

Table 2 shows the findings of several items on the questionnaire designed to gain information about how much time the residents spent teaching medical students, nurses, occupational therapists, social workers, psychiatric residents, and nonpsychiatric physicians. They were also asked to indicate what they thought was an ideal amount of time to devote to each of these activities. There was a significant difference in each case: estimates of actual time spent were considerably lower than the residents felt they should ideally spend teaching. Overall, the group estimated that they spent about 9 hours a week in teaching activities and that two-thirds of that time was devoted to medical students, nurses, occupational therapists, and social workers. The smaller amount of time spent teaching nonpsychiatric physicians is surprising in view of the recent emphasis on the educational component of consultation/liaison work. First-year residents are often not involved in consultation and liaison, and one might suspect that their responses may have lowered the overall result. However, comparison of results shows no significant difference between first- and third-year residents.

The residents were also asked to estimate the percent of time in each year of training that they felt should be devoted to teaching. The results show a range of 9% in the first year, 14% in the second year, 21% in the third year, and 28% in the fourth year of training. If one assumes a 60-hour work week, these percents translate into about 5 hours per week in the first year, increasing to 17 hours per week in the fourth year, and an overall average of 10.8 hours per week for all 4 years.

In spite of the rather large amount of time these residents spent in teaching, they were not particularly interested in attending training sessions to make them better teachers. Only 32% felt they should be required to attend courses to make them better teachers, and only 13% indicated they had received any training designed to improve their teaching. Feedback regarding

teaching skills from experienced instructors was apparently uncommon. Two-thirds of the residents indicated that they had received written feedback from students about lecture presentations; however, very few had received any type of criticism or suggestions from their faculty supervisors.

The respondents' opinions about what skills are important in making them better teachers followed the traditional lines of medical education. Interviewing, differential diagnosis, psychopharmacology, and emergency psychiatry received the highest ratings of importance; the residents were less concerned with developing their skills in preparing written materials, self-instructional courses, or the use of videotapes, movies, or other technological aids.

DISCUSSION

These results indicate that most psychiatric residents have a strong commitment to teaching and are willing to devote a considerable amount of time to educational activities. Our respondents spent almost 2 hours per day teaching and felt that this should be increased to more than 3 hours per day, especially in the last year of training. This amount of time would make teaching one of the major components of any residency program; however, it appears that very little if any effort was made to determine if these residents were doing a satisfactory job or to provide training designed to make them better teachers. The reason for this is probably the residents' own lack of motivation in asking for courses to make them better teachers and the likelihood that most faculty prefer to teach clinical subjects. It is unfortunate that many of the residents were not concerned with improving their skills in preparing written materials and using audiovisual aids. Both of these are relatively easy to teach and enhance the learning of some topics difficult to present orally.

Residents from western states seemed to feel less capable of teaching in the first year of training and, paradoxically, were less interested in attending courses designed to make them better teachers. The reason for this difference in attitudes is not clear from the data obtained in this study.

The old adage, "See one, do one, and teach one," although possibly appropriate for some specialties, is simply not adequate for psychiatry, with its abstract concepts, invisible pathology, and need for confidentiality. We must improve the communications skills of residents (and faculty) if we are to alter the frequent complaints of poor teaching in psychiatry.

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CLINICAL AND RESEARCH REPORTS

This section contains 1) new research findings, including preliminary data from pilot studies, either clinical or laboratory; 2) worthwhile replication studies; 3) case reports that describe a truly new syndrome or cast new light on established ones; and 4) case reports that indicate a new therapeutic procedure of potential value or call attention to adverse effects of drugs or previously unreported complications of therapeutic interventions. Program descriptions and literature reviews cannot be printed in this section. Criteria for format are listed in "Information for Contributors" in each issue; papers that do not adhere to these criteria will be returned to the author.

Intoxication Associated with Lithium and ECT

BY MICHEL R. MANDEL, M.D., JOSEPH MADSEN, ALEXANDER L. MILLER, M.D., AND ROSS J. BALDESSARINI, M.D.

Administration of lithium carbonate during a course of ECT has recently been associated with the development of delirium in a few cases (1-3). Whether a specific ECT-lithium interaction is responsible for this toxic state is unclear, and a mechanism of action has not been elucidated, although increased entry of Li^+ into the brain after ECT is one possibility. This report describes two additional cases of persistent delirium following concurrent administration of ECT and lithium and our attempts to evaluate the distribution of Li^+ in laboratory rats given lithium and electroconvulsive shock (ECS).

Case Reports

Case 1. Mr. A, a 55-year-old, right-handed engineer with bipolar affective illness, developed agitation, depressed mood, and ruminative thinking. Amitriptyline (250 mg/day), lithium carbonate (300 mg t.i.d.; serum levels of 0.8-1.2 mEq/liter), and their combination had been given without success before Mr. A was hospitalized on a psychiatric unit. Chlorpromazine (50-150 mg/day) reduced his agitation, but led to sedation and anticholinergic effects. In preparation for ECT, all drugs except 300 mg of lithium carbonate t.i.d. were discontinued 48 hours before beginning ECT. Laboratory values, including serum electrolytes and urinalysis, were all within normal limits; serum lithium was 0.98 mEq/liter. Mr. A was eating and drinking adequately and was fully oriented, alert, and able to perform mental calculations correctly. Right unilateral ECT (175 volts, sinusoidal, 500 milliamperes for 1 second) was given on alternate days and produced seizures lasting 50-60 seconds. Immediately after the third ECT Mr. A demonstrated signs of delirium including confusion, disorientation, and restlessness, but, in contrast to previous treatments, this state failed to clear. The findings from physical and neurological examinations within 24 hours after his third ECT were normal, except for the change in his mental status; serum lithium 3 hours after the third ECT (12 hours following the last dose of lithium) was 0.80 mEq/liter. As this clinical picture persisted, lithium was discontinued; 48 hours later Mr. A was much less confused and somewhat less depressed. ECT was therefore resumed; 4 additional sessions of ECT led to an adequate antidepressant response without further delirium following a usual degree of mild, brief initial postictal confusion. Maintenance lithium was resumed 10 days after completion of the ECT. Two months later Mr. A was well without further cognitive disturbance. He could not recall his early period of hospitalization, but he remembered being discharged from the hospital and had no other evidence of memory impairment.

Case 2. Ms. B, a 67-year-old, right-handed woman with recurrent unipolar major depressive illness that was inadequately responsive to antidepressant drugs, had been successfully maintained for 2 years on lithium carbonate (300 mg t.i.d.; serum levels of 0.6-0.8 mEq/liter). When she became progressively depressed over a 4-week period, she was

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admitted for ECT, the most successful treatment for her in the past. The findings from routine physical and neurological examinations and laboratory values, including electrolytes and urinalysis, were normal before ECT. Lithium was continued; following the first right unilateral ECT (200 volts, sinusoidal, 500 milliamperes, 1 second), she developed slurred speech and became disoriented. These dysfunctions improved somewhat over 24 hours after the treatment but worsened after the second ECT 2 days later. Lithium was then discontinued. Serum lithium 24 hours after the second ECT (12 hours after the last dose of lithium) was 0.6 mEq/liter; other electrolyte levels were normal. Within 72 hours she was much less confused. ECT was given four more times without further cognitive disturbance and with good antidepressant effect. Two weeks following the ECT sessions Ms. B resumed a maintenance dose of 300 mg of lithium carbonate t.i.d. without further complications.

Laboratory Studies

Methods. These clinical observations suggested that ECT and lithium might interact to produce delirium, possibly by increased entry of Li^+ into the brain. To test this hypothesis, we conducted two experiments with adult (300–350 g) Sprague-Dawley rats. They were given ECS by ear-clip electrodes after repeated (experiment 1) or single (experiment 2) doses of lithium chloride. Doses of lithium chloride were chosen so that blood concentrations of Li^+ would reach clinically encountered levels (about 1 mEq/liter) 10–12 hours later (4).

In experiment 1 rats were given 4 mEq/kg of lithium chloride in water (intraperitoneally) twice daily for 3 days; a final dose on the morning of day 4 was followed in 30 minutes by a single ECS (5). Controls received only restraint and application of the electrodes, not ECS. All rats were decapitated 60 minutes following the final dose of lithium chloride.

In experiment 2 rats were given 5 mEq/kg of lithium chloride intraperitoneally once only. Controls received no further treatment before decapitation. Those rats receiving ECS (6) 2 hours later were given atropine sulfate (0.2 mg, intramuscularly, 15 minutes before ECS) and succinylcholine chloride (0.2 mg by tail vein). Then an endotracheal tube was inserted into each rat, and it was artificially ventilated during muscular paralysis for 5 minutes before and 10 minutes after ECS. The respirator delivered a 97:3 or 90:10 mixture of O_2:CO_2 at a rate and volume previously found to yield normal arterial pH and pCO_2 with the 3% CO_2 mixture (6). EEG was monitored with bilateral subscalp electrodes and a Grass polygraph. ECS stimuli (150 pulses of 1-millisecond duration at 150 volts over 1 second) were repeated at 10-second intervals. Half of the rats received 3 ECSs, and the others received 10 ECSs in order to evaluate the effects of more intensive stimulation. The paralyzed rats recovered within 10 minutes and were decapitated 30 minutes after ECS (150 minutes after lithium chloride).

Serum was recovered from coagulated blood by centrifugation. Whole brains were removed, cleaned of vessels and blood, homogenized in trichloroacetic acid (10%, weight to volume in experiment 1) or a detergent in water (Acationex, 0.02% by volume in experiment 2) and centrifuged at 60,000×*g* for 20 minutes. The concentration of Li^+ was determined in the resulting supernatants of tissue extract (diluted 12-fold) or in serum (diluted 40-fold with deionized water) by atomic absorption spectrometry (Perkin-Elmer) (4); standards included known amounts of lithium chloride in appropriately diluted serum or tissue extracts from normal rats, and recovery was quantitative.

TABLE 1
Effect of Electroconvulsive Shock (ECS) on Lithium Distribution in the Rat

	Brain Concentration of Li^+ (mEq/kg)		Ratio of Brain to Serum Concentration of Li^+[b]	
Condition[a]	Mean	SE	Mean	SE
Experiment 1[c]				
Control rats	1.92	0.32	0.58	0.08
ECS rats	2.07	0.43	0.61	0.12
Experiment 2[d]				
Control rats	1.36	0.06	0.40	0.02
ECS rats	1.38	0.04	0.45	0.03

[a]There were 8 to 12 rats per group in each experiment.
[b]Ratio of brain to serum concentration of Li^+ was determined for each rat, and means were computed from the logarithm of each ratio; no difference between control and shocked rats was statistically significant by Student's *t* test.
[c]Four days of lithium chloride; unmodified ECS.
[d]Acute lithium chloride; modified ECS.

Results

There were no significant increases in brain concentrations of Li^+ or in the ratio of brain to blood levels after ECS (see table 1). There was also no difference between ECS given 3 times versus 10 times or ventilation with 3% versus 10% CO_2 in experiment 2; thus, these data were pooled. In addition, the use of atropine and succinylcholine, as encountered in clinical ECT, had no effect on the distribution of Li^+ between blood and brain. Ratios in experiment 1 were higher due to the use of a 4-day treatment regimen that leaves some Li^+ in the brain before the final dose of lithium chloride.

Another laboratory reported that the efficiency of extraction of radio-labeled ions by brain, per unit blood flow, was unchanged by ECS but suggested that *total* extraction was increased due to increased blood flow during and closely after ECS (7). Thus, it seemed reasonable to hypothesize that the equilibration of Li^+ between blood and brain might be accelerated by ECS. In order to maximize the chances of observing increased entry of Li^+ into rat brain after ECS; we selected those times after the rats received doses of lithium chloride so that large differences would exist between blood and brain concentrations of Li^+ (4) because later times closer to steady-state conditions would tend to obscure influences of ECS due to accel-

erated blood-brain exchange of Li^+ (7). Moreover, we included conditions of intensive stimulation and the addition of high concentrations of inspired CO_2 in order to maximize the chances of observing changes due to increased cerebral blood flow (7).

Conclusions

Even under rather extreme conditions, no significant increases in the accumulation of Li^+ in the brain or efficiency of its entry from blood to brain could be detected. Thus, our present laboratory models do not support the hypothesis that electrically induced seizures, or their modification by drugs used in clinical ECT, significantly increase the levels or entry of Li^+ in the brain even at times of maximum differences between blood and brain concentrations and during conditions of strong stimulation and large elevations of cerebral blood flow. We suggest, rather, that the occasional development of a persistent but reversible organic mental syndrome during the association of ECT with lithium therapy, as in the case reports described above, is not likely to be a consequence of markedly increased brain levels of Li^+. Other, possibly additive but reversible, toxic effects of the two treatments in susceptible individuals should be considered.

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Treatment of the Child Pornography Patient

BY ULRICH C. SCHOETTLE, M.D.

Although there has been a burgeoning interest in child abuse, especially sexual abuse, little has been written on child pornography (1–4). Until recently the literature was devoid of case material about adults and children involved with child pornography either as viewers or as participants (5). In this paper, child pornography refers to any visual reproduction of the sexual abuse of children. With the aim of addressing special treatment issues, I will present a case report of an early pubescent girl involved in a child pornography ring.

Case Report

Kathy, a 12-year-old girl, had casual nonsexual meetings with two pornography ring members for at least 2 years before her first encounter with pornographic pictures at age 8. Subsequently, she, her siblings, and a number of boys and girls engaged in photographed oral, anal, and genital sexual acts with adults and each other during a $3\frac{1}{2}$-year period. After police investigation of the child pornography ring, Kathy and her family were referred to our sex abuse clinic for psychiatric evaluation and weekly therapy.

At the first session Kathy's complaints related to feeling guilty about being accused of involving her younger siblings in the sexual exploration schemes. Kathy said she had threatened to kill herself the day neighborhood children harassed her family after the news media exposed her family's participation in the ring. Initially she denied all pleasurable feelings associated with the sexual interactions, and she said that throughout her participation in the pornography ring she feared recrimination from the perpetrators.

Kathy was a short-statured, verbal, early pubescent girl who wore age-appropriate clothing. Although she was initially anxious and depressed, she subsequently demonstrated an appropriate range of affect. Psychological testing revealed she had a low-average intelligence; her projective testing themes included helplessness, depression, victimization, and a denial of, or pulling away from, sexual concerns. Kathy had no history of psychotherapy, psychosis, drug abuse, or major medical illness. Her developmental milestones were age appropriate, except for the deviant sexual experiences.

The family's history was remarkable in that when Kathy was 2 years old, her alcohol-abusing father allegedly threat-

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ened to kill her with a knife during a family argument. No further history of sexual or physical abuse was reported. The mother's second marriage was likewise beset with turmoil but with no demonstrable abuse. Kathy's mother described her own youth as one of being unloved and physically abused; she ran away to marry Kathy's father at age 15. Kathy's mother repeatedly denied all knowledge of her children's involvement in pornography.

Kathy's history and presentation were consistent with a diagnosis of sexual deviation with associated depression. After the initial family evaluation sessions, the sexual abuse clinic staff felt that each family member warranted individual therapists because of unique individual dynamics and significant differences in age. The children were primarily seen in psychoanalytically oriented play therapy. Kathy's mother had supportive psychotherapy with an emphasis on role modeling. Neither the biological father nor the stepfather was available for intervention. Biweekly therapist meetings coordinated treatment strategies and allowed for clarification of interagency communication, especially regarding legal matters.

Discussion

There appear to be three phases in treating children involved in pornography. In the first phase, the sexual "ticket of admission" phase, patients are often asked to give detailed accounts of their sexual encounters to police investigators, the court system, social service agencies, and also the medical/psychiatric community for purposes of initiating treatment as well as prosecuting perpetrators. Concurrent with a flood of emotion (6) and ambivalent relief that the secret is broken (7), guilt feelings begin to emerge (5, 7, 8). Kathy felt guilty for being declared an innocent minor by the juvenile justice system while her adult companions were incarcerated.

In early treatment sessions, the pornography victim relatively freely discusses sexual matters, albeit there are often giggles, smirks, and an avoidance of eye contact. As a transference relationship develops, the patient may deny earlier statements of sexual involvement in hopes of seeking the therapist's positive regard. This is in marked contrast to the more typical early adolescent patient, who is often initially reticent to reveal sexual material but becomes less inhibited as a therapeutic alliance develops.

In the second phase of treatment, the ambivalent phase, the issue of sexual promiscuity assumes less importance. Disturbed early parent-child relationships come to the forefront. For instance, Kathy repeatedly spoke and dreamt of her ambivalent feelings toward one of the ring members whom she regarded as "warm, giving, and father-like," yet he allegedly exploited the children sexually. Paradoxically, the child's anger over repeated sexual assaults is not necessarily directed at the offenders but rather at the failure of the mother and the absent father to protect the victim from adult transgressions (7). In addition, there is an active attempt by the child to split off the good and bad parts of herself and of significant adults.

In the third phase of treatment, the integration phase, there is a gradual resumption of the normal developmental process. Sexuality becomes integrated into the child's whole personality (5). The patient has developed additional nonsexual ways of communicating with peers and adults. Now, the child is able to appreciate good and bad qualities in herself or himself and others.

Throughout treatment, countertransference issues can hamper effective intervention (9). Rescue fantasies, punitive urges, curiosity, and fears of over- or undersexualizing the therapeutic transference relationship are recurrent countertransference themes. The child's cathartic description of the sexual transgressions can be stimulating, enticing, and also threatening to the therapist, who attempts to be objective and empathetic while listening to a barrage of sexually laden transference material.

Although there are unique issues in the treatment of pornography ring victims, in this case standard methods of psychoanalytically oriented psychotherapy proved useful in eliciting dynamics and intrapsychic conflicts and in working through issues of resistance and transference. The child pornography victim, although emotionally and physically traumatized, appears to be able to reintegrate herself or himself into an age-appropriate lifestyle.

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ECT and Cardiac Patients with Pacemakers

BY GEORGE S. ALEXOPOULOS, M.D., AND RICHARD J. FRANCES, M.D.

ECT is likely to be indicated in cardiac patients with pacemakers because heart disorders occur most frequently in middle or advanced age, a period of high incidence of depression (1). We discuss below various considerations involved in using ECT on patients with pacemakers and describe a successfully treated cardiac patient with a pacemaker.

Case Report

Mr. A, a 78-year-old man with a history of bipolar affective disorder, was admitted to the hospital during an episode of severe depression with endogenous features. For 1 month his symptoms failed to respond to 250 mg/day of imipramine. Mr. A's tricyclic blood level of 74 ng/ml was not in the range (>180 ng/ml) associated with frequent antidepressant response. He also had at least 1 year's history of chronic slow atrial fibrillation probably due to arteriosclerotic heart disease. One year before this admission he had a transvenous permanent unipolar demand pacemaker (CPI 0505) implanted following two instances of syncope; during each syncope Mr. A's ventricular rate was 45 beats per minute.

Because of the severity of Mr. A's depressive symptoms and his poor response to tricyclics, he was treated with five bilateral ECTs (140 volts for 1 second), and he had an excellent antidepressant response. Before ECT was applied appropriate X rays and ECGs were taken of Mr. A under different body positions to establish that the pacemaker's electrode was in place and operating properly. He was medicated before each treatment with 300 mg of pentobarbital i.v. and 60 mg of succinylcholine i.v., and his heart was monitored by a regular ECG machine that was plugged in immediately after ECT was delivered. Precautions were taken to keep Mr. A from coming into contact with a low resistance electrical pathway. The ECT room had an insulated floor. As a precaution in case Mr. A developed a prolonged low pulse rate a magnet was available; it could be placed on the pulse generator to convert the demand pacemaker to a fixed rate if necessary. The only complication we observed was the development of infrequent premature ventricular contractions that lasted for a period of 3–6 minutes following each ECT. Although premature ventricular contractions inhibited the pacemaker, Mr. A's pulse rate was never reduced for any significant period, and thus no intervention was required.

Discussion

Implanted cardiac pacemakers are used for a variety of heart diseases in patients who have impaired electrical conduction resulting in dangerously decreased cardiac output (1). Three main types of pacemakers are used—fixed-rate pacemakers, synchronous pacemakers that are triggered by R or P waves, and demand pacemakers that are inhibited by R or P waves (1). Use of ECT on patients with pacemakers requires certain considerations.

A high percentage of ECT-induced arrhythmias result from vagal discharge. Premedication with atropine is unnecessary because the pacemaker protects the patient from vagotonic arrhythmias (2–5). Atropine is relatively contraindicated in patients with fixed-rate pacemakers. The atropine-induced tachycardia increases the chance of competition of heart beats with pacemaker beats and thus may lead to dangerous arrhythmias (4, 5).

During ECT only a minimum amount of electricity reaches the heart because the body tissues have high resistance. However, if the insulation of an electrode of the pacemaker is broken or if the patient is in contact with a low resistance pathway, e.g., an improperly grounded monitor or an assistant in contact with the patient and a noninsulated floor, a large current will pass through the heart and may result in ventricular fibrillation and sudden death (4, 6). Therefore, patients should be examined for intermittent pacing related to body posture, and appropriate chest X ray films should be taken to insure that the electrode's insulation is intact. Heart monitoring may be done safely by telemetry, by a battery-operated ECG machine, or by a regular ECG machine that can be plugged in immediately after ECT is delivered.

The pacemaker is not affected by ECT discharge because it has a built-in protection for large electrical potentials (4). However, skeletal muscle potentials that occur during ECT may fall within the range of the pacemaker's sensing circuit and inhibit the demand pacemakers (7). For this reason it is necessary sometimes to convert the demand pacemaker to a fixed-mode pacemaker if severe bradycardia occurs. The majority of pacemakers have a magnet-activated switch that allows this conversion to be made by placing a magnet over the pulse generator. The pacemaker returns to a demand mode when the magnet is removed. The magnet-activated switch can also be helpful in evaluating whether the pacemaker is capable of providing the programmed rate (8). The magnet may be used prophylactically on patients who have manifested pacemaker inhibition during previous ECTs.

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Approximately 78% of patients with heart disease develop premature ventricular contractions during ECT (2, 3). These contractions may compete with fixed-rate and synchronous pacemakers, and thus they increase the chance of the patient's developing dangerous arrhythmias. If premature ventricular contractions lead to ineffective heart contractions, as occurred with Mr. A, the contractions may inhibit the function of the demand pacemaker and result in prolonged pulse pauses. This complication is relatively benign and can be effectively treated by converting the demand pacemaker to fixed mode if severe bradycardia occurs. Antiarrhythmic drugs like procainamide, quinidine, and propranolol have been used prophylactically in order to avoid arrhythmias following ECT (9, 10); these drugs may be used prophylactically in patients who developed dangerous arrhythmias during previous ECTs.

Tricyclic antidepressants may also be used safely in cardiac pacemaker patients because the pacemaker protects against bradyarrhythmias due to conduction defect. The sinus tachycardia due to tricyclics' anticholinergic properties is not a problem for those patients with A-V block, a group that constitutes the majority of patients with pacemakers. Certainly these patients are at risk for ventricular tachycardias, but it is questionable whether this risk is higher than for patients without a pacemaker. The decision to use tricyclics or ECT depends on a clinical judgment that takes into consideration the patient's psychiatric indication and the patient's heart condition.

ECT is a reasonably safe procedure in patients with pacemakers if the following precautions are taken before treatment begins: 1) the clinician is aware of the nature of the patient's heart disease and the type and properties of the pacemaker used, 2) appropriate X rays and ECGs are taken of the patient to establish that insulation of the electrodes is intact, and that placement of the electrodes is proper, 3) a magnet is available to convert demand pacemakers to fixed mode if necessary, 4) premedication of the patient with atropine is avoided whenever possible, 5) every precaution is taken to keep the patient from coming into contact with a low resistance pathway, and 6) the patient's heart is monitored.

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Cimetidine Toxicity Manifested as Paranoia and Hallucinations

BY LAWRENCE E. ADLER, M.D., LEE SADJA, M.D., AND GREGORY WILETS, M.D.

When cimetidine, an H_2-receptor antagonist, was first released as treatment for peptic ulcer disease, no significant CNS side effects were noted (1). Investigators considered its major action to be a competitive inhibition of histamine type-2 receptors in the gastrointestinal tract that reduced acidity levels by interfering with the action of histamine. During the past few years there has been an increasing number of reports in the English and American literature of acute confusional psychoses associated with cimetidine toxicity. This literature was reviewed by Barnhart and Bowden (2). Predisposing factors include high-dose medication, older age, preexisting psychiatric illness, poor renal function, cerebral impairment, and simultaneous treatment with psychotropic medications. Recently we observed a somewhat different presentation of CNS cimetidine toxicity occurring in a paranoid patient who experienced vivid hallucinations.

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Case Report

Mr. A, a 51-year-old man, underwent an aortofemoral bypass operation for claudication. He had no other medical problems, history of drug or alcohol abuse, or previous psychiatric treatment. Mr. A had an uncomplicated postoperative course except for minor GI bleeding. Routine postoperative medications included suppositories, cephalothin sodium, meperidine hydrochloride, 100 mg i.m. every 3–4 hours, and hydroxyzine pamoate, 25 mg i.m. every 3–4 hours. Thirty-six hours after the operation, Mr. A received a single 300 mg-dose of cimetidine intravenously for minor GI bleeding. Over the next 72 hours, Mr. A received 9 doses of cimetidine, 300 mg p.o.

Twenty-four hours after cimetidine therapy was begun, Mr. A became belligerent but responded to reassurance. His physician discontinued meperidine hydrochloride and hydroxyzine pamoate. Forty-eight hours after the initiation of cimetidine therapy, Mr. A became extremely agitated, with visual and auditory hallucinations that he said were "like pornographic movies." He also had difficulty finding his room. Sixty hours after the initiation of cimetidine therapy, Mr. A's visual hallucinations subsided. His physician then requested a psychiatric consultation.

Mr. A was extremely paranoid but revealed no major abnormalities on formal mental status examination. He was oriented to time, person, and day of the week, but missed the date by 2 days. He could recall 3 objects after a 5-minute time lapse. His digit span was 7 forward and 3 backward; he was later able to recall 5 backward. He performed serial sevens correctly but had some difficulty concentrating. Mr. A had a normal speech pattern, his judgment was intact, and he could abstract proverbs well.

We discontinued cimetidine, gave Mr. A 1 mg of haloperidol p.o., and transferred him to the psychiatric service. His psychosis resolved within 36 hours of discontinuation of cimetidine; Mr. A received no further psychotropic medication.

Discussion

Previous reports of cimetidine toxicity have emphasized the patients' confusional state, bizarre speech, and amnesia for the episode. In contrast, Mr. A demonstrated a relative lack of confusion behaviorally and on formal mental status examination. His speech was normal and he had no amnesia. The most striking features of Mr. A's cimetidine toxicity were paranoid delusions, with visual and auditory hallucinations that persisted after the resolution of his initial confusional state. Mr. A did not exhibit any of the predisposing factors to toxicity, although one might speculate that given the severity of the atherosclerotic process in his femoral arteries, there might be subclinical cerebral artery disease.

From this case report it becomes apparent that cimetidine toxicity can trigger a predominantly paranoid psychosis that must be differentiated from other functional or organic paranoid psychoses. Physicians treating patients with cimetidine should be alert to prodromal personality changes, like Mr. A's sudden belligerence, as possible indications of impending psychosis.

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A Case Report of Lorazepam Withdrawal

BY RONALD B. STEWART, M.S., RONALD B. SALEM, AND PHILIP K. SPRINGER, M.D.

Reliable reports of true addiction to benzodiazepines have existed in the medical literature since 1961. Hollister and associates (1), in the first published report on the topic, produced signs of withdrawal in 10 of 11 subjects who had been given high doses of chlordiazepoxide. These symptoms did not appear until the third or fourth day following discontinuation of the drug and peaked on the fifth day. Most published reports of benzodiazepine withdrawal in this country have involved long-acting agents such as diazepam and chlordiazepoxide; however, there are a few reports (2, 3) involving oxazepam, a short-acting agent and more recently one report involving lorazepam. Benzodiazepines have a shorter half-life, and theoretically they should produce a more abrupt onset of withdrawal because discontinuation should result in a rapid fall of plasma levels.

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We will describe a patient who experienced an apparent withdrawal reaction after ingesting large doses of lorazepam.

Case Report

Ms. A, a 51-year-old woman, was admitted to the inpatient psychiatry facility of the Shands Teaching Hospital with a referring diagnosis of involutional depression. She complained of insomnia, fatigue, and nervousness, and stated she no longer looked forward to things. Ms. A had noticed these symptoms 3–4 years earlier; they were mild initially but gradually worsened.

Ms. A's medications at the time of admission, according to her recount, included 75 mg of imipramine h.s., 1 mg of lorazepam t.i.d., and 1.25 mg/day of conjugated estrogens. For 4 years she had used increasing doses of diazepam, but when she developed a problem of habituation, her prescription was changed (4 months before her admission) to lorazepam.

Ms. A's initial physical examination was within normal limits. Her mental status examination on admission showed a normal thought process, and she was alert and oriented; however, her affect was flat. Ms. A's laboratory studies were within normal limits except for a mildly elevated lactate dehydrogenase.

On the evening of her admission Ms. A began to complain of progressively worsening nausea, which did not respond to repeated doses of prochlorperazine i.m. The nausea persisted for about 36 hours. Approximately 48 hours after admission Ms. A became very disoriented and rigid, with her head tilted back; she maintained catatonic stances and would not respond to verbal commands. She also displayed a staggering, stumbling gait disturbance and clutched at air, walls, and other objects for support. By the fourth hospital day these symptoms disappeared and Ms. A stated that during the last 2 days her body "refused to do what my mind asked it to do."

On the fourth day of Ms. A's hospitalization her mental status cleared, and a drug history obtained from her revealed that she may have been abusing lorazepam. A phone call to her local pharmacy revealed that Ms. A had received a prescription two months earlier for 100 2-mg lorazepam tablets with directions to take 2 mg of the drug q.i.d. That same afternoon she returned to the pharmacy for a refill, stating she had misplaced the first vial of medication. She returned to the pharmacy approximately every 10 days for a refill of 100 tablets until she was admitted to our unit. A blood sample obtained the morning following admission (18 hours after admission) was sent for analysis and showed a lorazepam level of 31 ng/ml. Because the blood sample was sent to an out-of-state laboratory, results of this analysis were not made available to the staff until after Ms. A had been discharged.

Ms. A remained in the psychiatric unit for 2 weeks and was treated with imipramine. No further episodes of bizarre behavior were noted, and she was discharged and seen thereafter by a local psychiatrist.

Discussion

Withdrawal symptoms after discontinuation of benzodiazepines have been noted in the medical literature for nearly 20 years. Diazepam and chlordiazepoxide have been noted to induce withdrawal symptoms 3–4 days following discontinuation of medication. Although little information exists concerning withdrawal from short-acting benzodiazepines, the pharmacokinetic properties of lorazepam suggest that signs and symptoms would occur sooner after discontinuing use of this drug than with longer-acting agents. Most benzodiazepines have long half-lives and active metabolites that allow for a gradual reduction of blood levels. The half-life for lorazepam is approximately 12 hours, and no metabolites of major importance other than lorazepam glucuronide have been found with this drug (4, 5).

The rapid appearance of gastrointestinal symptoms observed in Ms. A would be expected after discontinuation of lorazepam. It was not clear when she took the last dose of lorazepam; however, 18 hours after admission her drug level was 31 ng/ml. Based on this drug's half-life, her lorazepam level at the time of admission could have been more than 100 ng/ml. Although Ms. A's mental status changes and physical condition could have resulted from a hysteric reaction, the history and blood level data make drug withdrawal a more likely etiology.

This case report should serve to alert physicians to the importance of obtaining an accurate medication history of patients who have a potential for excessive or long-term use of benzodiazepines. It should also be apparent that the newer, short-acting benzodiazepines also have dependence and withdrawal potential.

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Changing Perceptions of the Mental Health Needs of Inmates in Local Jails

BY HENRY J. STEADMAN, PH.D., AND STEPHEN A. RIBNER

The complex relationship between mental illness and crime, which has vexed researchers and policy makers for centuries, has taken on renewed importance during the current controversies over changing mental health statutes and their impact on the criminal justice system. Correctional administrators, especially those at local jails, strongly contend that their facilities have become depositories of persons who should be, and formerly would have been, in mental hospitals. Several clinicians support these observations (1-3). Furthermore, the mental health services in criminal justice settings, especially in local jails, are receiving new attention from the American Medical Association Standards for Psychiatric Services in Jails and Prisons and in various journal articles (4).

At the same time these mental health problems are being discussed in a criminal justice context, the staffs at state mental hospitals are concerned because recently admitted patients are now more agitated, hostile, and assaultive (5). In at least one state's mental hospitals the proportion of men with previous arrest records has dramatically increased over the past 30 years (6). Thus, while correctional administrators see themselves as inundated with mentally ill offenders, state mental hospital staffs perceive a significant increase in newly admitted patients who have problems associated with violence—problems that could or should have been handled by the police. Because these views have been expressed frequently by many highly respected professionals in the correctional field they must be considered seriously. In the data reported here we attempt to clarify the accuracy of the correctional staffs' perceptions that the mental health needs of their inmates are changing.

Method

The data reported here were drawn from a larger study (7) of all persons released to Albany County from New York state prisons and the local Albany County Jail in 1968 and 1975. We obtained each offender's mental hospital history from state mental hospitals as well as "rap sheets," which included a complete criminal history as well as data on the offender's subsequent criminal behavior for the first 18 months after his or her release. The previous mental hospitalizations of these offenders may be underestimated because only state mental hospitalization data were available; however, this will not distort any differences across our study groups or time periods.

Results

In 1968, 167 offenders were released from correctional institutions to Albany County—91 inmates from state prisons and 76 inmates from the local jail. In 1975, 252 offenders were released—76 inmates from the state prison and 176 inmates from the local jail. We found that 93% of all offenders in 1968 and 90% in 1975 were men; they had a mean age of 32 years and 28 years, respectively; and in 1968 and 1975 the percentage of black inmates was 42% and 49%, respectively.

We examined first whether the proportion of inmates with previous mental hospitalizations released from state and local correctional facilities had increased from 1968 to 1975. We found no consistent or statistically significant trends. In 1968, 19% (N=17) of the offenders from state prisons had previous mental hospitalizations and in 1975 only 13% (N=10) had a previous mental hospital record. Of the 76 offenders released in 1968 from the county jail, 9% (N=7) had prior mental hospitalizations and 12% (N=21) had such histories in 1975.

Even though there was a rise of 3% in the number of inmates with prior mental hospitalizations released from the local jail, this rise would not explain the dramatic rise in the perceptions of the jail staff that they were seeing more mentally ill inmates. Similar data also have been reported for Colorado; 14% of the inmates admitted to the Denver County Jail in 1974 had had prior inpatient hospitalizations (8). In addition, we found that for those inmates with mental hospitalization histories released from the county jail, the average number of previous admissions to a mental hospital increased from 1.9 admissions in 1968 to 4.1 admissions in 1975. For the state prisoners the admissions rose from 1.5 admissions in 1968 to 2.9 admissions in 1975. Thus, at the local county jail level there is modest empirical support for the altered perceptions of the correctional staff, but the empirical data do not support the vast increase in the perception of the problem. It may be that the same offenders are coming through both systems more often.

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We analyzed next the relationship between the mental hospitalization records of the offenders and subsequent crimes committed by them. We could find no relationship. In 1968 3.5% of the state and local offenders without prior mental hospitalization were arrested for violent crimes and 4.2% of those with previous mental hospitalizations were arrested for violent crimes. The proportion of offenders arrested for violent crimes and released in 1975 increased to 16.1% of the offenders with prior mental hospitalizations and 12.7% of the offenders without prior mental hospitalizations. Clearly, however, neither of these differences is of any consequence. There were no substantial differences among the offenders rearrested for violent crimes based on their previous mental hospitalizations. There was also no relationship between a history of state mental hospitalizations and subsequent arrests made within 18 months after the offenders were released.

It is difficult to determine whether the psychopathology of inmates is increasing as a result of more restrictive civil commitment statutes or if correctional administrators have a new set of expectations about psychiatric contributions to correctional institutions. There is little empirical evidence to show that the mental health problems of inmate populations at local or state correctional institutions have changed, and in a very limited way the data presented here suggest that the changes may have occurred in the perceptions and expectations of the correctional staffs rather than in the characteristics of the inmates. More empirical evidence is required before one can accept at face value the claims of correctional administrators and some clinicians about the radical changes in the mental health needs of U.S. jail inmates.

It is unfortunate that often the introduction of a psychiatrist into the jail is seen by the correctional staff as the sole solution to a wide variety of social and community problems. The limits of direct psychiatric intervention for disturbed inmates must be communicated to the correctional staff so that inappropriate expectations by the correctional staff will not doom the psychiatric services to failure in their eyes and contribute to the truncated development of the full range of social support services that are needed. As stated by Nielsen (4), it is important to articulate how the psychiatric services are related to other community mental health services. Contrary to the expectations of the public and often correctional staffs, psychiatric treatment goals do not generally include lowering the recidivism rate. Nevertheless, if a former inmate is rearrested, the failure is often attributed to the mental health treatment, which is usually incidental to the crisis intervention services that characterize the jail or prison treatment.

Thus, it is important that the goals and promises for mental health programs in jails and prisons be made very explicit. The character of the inmates' needs may not have changed to the extent that they have been perceived to have changed, but this fact does not diminish the current mental health needs of inmates. It simply means that the mental health problems that were found in prisons previously still remain.

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Prolactin Changes in Major Depressive Disorders

BY GREGORY M. ASNIS, M.D., R. SWAMI NATHAN, M.D., URIEL HALBREICH, M.D., FREIDA S. HALPERN, M.A., AND EDWARD J. SACHAR, M.D.

According to current knowledge, prolactin secretion is controlled by the inhibitory effect of dopamine and the stimulatory effect of serotonin (1). Alterations in basal prolactin secretion may therefore indicate central neurotransmitter changes. There have been inconsistent reports about basal prolactin levels in depression (during which hypothesized neurotransmitter deficits occur). Some researchers have reported no change in prolactin levels, while others have reported a small increase compared with levels in either normal subjects or in the same patients after recovery (2). We assessed basal prolactin levels in a group of depressed patients during illness and after recovery and found a small but significant increase in basal prolactin after recovery.

Method

We evaluated 19 medically healthy, depressed inpatients (15 women, 4 men), who gave informed consent to the research. Their mean age was 48.4 years±11.4 years. All of our patients met Research Diagnostic Criteria for major depressive disorder, endogenous subtype (3) and had Hamilton Depression scores (4) of greater than or equal to 18 (mean Hamilton score was 28.3±6.5). We determined the patients' basal morning plasma prolactin levels after at least 10 days of drug-free hospitalization and again approximately 6 weeks later after full clinical recovery (Hamilton score of less than or equal to 8). All but 3 patients were receiving antidepressant medication at the time of their clinical recovery. Seven patients received desipramine, 175–400 mg/day; 5 patients received imipramine, 100–375 mg/day; 1 patient received doxepin 150 mg/day; and 3 patients received Mianserin (a tetracyclic), 120 mg/day.

TABLE 1
Comparison of Basal Prolactin Levels in 19 Patients During Depression and After Recovery

Time of Blood Samples	Basal Prolactin Levels (ng/ml)			
	During Major Depressive Disorder		After Recovery	
	Mean	SD	Mean	SD
9:00 a.m.	8.7	3.2	12.5[a]	7.2
9:15 a.m.	7.5	3.4	11.3[b]	4.5
9:30 a.m.	7.0	3.2	10.7[b]	4.5

[a] $p<.02$, two-tailed t test.
[b] $p<.001$, two-tailed t test.

Our patients were awakened at 7:00 a.m. but continued to fast from the previous night. At 9:00 a.m. we inserted a cannula with a heparin lock into an antecubital vein and drew 3 blood samples at 15-minute intervals. We immediately centrifuged the samples at 4° C and stored the plasma at −20° C until it was assayed. We analyzed prolactin by homologous double antibody radioimmunoassay (5). We analyzed all samples in duplicate and in one assay. The intra-assay and inter-assay variations were 4.9% and 9.6%, respectively.

Results

Prolactin levels were significantly greater at each baseline point after patients had recovered than during their illnesses (see table 1). After we log transformed the data to equalize the variances, the Student's t test remained highly significant each time. However, even though there was a significant difference in prolactin levels during illness and after recovery, at both of these times prolactin levels remained within normal range. In the male patients prolactin levels ranged from 3.3 ng/ml to 11.2 ng/ml; in the female patients prolactin levels ranged from 4.6 ng/ml to 20.0 ng/ml (except for 1 female patient who had a prolactin value of 36.2 ng/ml during illness after the initial insertion of the cannula, which was most likely a transient stress response).

Discussion

Since all prolactin values, before and after recovery, were in the normal range, and since all but 3 patients were receiving antidepressants at the time of recovery, we could interpret our results in two ways: 1) as a modest but significant reduction in prolactin secretion, associated with depressive illness or 2) as a modest but significant increase in prolactin secretion associated with antidepressant therapy. We are now conducting

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further studies of recovered patients after antidepressant withdrawal or of patients before and after ECT-induced remission to clarify this issue.

If we find that relative reduction in prolactin secretion is associated with depressive illness, current controversial knowledge of neurotransmitter regulation of basal prolactin levels would implicate increased dopaminergic tone or possibly decreased serotonergic tone.

If we find that chronic antidepressant therapy modestly elevates prolactin secretion, we could attribute this to increased serotonergic tone or decreased dopaminergic tone. However, there is considerable controversy in the literature about the effect of chronic antidepressant therapy on prolactin secretion (2). Researchers who have assessed prolactin levels before and after chronic administration of tricyclic antidepressants in the same patients noted that amitriptyline and imipramine had no effect on prolactin levels but that clomipramine, a highly serotonergic drug, increased prolactin levels (6, 7). We treated our patients with various agents that had widely different potencies on neuronal serotonin reuptake blockade and little known antagonistic effect on dopamine transmission. All 3 patients who were tested unmedicated after clinical recovery also had increased prolactin levels. The universality of the relative increase we noted in 17 of 19 patients who received different forms of therapy might suggest a relative reduction in prolactin secretion during depressive illness as the most likely interpretation. An underlying mechanism of reduced serotonergic tone during depression would then seem to be the explanation most consonant with current biological concepts of depressive illness and with our incomplete knowledge of neurotransmitter regulation of basal prolactin secretion.

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Acetylcholinesterase and Pseudocholinesterase Activities in Anxiety

BY ROY J. MATHEW, M.D., LOUISE L. HSU, PH.D., KAREN M. SEMCHUK, B.S.N., AND JAMES L. CLAGHORN, M.D.

Cholinesterase, the enzyme responsible for metabolic degradation of acetylcholine, is presumed to exist in at least two forms: acetylcholinesterase (true or specific cholinesterase) and propiono cholinesterase (pseudo or nonspecific cholinesterase). Acetylcholinesterase hydrolyzes acetylcholine faster than pseudocholinesterase. Although the function of acetylcholinesterase is thought to be the hydrolysis of acetylcholine, no function has been ascribed to pseudocholinesterase (1).

Anxiety is associated with autonomic imbalance involving both adrenergic and cholinergic systems. Acetylcholine serves as the neurotransmitter at the ganglionic level for sympathetic and parasympathetic systems, motor nerve endings, parasympathetic postganglionic terminals, and sympathetic postganglionic terminals to sweat glands and adrenal medulla; all of these are involved in autonomic arousal. Seemingly, then, imbalances in the cholinergic system should occur in anxiety (2). However, only a few studies have been conducted in this area whereby investigators reported an increase in pseudocholinesterase activity in anxiety (3–5). In this study we examined the levels of true cholinesterase and pseudocholinesterase and compared the levels of anxiety in anxious patients and

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TABLE 1
Comparison of Psychological Measures and Cholinesterase Levels in Patients with Anxiety Disorders and Healthy Volunteers Before and After Relaxation Training

Measure	Patients (N=15) Mean	Patients (N=15) SD	Healthy Volunteers (N=15) Mean	Healthy Volunteers (N=15) SD	t[a]	Significance
Before relaxation training						
State anxiety	50.93	11.76	31.93	7.14	5.35	p=.001
Trait anxiety	53.93	9.53	30.53	7.56	7.44	p=.001
Acetylcholinesterase[b]	5.01	.96	4.94	1.50	.16	n.s.
Pseudocholinesterase[c]	2.59	.68	1.79	.61	3.35	p=.002
After relaxation training						
State anxiety	34.40	8.54	33.66	13.24	.18	n.s.
Trait anxiety	40.93	11.86	32.53	10.80	2.03	p=.05
Acetylcholinesterase[b]	4.90	1.04	5.19	.95	.79	n.s.
Pseudocholinesterase[c]	2.56	.92	1.74	.56	2.92	p=.007

[a]Two-tailed t test.
[b]Expressed as moles per milliliter of packed red blood cells per minute.
[c]Expressed as micromoles per milliliter of plasma per minute.

control volunteers before and after relaxation training sessions.

Method

A total of 15 outpatients, 6 men and 9 women (mean age±SD=38.26±12.03 years) with a diagnosis of generalized anxiety disorder (6), took part in our project. Most of the patients were not receiving any medication when they first contacted our unit; 3 were taking benzodiazepines and none were receiving monoamine oxidase inhibitors. All of the patients underwent a 2-week washout period before their participation in the study. Control subjects, 7 men and 8 women (mean age=32.80±7.92 years) were healthy, drug-free volunteers.

The procedures were fully explained to all potential subjects in lay terms, and we obtained written consent from each of them. At the initial visit physical examinations and routine laboratory tests were performed. The participants were instructed to avoid all forms of medication throughout the study.

Before the relaxation training began, cholinesterase levels and psychological measures were determined at the second visit. During this visit the participants were asked to relax on a couch in a supine position for 35 minutes in a semidark and quiet room. Then enzyme assay blood samples were drawn. At this time the participants also completed the State-Trait Anxiety Inventory (7), which is composed of separate self-report scales for measurement of two distinct anxiety concepts—state anxiety and trait anxiety. State anxiety is conceptualized as a transitory human emotional state or condition that is characterized by heightened autonomic nervous system activity and subjective, consciously perceived feelings of tension and apprehension. Trait anxiety refers to individual differences in anxiety proneness, that is, differences that exist among individuals in their tendency to respond to situations perceived as threatening with elevations in state anxiety intensity.

For the next 4 weeks the participants received 10 to 12 biofeedback-assisted relaxation training sessions lasting 35 minutes each; they were instructed to practice the exercises at home for 20 minutes per day. When the relaxation training was completed, the subjects returned to the laboratory for a posttraining evaluation, which was conducted according to the procedures used during the second visit.

We collected 5–7 ml of whole blood from each subject in a vacutainer containing EDTA; these samples were kept in crushed ice at 4° C for 1–2 hours. Then the sample tubes were centrifuged at 1,700 rpm for 5 minutes at 40° C to separate plasma and red blood cells. Plasma layers were transferred to plastic tubes for assay of pseudocholinesterase. After the platelets were removed from the interface, the red blood cell fractions were washed twice with 4 volumes of normal saline by gentle inversion and centrifugation at 2,000 rpm at 4° C for 5 minutes. The upper saline layers were discarded by aspiration and aliquots of red blood cell fraction were used for acetylcholine assay. All samples were drawn at the same time of day.

Enzyme activity of acetylcholinesterase in red blood cells and pseudocholinesterase in plasma was determined, with only slight modifications, according to the method of Garry and Routh (8). The procedure is based on the liberation of thiocholine from the substrate acetylthiocholine by action of acetylcholinesterase in red blood cell or from the substrate propionylthiocholine by action of pseudocholinesterase in plasma. The thiocholine then reacts with 5, 5′-dithiobis-(2-nitrobenzoic acid) to form the yellow anion of 5-thio-2-nitrobenzoic acid, which is measured at 410 mμ in a Beckman DB-G spectrophotometer.

Results

The patient and control groups were compared by levels of anxiety and enzyme activity using values taken before and after the relaxation training sessions, as shown in table 1. The posttraining session values for anxiety levels were compared with the pretraining ses-

sion values, and significant reductions were found after the training sessions for the index group only ($t=6.75$ and $t=5.89$, $p<.001$, for state anxiety and trait anxiety, respectively). There were no significant differences between groups before and after the relaxation training sessions in acetylcholinesterase and pseudocholinesterase activities. No meaningful correlations emerged when levels of enzyme activities were correlated against state anxiety and trait anxiety scores for the patient and control groups using pretraining and posttraining data.

Discussion

These findings of increased plasma pseudocholinesterase activity and unaltered true cholinesterase activity in anxious subjects substantiate the findings of previous investigators (3–5). The failure of the enzyme levels to decrease with anxiety relief is in keeping with the report by Richter and Lee (5), who found no significant alteration in the enzyme levels following administration of barbiturates. It must be noted that other conditions characterized by autonomic arousal (thyrotoxicosis and insulin shock) have been associated with increased pseudocholinesterase activity (9, 10). Because no physiological function has been ascribed to the pseudo form of the enzyme, the pathological significance of our finding of increased activity in chronically anxious patients is unclear.

These findings seem to suggest that increased plasma pseudocholinesterase activity may be indicative of a constitutional predisposition to anxiety. This possibility is weakened by the absence of significant correlations between trait anxiety scores and pseudocholinesterase activity. However, it must be noted that our finding of decreased trait anxiety scores following relaxation training raises a question about the validity of the trait anxiety score, which is considered a stable characteristic of the Inventory, as an index of constitutional predisposition. No firm conclusions can be drawn about constitutional predisposition to anxiety and plasma pseudocholinesterase based on the findings reported here.

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Clonidine for Opiate Detoxification: Outpatient Clinical Trials

BY ARNOLD M. WASHTON, PH.D., AND RICHARD B. RESNICK, M.D.

Recent studies (1, 2) have shown that a single dose of clonidine hydrochloride relieves signs and symptoms of opiate withdrawal and that addicts can be successfully detoxified with clonidine under the close supervision of an inpatient setting. The rationale for testing clonidine's efficacy in opiate withdrawal was based on animal studies showing that increased brain noradrenergic activity associated with opiate withdrawal was markedly reduced by clonidine (1).

The limited efficacy of standard detoxification methods, such as gradual methadone dose reductions, and the fact that clonidine is the only nonopiate drug shown to have specific antiwithdrawal effects underscores the need for further clinical investigation of clonidine's usefulness in opiate detoxification. If clonidine could be used not only for inpatient detoxification but also on an outpatient basis, it might become more widely available to patients and thus play a more significant role in addiction treatment. The inpatient clonidine dose regiment of 17 μg/kg used by earlier researchers (2) generated profound sedation and hypotension in patients, which necessitated keeping them supine at bed rest; therefore, determination of a safe and effective dose regimen for nonhospitalized ambulatory patients must precede any further evaluation of clonidine's suitability for outpatient use.

We have now administered clonidine to 70 outpatients addicted to heroin or methadone. We have found that clonidine is a safe and effective treatment for outpatient opiate detoxification when the dose regimen is individualized and titrated according to each patient's clinical course.

Method

Our subjects were 70 opiate-dependent outpatients who requested detoxification, had been addicted to opiates for at least 2 years, showed no evidence of serious medical or psychiatric illness, and gave informed consent to participate in the study after receiving a full explanation of the experimental procedures. Of the 70 patients, 59 patients were taking clinic methadone doses ranging from 5 mg to 40 mg/day (average dose, 20 mg/day) and 11 patients were using from $10 to $150 worth of heroin a day (average, $45/day). Twenty of the methadone-dependent patients received clonidine in conjunction with gradual methadone dose reductions, and the remaining 50 patients received clonidine after abrupt termination of methadone or heroin.

On day 1 of the study, all patients were instructed to take a 0.1-mg tablet of clonidine every 4–6 hours as needed for withdrawal discomfort. The total daily clonidine dose was then increased on each succeeding day by 0.1 mg or 0.2 mg to a maximum of 1.2 mg according to each patient's blood pressure and symptomatology. This was done to minimize the withdrawal discomfort without generating untoward side effects, such as oversedation and orthostatic hypotension. Clonidine doses were held or decreased in some cases to compensate for sedation or hypotension. Maximum daily clonidine doses averaged 0.8 mg and ranged from 0.3 mg to 1.2 mg. Patients who received clonidine during gradual methadone withdrawal were placed on a methadone dose reduction schedule of 5 mg or 10 mg/week and later switched to placebo methadone upon reaching a zero dose. Patients who switched abruptly to clonidine from heroin or methadone were placed directly on placebo methadone on the first day that clonidine was introduced.

Patients who were able to remain opiate free on clonidine for at least 10 days after their last dose of methadone or heroin, as indicated by opiate-free urines and verbal report, received a naloxone challenge of 2.0 mg i.v. to confirm readiness to begin post-detoxification treatment with the narcotic antagonist naltrexone so that continued abstinence would be fostered (3, 4).

Results

Detoxification success rates, defined by the number of patients who were able to remain opiate free on clonidine for the 10 days necessary to initiate naltrexone treatment, are presented in table 1 for the gradual and abrupt withdrawal procedures. These data show superiority of the abrupt withdrawal procedure as compared with the gradual procedure in helping patients achieve an opiate-free state. The data also indicate that patients who switched abruptly from methadone to clonidine were more successful than those who switched abruptly from heroin to clonidine. In general, patients using large amounts of heroin, i.e.,

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TABLE 1
Detoxification Success Rates[a] with Clonidine for 70 Outpatients

Withdrawal Procedure	Patients: Successful N	Successful %	Unsuccessful N	Unsuccessful %	Significance[a]
Gradual, from methadone (N=20)	10	50	10	50	p<.05
Abrupt (N=50)	35	70	15	30	
From methadone (N=39)	31	80	8	20	p<.01
From heroin (N=11)	4	36	7	64	

[a]By chi-square analysis.

$100 worth or more a day, and those taking more than 30 mg/day of methadone, experienced the greatest difficulty in completing detoxification. Clonidine reduced or eliminated most of the commonly reported withdrawal signs and symptoms, such as chills, lacrimation, rhinorrhea, restlessness, muscle pains, and stomach cramps. Lethargy and insomnia were the most frequent and persistent complaints. While taking clonidine most patients reported at least one episode of lightheadedness or dizziness upon standing. Six patients experienced unacceptable dizziness and/or sedation while taking only 0.3 mg/day of clonidine. Because most patients developed a tolerance to clonidine's sedative effect after taking the drug for several days, they required additional nighttime sedation to alleviate persistent insomnia.

Rapid decrements in the daily clonidine dose at the end of the study produced headaches and a reemergence of withdrawal symptoms. These ill effects were avoided by tapering the clonidine dose by 0.1 mg/day or 0.2 mg/day.

Discussion

This clinical study confirms clonidine's antiwithdrawal effects and demonstrates that outpatients can be detoxified with clonidine in a safe, acceptable, and effective manner. Our 80% success rate following abrupt withdrawal of 5–40 mg/day of methadone suggests that clonidine might be superior to the usual methadone dose reduction procedures that have typically shown much lower success rates (5). However, our rather high success rate in this study may have been due in part to nonpharmacologic factors, such as patient selection, daily contact with a supportive staff, and expectations of success with a new treatment. In order to obtain a more definitive assessment of clonidine's efficacy as a detoxification treatment we are now conducting a randomized double-blind outpatient study that compares clonidine and methadone detoxification procedures.

Our clinical experience in this study indicates that clonidine is a highly effective detoxification treatment for outpatients who are taking no more than 30 mg/day of methadone; however, no single clonidine dose regimen is best for all patients. In order to achieve a safe and effective control of withdrawal symptoms with clonidine, the dose regimen must be individualized according to each patient's blood pressure and symptomatology. Some patients are acutely sensitive to clonidine's hypotensive and sedative effects and cannot tolerate dosages large enough to control withdrawal symptoms. In most cases, however, a dose regimen of 0.1 mg or 0.2 mg every 4–6 hours with a larger dose at bedtime provides adequate relief of withdrawal symptoms with little or no untoward side effects.

Our evidence of clonidine's efficacy in opiate detoxification lends clinical support to the hypothesis (1) that noradrenergic hyperactivity is significantly involved in mediating the opiate withdrawal syndrome. This 10-day outpatient clonidine detoxification procedure may be extremely useful in allowing a rapid switch from opiate dependence to drug-free or opiate antagonist treatment without hospitalization.

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Differences Between Psychologists and Psychiatrists in the Use of *DSM-III*

BY LESLIE C. MOREY, M.S.

The recent introduction of *DSM-III* has stimulated new debate among mental health professionals. Schact and Nathan (1), for example, expressed the concern that psychology as a profession was not much involved in the development of this manual. As a result, one might suspect that psychologists would see *DSM-III* as less valid than would psychiatrists.

Other studies, e.g., that of Seegars and Miller (2), have demonstrated that professionals from different mental health disciplines conceptualize diagnostic categories and the corresponding symptomatology in differing ways. From a theoretical standpoint, this is almost certainly true. However, *DSM-III* eschews theory to a large extent and relies heavily on the identification of specific symptoms and behavioral signs in making a diagnosis. Once the clinician has made this identification the rest of the diagnostic process is more or less automatic. Thus, an investigation of professional differences might best be done at the symptom level.

This study attempted to assess whether psychologists and psychiatrists differ in their perception of the validity of *DSM-III* defined signs and symptoms. If one assumes extreme professional differences in diagnosis, the expectation would be that psychologists would see symptoms that were based on psychological research as highly valuable (e.g., "constructional difficulty—inability to assemble blocks" as diagnostic of dementia) and that psychiatrists would rely on traditional medical model symptoms. Although examining these possible differences was the primary focus of this study, another important clinician variable was also examined. This second variable, the level of experience of the diagnostician, has been proposed to be a salient factor in diagnostic studies (3).

Method

Participating in this experiment were 30 clinicians: 15 psychiatrists and 15 clinical psychologists. These clinicians ranged in level of experience from 1.5 years to 44 years (mean=13.6 years). Of the 30 clinicians, 6 (20%) were women. All clinicians worked in and around Gainesville, Fla., in many diverse settings. In total, 16 of the clinicians held full-time academic appointments in a teaching or Veterans Administration hospital, and 9 worked primarily in the community. The remaining 5 clinicians were psychiatry residents or psychology interns.

A card sort technique was used to obtain the clinicians' ratings. The stimuli were index cards with one symptom typed on the front. A symptom was defined as the smallest subunit of a *DSM-III* criterion that could be retained without losing its meaningfulness. Thus, a list of symptoms joined by an "or" was separated into individual symptoms. For instance, one criterion in the *DSM-III* definition of schizophrenia was defined as follows (criterion A-6): "Somatic, grandiose, religious, nihilistic or other delusions without persecutory or jealous content." In this study, that criterion was divided into 6 separate symptoms.

For the purposes of this study, three diagnoses were selected for intensive study: schizophrenia, mania, and dementia. In addition, symptoms were included from the personality disorder axis so as to present a less clear-cut diagnostic picture. In total, there were 47 schizophrenic symptoms, 20 dementia symptoms, 22 mania symptoms, and 15 personality disorder symptoms in the card sorting task.[1]

A high rate (94%) of all clinicians contacted agreed to participate in the study. At the time the study was undertaken *DSM-III* had not been officially put into use, and thus the clinicians were unfamiliar with the criteria from which the stimuli had been derived.

Each clinician was given a shuffled stack of index cards and asked to sort the cards into five piles. They were to arrange the cards from the lowest (value=1) to the highest (value=5) information/importance of a symptom they would use in making a particular diagnosis. The clinician was not told the diagnoses in advance. The categories used were (in random order) mania, schizophrenia, and dementia resulting from an organic brain syndrome. Hence, each symptom was rated on three disorders.

Results

To assure that profession and experience were not confounded factors, a test was performed to determine if the mean level of experience was different between the psychologists (13.70 years) and the psychiatrists (13.47 years). This difference was not significant ($t=.06$, $df=28$, $p>.95$).

An analysis of variance was then performed for each

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[1]A complete list of all symptoms is available from the author.

symptom rating using profession and years of experience as main effects and including the interaction term in the model. This resulted in a total of 314 analyses because all symptoms were rated on three diagnoses. A multivariate analysis of variance would have been desirable, but this was contraindicated by the large number of variables relative to the number of subjects. By examining the number of significant analyses we may obtain an idea of the effect of these clinician variables. Of the 314 analyses, only 18 analyses (5.7%) were significant at the $\alpha=.05$ level, and 2 analyses (0.6%) were significant at the .01 level. These numbers are essentially the number of analyses that would be expected to be significant by chance alone. Because in general all the clinicians rated the symptoms similarly (median correlation between clinician=.55), this finding cannot be an artifact of entirely unreliable data. On the whole, the clinician variables of profession and experience, as well as their interaction, were not significant factors in the rating of symptom validity.

Discussion

It is interesting that neither the profession nor the experience of a diagnostician had any effect on his or her perception of *DSM-III* symptom importance. Primarily, these results indicate that when psychiatrists and psychologists operate at a very specific level of evaluation, any global biases in their perspectives do not seem to have an effect. Thus, *DSM-III* may well have been effective in removing a possible source of diagnostic unreliability because it mandates decisions at this specific level. In addition, these findings indicate that the study of the clinician in diagnostic research has utility, at least when considering specific symptom information, because at this level theoretical bias tends to be minimized.

This study presents a new methodology for diagnostic research that might be very powerful if it were expanded beyond the somewhat limited sample of this study. Perhaps when the official diagnostic system is revised again, this model of studying the clinician's use of specific information might be applied.

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LETTERS TO THE EDITOR

This section contains reactions to Journal *articles, statements of opinion, brief research findings or ideas, short case descriptions, requests for information, etc. Letters should be submitted in duplicate to the Editor, who makes all decisions regarding publication. Letters must be typed double spaced throughout and should not contain more than 500 words and 5 pertinent references. Criticisms of published articles will automatically be sent to the author(s) for response. Letters will be edited for clarity and conformance with* Journal *style. We regret that we cannot inform writers of the disposition of letters or return those which are not printed.*

Propranolol, Chlorpromazine, and Tardive Dyskinesia

SIR: The exciting results reported in "Low-Dose Propranolol in Tardive Dyskinesia" (April 1980 issue) by Norman M. Bacher, M.D., and Harvey A. Lewis, M.D., warrant further investigation. However, we wish to alert the authors that chlorpromazine was recently demonstrated to significantly increase plasma levels and bioavailability of propranolol in man (1). Chlorpromazine (50 mg given orally t.i.d. for 5 days) significantly reduced intrinsic clearance and elevated steady-state plasma levels of propranolol (80 mg given orally t.i.d. for 3 days). There were no associated changes in liver blood flow, volume of distribution, half-time, or plasma binding of propranolol, and the investigators concluded that chlorpromazine inhibits the metabolism of propranolol.

If this interaction occurs at an enzymatic level, it is also possible that propranolol, by competing with chlorpromazine for enzymatic degradation, might increase plasma levels and bioavailability of chlorpromazine and other neuroleptics. It is well known that symptoms of tardive dyskinesia may be temporarily improved by increasing neuroleptic dosage, but that the symptoms may eventually worsen. Consequently, it will be important to monitor plasma neuroleptic levels in patients receiving combined neuroleptic-propranolol regimens, and to be aware that the combined administration may increase the toxicity of both agents.

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SAMUEL C. RISCH, M.D.
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Drs. Bacher and Lewis Reply

SIR: Dr. Risch and associates have provided important new information, postulating a possible mechanism of action of combined propranolol-neuroleptic in tardive dyskinesia. Propranolol may increase neuroleptic levels, thereby suppressing tardive dyskinesia symptomatology, and Dr. Risch and associates issue a caution regarding toxicity. They point out the importance of monitoring plasma neuroleptic levels in future studies using combined medications.

We are continuing trials with low-dose propranolol in outpatients with tardive dyskinesia who are receiving neuroleptics. In two resistant cases we have increased the propranolol dose to 80 mg/day, without side effects. Many of our psychiatric outpatients are seen for concurrent medical problems and receive neuroleptics combined with much higher propranolol dosages. We have not seen significant side effects or toxicity in these patients.

One of our original patients, who showed improvement in tardive dyskinesia with low-dose propranolol, was not receiving neuroleptics; this suggests that the improvement may have been caused by propranolol alone. We agree that further investigation is warranted.

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More on Suicide and Women Physicians

SIR: In "Suicide Among U.S. Women Physicians" (May 1979 issue) Ferris N. Pitts, Jr., M.D., and associates calculated the morbid risk for primary affective disorder in women physicians as 65.2%. This resembles a claim made by Craig and Pitts (1) more than 10 years ago. It is based on Pitts and Winokur's conclusions that all suicides are the result of primary affective disorder and that 10% of the deaths of women with primary affective disorder result from suicide. Aside from the issue of the first conclusion's validity, we question the applicability of the second conclusion to this situation. The figure of 10% is based on a sample of psychiatric patients from the general population. Women physicians constitute a subgroup that differs considerably from the total group of women diagnosed as having primary affective disorder. For example, socioeconomic status and intelligence probably differ between the two groups, and these variables may be associated with psychological factors significant to suicidal risk, such as impulse control and variety of coping mechanisms. Within a subgroup that was homogeneous with respect to these variables and diagnosed as having primary affective disorder, the suicide rate might be quite different from that of the larger group of patients with primary affective disorder. As the authors suggested, the only valid

way of determining the rate of primary affective disorder among women physicians would be through structured interview studies. Until these are reported, claims that two-thirds of women physicians suffer from primary affective disorder can only be viewed as highly questionable.

Women physicians have chosen to pursue professional careers in an area that has been traditionally male dominated. These women may not display some of the as yet unidentified aspects of psychological functioning that have served in the past to render women less vulnerable to suicide than their male contemporaries. With this possibility in mind, it is striking to note that Dr. Pitts and associates reported no statistically significant difference between the suicide rates of men and women physicians.

Suicide research would benefit greatly from an understanding of the factors underlying differences in suicide rates observed between men and women and between physicians and nonphysicians.

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Migraine and Schizophrenia

SIR: It is our clinical impression that patients with chronic schizophrenic illness do not suffer from migraine headaches. Vascular headaches occur in 4% to 15% of the population, and recently prevalence of migraine was reported at 7.3% in a general adult sample. The incidence of schizophrenia is generally estimated at 0.8% to 1%. On epidemiologic grounds, one would expect to occasionally see some schizophrenic patients with migraine. However, in our psychiatric clinics and our biofeedback laboratory we have yet to see a migraine headache sufferer with chronic schizophrenia, or a patient with schizophrenia who also has migraine headaches.

If this observation of negative correlation between chronic schizophrenia and migraine turns out to be valid, it might have heuristic value. Both disorders, migraine and schizophrenia, occur in families, suggesting the possibility of a genetic component. Abnormal monoamine oxidase functioning has been reported in both disorders (1, 2). Both conditions show some response to propranolol, a beta-blocker (3, 4). Most psychiatrists would agree that chlorpromazine, a phenothiazine antipsychotic drug, is useful in the treatment of schizophrenic illness. Chlorpromazine has been reported to be dramatically effective in prophylaxis against severe paroxysmal vascular headaches that are refractory to other measures. Methysergide, a 5-hydroxytryptamine receptor blocker, is useful in migraine prophylaxis, but reportedly aggravates symptoms in some chronic schizophrenics.

This may be looking for the proverbial needle in the haystack, but biological psychiatry has recently produced some interesting insights into the complexity of the schizophrenic syndrome. Perhaps an alteration of biogenic amines protects patients with schizophrenia from migraine, a quite common disorder. Meltzer (5) suggests that there could be a significant relationship between indoleamine and the dopamine hypotheses of schizophrenia.

We would be interested in the comments of *Journal* readers on this subject, especially clinicians who run large migraine clinics.

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DINESH MEHTA, M.D.
HOWARD WOODEN, PH.D.
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The Outlook for Children of Manic-Depressive Patients

SIR: In "Children at Risk for Manic-Depressive Illness: Possible Predictors" (September 1979 issue) Clarice J. Kestenbaum, M.D., theorized that the children of bipolar manic-depressive patients may exhibit symptomatology which does not take the form of classical primary affective disorder but reflects the "fundamental genetic liability" to the condition. This is an attractive hypothesis. Dr. Kestenbaum cited the report of McKnew and associates (1) and alluded to others (2) as support. However, after having interviewed or supervised the interviewing of 67 offspring of patients with bipolar manic-depression, I question Dr. Kestenbaum's proposition.

I originally intended to focus on the children of bipolar patients in the type of prospective study Dr. Kestenbaum advocated. After interviewing 19 such children (6-15 years old), I found that the psychopathology of the 11 I judged to be disturbed was not distinctive in any way from that of the rest of the patients in my clinical practice and did not appear to be definite primary affective disorder.

I shifted the focus of the study to the older offspring (16 years and older) who had entered the age of risk to primary affective disorder and for whom there were reliable primary affective disorder criteria. Of the 48 interviewed to date, representing the offspring of 16 bipolar patients (2 had committed suicide and 1 refused to be interviewed), 18 met the Research Diagnostic Criteria (3) for primary affective disorder and 8 for other psychiatric disorders. The mean age of the offspring was 27 years and the mean age of primary affective disorder onset was 16 years (range, 10-31 years).

Compared with their well siblings, those offspring with primary affective disorder did not show a decline in their achievement before the onset of their illness nor were they more likely to have previously come to the attention of psychiatrists or other mental health workers.

These results are in accord with those reviewed elsewhere (2). Thus, in answer to Dr. Kestenbaum's question, "What are the indications that a child with behavior disorder, hyperactivity, or neurotic symptoms may be at risk for future

manic-depressive illness, may be exhibiting a manic-depressive variant, or that the symptoms are evidence of manic-depressive illness per se?" one may reply that although the children of bipolar patients may be at enhanced risk to psychopathology and to school-related problems, unless the condition is clearly concordant with primary affective disorder there is no substantial evidence it will evolve into frank primary affective disorder.

Nevertheless, living with a parent suffering from bipolar manic-depression can in itself be a traumatic experience and may explain some of the symptomatology that younger offspring exhibit, either as a reaction to, or an identification with, the sick parent.

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Dr. Kestenbaum Replies

SIR: Dr. Waters has observed that in his clinical evaluation of the offspring of bipolar manic-depressive patients, only 11 of the 19 children interviewed (aged 6–15 years) were psychiatrically disturbed; the type of psychopathology, moreover, was not found to be distinctive in any way from that of other patients in his clinical practice. My intention has been to search for possible biological markers before the appearance of clinical symptoms, which are usually manifest by late adolescence in those destined to have a manic-depressive disorder. Thus I noted the finding that psychological test scores of children of bipolar probands had, to a significant degree, a substantial verbal-performance WISC differential in the direction of lowered performance scores. I agree that the clinical symptomatology itself was not distinctive, but coupled with positive family history and lowered performance scores, it suggested vulnerability to a manic-depressive disorder in adult life.

I have noticed that many children of manic-depressive patients, even when exhibiting some psychopathology, were not given psychological tests. Often these tests were omitted because the children were bright, verbal, and functioning well at school. A double-blind study currently under way should help to clarify some of these issues. I certainly agree with Dr. Waters about the traumatic effects of living with manic-depressive parents; psychopathological reactions in the face of intrafamilial stress cannot be ignored.

CLARICE J. KESTENBAUM, M.D.
New York, N.Y.

Empirical Data and Insanity Acquittals

SIR: Henry J. Steadman, M.D.'s call for empirical data to support legislative and administrative decisions in "Insanity Acquittal in New York State, 1965–1978" (March 1980 issue) deserves hearty support. Dr. Steadman showed that data from different states can add to our general understanding of the effects of different legal procedures. However, data derived only from mental health files, correctional admissions, or the judicial system cannot tell all that is needed to trace the operation of a statute.

Comparing insanity acquittals with corrections admissions on the basis of sex and ethnic background could enhance the meaning of other cross-departmental comparisons within the same state. The value of data coordinated among several governmental departments may be further illustrated by questions that arise from review of Dr. Steadman's tables.

For data arranged by type of crime, the disparity between the relative frequency of offense and insanity acquittals is striking. Burglary, a frequent offense, leads to only 2.5% (1965–1976) or 7.3% (1976–1978) of insanity acquittals, while murder, a far less frequent offense, leads to 53.4% or 43.5% of insanity acquittals. New York criminal justice data on the frequency of different offenses, frequency of prosecutions, and frequency of insanity pleas, considered with Dr. Steadman's data, could tremendously enhance efforts to trace the workings of insanity defense statutes.

Cross-departmental data would be indispensible in objectively analyzing whether the higher rate of insanity acquittals for murder results more from a murderer's psychological condition (compared with a burglar's psychological condition), from underlying assumptions by trial participants (murder is insane, but burglary isn't), or from trial participants' contemplation of the severity of punishment should the defendant be found guilty. Judicial system information about the prevalence of both pleas and acquittals, arranged by judicial district, by court, by prosecutors, by defense attorneys, and even by expert witnesses could also help in analyzing the operation of such laws and local ground rules.

Dr. Steadman is more aware than most of us of the administrative difficulties in attempting to collect systematic and comparable data from different state departments. However, if his call for more empirical foundations for policy decisions is to be adequately implemented, some central authority must mandate funding for studies that cross departmental lines within the same state.

DAVID W. ROSE, PH.D.
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A Sterile Theory of Depression?

SIR: Only with some trepidation would I criticize the work of Dr. Aaron T. Beck, who has contributed so much to our knowledge of depression; yet it would be a disservice to him to withhold a candid response. Despite the success of his cognitive therapy, Dr. Beck's view of depression as a primary thought disorder is fallacious as discussed in *Cognitive Therapy and the Emotional Disorders* (reviewed by Mardi J Horowitz, M.D., December 1979 issue).

In response to Drs. Kovacs and Beck's "Maladaptive Cognitive Structures in Depression" (May 1978 issue) Mark T. Schreiber, M.D., pointed out that "the authors interpret in a causative role what others have considered to be merely the symptoms or effects of depression" (1). Drs. Kovacs and Beck replied "that the cognitive model has a greater explanatory power of the phenomena of depression than other

psychological models presented to date. The model, however, does not address the ultimate etiology or cause of unipolar depression, which may encompass hereditary predisposition, faulty learning, brain damage, biochemical abnormalities, or any combination thereof" (2).

I agree that current theories of depression are limited. However, regardless of ultimate etiology or cause, the primary level of psychopathology of depression is affective, not intellectual, although an intellectual approach in therapy may be successful. Unfortunately, the clinical sciences have a relatively minimal effect upon unipolar depression given the incidence of this syndrome in our society today. We urgently need a fresh approach to this ubiquitous problem. Perhaps something as simple as viewing depression as the absence of pleasure might point us in a more positive direction. I do not mean to underestimate the complexity of the problem but it does seem that current psychological theories of depression have been sterile.

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Apartheid and Psychiatry

SIR: Distance has shielded me for almost 18 years from having to grapple mentally with the complex political situation in South Africa. However, the stirring Report of the Committee to Visit South Africa (November 1979 issue) demands that from my knowledge of my native land until 1962 I either agree with the observations, repudiate them, or comment constructively.

Unfettered political comment in South Africa became increasingly difficult after 1948, when the current governing party first gained control on the strength of their apartheid policies. The battle lines were drawn and the crossfire between white Afrikaner Christian nationalism and black African nationalism became a real threat to those who could not comfortably identify with either group. For those like myself, progressively extruded by a process which made little allowance for compromise or for shades of political difference, the conscious choice to leave became more attractive. After the 1960 Sharpeville event, the emigration of those who felt disenfranchized gained momentum, and the meager ranks of South African psychiatrists became more meager still.

I believe I have some understanding of the conflict confronting my South African colleagues who attempt to present South African psychiatry in a favorable light. I personally have no knowledge of the Smith Mitchell operations, but am familiar with the "bachelor compounds" for the black mine laborers and completed part of my psychiatric training in both Weskoppies and Sterkfontein, two of the mental hospitals visited by the committee. In 1977, when the accusations were first levelled at South African psychiatry for incarcerating political prisoners in mental hospitals and abusing ECT and psychotropic drugs for political purposes, I knew them to be absurd, and I knew from my contact with former colleagues that no major shifts in mental hospital policy had occurred since my departure in 1962. The APA committee's findings confirmed that view. The APA committee also confirmed that the psychiatrists and the professional staff they interviewed were hospitable, gracious, and candid. They were not evil people.

South Africa is unique in its intense contrasts and contradictions. Black domestic servants daily touch on the most intimate aspects of their white employers' lives, but genuinely share few experiences. The committee aptly described this paradox: "We have tried to be as objective as we could be, as foreigners confronting for the first time a country whose astonishing physical beauty and hospitable people were in such stark contrast to the ugliness of its apartheid system."

The South African ruling party has been consistently returned to power by the white electorate for more than 30 years. It is driven by the long-standing Afrikaner fear of being overwhelmed not only by blacks but also by whites of any different ethnic or religious background. Despite the opportunity to create a more comfortable racial balance by encouraging white immigration, when this governing party gained power in 1948 it immediately restricted the flow of immigrants from Europe. Having lost opportunities to change gradually, many members of the governing party have become locked even more rigidly into their apartheid posture, believing any shift that increases the financial or political power of blacks will hasten black majority rule.

Most of the APA committee's observations probably reflect or approximate the true state of affairs. Apart from some exceptional facilities, hospitals for blacks provide less comfort and fewer amenities. If the medical records the committee inspected reflected no physical examination before transfer from the mental hospital to the Smith Mitchell facilities, one must assume that no such examination was done. This applied in larger measure to the black patients, yet the records of some white patients similarly lacked any reference to a physical examination. An inspection of U.S. state hospitals and the medical plight of our deinstitutionalized mentally ill would give us little to crow about, but the discrimination here is against the mentally ill, not on the basis of race.

The APA committee roundly condemned the policies of apartheid and the inferior care provided to black psychiatric patients. From their observations it would be difficult to draw other conclusions. However, I find myself unable to agree entirely that the committee's findings substantiated the allegations of social and political abuse of psychiatry. The absence of any evidence of the punitive use of psychiatric incarceration and treatment, of evil intent on the part of the caretakers, and the total disregard for political party affiliations among the mental health professionals characterize the system as vastly different from that which exists in the Soviet Union. The committee's criticism focuses essentially on the fact that the policy of apartheid has infiltrated deeply into every aspect of living, including psychiatric care. The quarrel is therefore with the official policy of apartheid, not with psychiatry or its practitioners who, sorely depleted in ranks, battle against great odds to provide what care they can in the best tradition of medicine.

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The Biochemistry of Panic and Depression

SIR: In "Mitral Valve Prolapse Syndrome in Agoraphobic Patients" (April 1980 issue) Jerry Kantor, M.D., and associates described the high rate of mitral valve prolapse syndrome (MVP) in agoraphobic patients. Like these authors and other clinicians, I have noted this in my patients and would like to comment on pharmacotherapy. Of a group of 5 agoraphobic patients with MVP (diagnosed by auscultation and/or echocardiography), 3 responded to imipramine and 2 to phenelzine after an unsuccessful trial on tricyclics. In all instances the drug response occurred within 1 week after initiation of an appropriate dose of medication. Dr. Kantor and associates suggested a feedback loop in this syndrome, partially dependent on the hyperadrenergic state demonstrated by Boudoulas and associates (1). This feedback loop is antithetical to a therapeutic tricyclic or MAOI response if one is an advocate of the catecholamine hypothesis. If one does not "buy" the latter paradigm, the idea of an antidepressant drug inhibiting a hyperadrenergic state poses some fascinating possibilities about the biochemistry of panic, depression, and the mechanism of action of antidepressant medication. How intimate is the relationship between panic and depression? Do both include a hyperadrenergic state? Do antidepressants exert a direct antiadrenergic effect?

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On Suing Psychiatrists

SIR: I would be most grateful if members of the profession would share any information on suits filed or threatened against psychiatrists for a publication on litigation involving psychiatrists.

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Failures of the Asylum or of Community Treatment?

SIR: In *The Death of the Asylum: A Critical Study of State Hospital Management, Services, and Care* (reviewed by Robert L. Okin, M.D., January 1980 issue) John A. Talbott, M.D., wrote, "State hospitals as they are currently constituted do not work, seem designed not to work, and have never worked."

I got the book to see for myself the evidence for this statement. I found a thorough exposition of why mental hospitals should not work and seem designed not to work. They are remote and big, managed by crises instead of by plan. They are over-regulated, buried in paperwork, have poor press, poor leadership, and are beset by legal and political interference. The staff is untrained, employee incentive is low, patients are lower class, budgets are insufficient, and the public and legislature are indifferent.

However, I found no evidence in the book of a scientific or research nature to prove that mental hospitals do indeed not work. In the long life of a public mental hospital chronic stayers and frequent repeaters accumulate, but there are also thousands who pass through who are never seen again. There is also the question of what the patients who make up the chronic population would be doing, if they survived at all, in the absence of the asylum. During the 1960s it became a truism that mental hospitals created all those chronic patients through mechanisms called dehumanization and institutionalization; this remains a truism in the minds of many. One of the problems that has beset asylums over the years is lack of funding for outcome research (although anthropologists and sociologists have come from academia to spend a few months looking at the cross-sectional picture). As far as I can ascertain there is relatively little research evidence to prove or disprove a thesis about whether asylums work.

Fort Logan Mental Health Center, one of the outstanding public mental health facilities in the 1960s, was designed to avoid the effects of institutionalization. Kraft and associates (1) documented the accumulation of hardcore, long-stay patients at Fort Logan after 4½ years of operation. They found the diagnosis of these long-stay patients to be "the old familiar one of chronic schizophrenia."

Dr. Talbott mentioned that the state hospital population decreased from a high of 558,992 in 1955 to 193,436 in 1976. This was largely due, Dr. Talbott implied, to an increased number of inmates in other kinds of institutions. No doubt there is an irreducible basic population of institutional dependents, but during the years 1955 to 1976 millions entered and left the mental hospital system, some never to return. Why are those who do return considered failures of the asylum rather than failures of community treatment?

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Intrapsychic Structural Effects of Psychiatric Research

SIR: In "Intrapsychic Structural Effects of Psychiatric Research" (December 1979 issue) Samuel G. Siris, M.D., and associates presented a stimulating, humane approach to some major issues about inpatient psychiatric research. Although I am in almost complete agreement with them (1), I noticed several points that warrant further discussion.

First, I thought that the authors tended to switch back and forth between the use of a phenomenologic model (capacity for delay) and the use of a psychoanalytic approach or model (relationship with the superego). I found the article to be particularly interesting when the authors concentrated on their observations and on the clinical vignettes, e.g., the patient who interpreted blood drawing as an act of rape. Did the authors feel that discussion of structural theory and superego constructs was needed to make their major points? Why include the additional philosophical/structural constructs?

Second, the concluding paragraph stated that "many aspects of the research may well be adopted into the patients' reintegrating structures," implying that this may well affect therapeutic outcome. It seems that such experiences mainly affect patients' behavior in the hospital, which is important for day-to-day management and for the building of the therapeutic alliance. To the extent that such alliance building is important and may affect outcome, the authors may be correct. Yet, outcome and psychic reintegration are complex issues. Without more convincing evidence on either issue, Dr. Siris and associates' more far reaching arguments are fundamentally unsupported. In my experience, these research issues are of notable but minor importance and are almost always "washed out" by such factors as individual medication response, family issues, and the availability and funding of adequate follow-up treatment. I am not convinced that the patients' intrapsychic structures and outcome are as strongly affected as the authors implied.

Finally, patient-research subjects must be treated humanely and fairly. It is sad that these patients are sometimes treated as research tokens. Although academic pressure was not specifically discussed in the article, it may be an important factor contravening the adoption of the procedures that Dr. Siris and associates recommend.

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Dr. Siris and Associates Reply

SIR: We appreciate Dr. Braff and associates' contribution to the literature on the interactions between inpatient care and psychiatric research, and his thoughtful comments on our article. We are happy to take this opportunity to rectify some misunderstandings that our article may have engendered.

We regret if we confused readers with our descriptive presentation of clinical phenomenology within the conceptual framework of the psychoanalytic "structural" theory. We observed that this model of psychic functioning was quite valuable in understanding interactions between the reevolving organizational structure of patients' minds and the organizational structure of the ongoing research. We intended no phenomenological model. Rather our intention was to present empirically observed phenomena within the structural model. In this, for example, "capacity for delay," as an executive mental operation, is an ego function (1). Similarly, "superego" is a set of executive mental operations constituting a related part of the psychic structure. We are stimulated by Dr. Braff's implication that other models of the mind may provide a fruitful conceptual background for understanding the impact of constellations of external organization, such as research, on the human mind. We found that the psychoanalytic structural model of the mind was particularly felicitous for this.

In his second point, Dr. Braff generously extended the therapeutic impact of the phenomenon we described to the point of comparison with medication response, provision of follow-up care, and the understanding of family interactions. It certainly was not our intention to suggest that the influence of the structure of research on manifest psychiatric symptomatology was comparable quantitatively to those potent clinic modalities. Properly executed research is not supposed to be a clinical modality; it is supposed to be research. Nevertheless, the structure of aspects of research functioning did seem to have a clear influence on the structure of aspects of patients' psychic functioning. This was a more qualitative observation, which we found instructive and which did suggest to us that a process was indeed afoot that could potentially affect patients' adaptive capacities one way or the other.

In respect to Dr. Braff's third point, we could not agree more that humane and fair treatment of patient-research subjects is intrinsically right. Although it was not central to the specific thesis of our paper, we certainly join him in his concern that academic pressures may work to the detriment of the best patient care. Since these pressures are, unfortunately, natural and to a certain extent unavoidable, the judgment and expertise of every researcher stand to be challenged.

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Positive Experiences in Bipolar Patients

SIR: As reported in "Clouds and Silver Linings: Positive Experiences Associated with Primary Affective Disorders" (February 1980 issue) Kay R. Jamison, Ph.D., and associates asked groups of euthymic or mildly depressed unipolar and bipolar patients if their mood swings had affected such personality characteristics as overall psychological sensitivity, sexual enjoyment, productivity, creativity, and social outgoingness and ease. Although the two groups did not differ on the overall psychological sensitivity variable, bipolar patients reported significantly more positive experiences related to the effect of their mood swings on personality and productivity than unipolar patients did. The only apparent explanation offered for these group differences concerns the scales used to assess the bipolar patients during periods of hypomania and mania. Since these scales were designed to assess positive rather than negative attributes of affective illness, a possible positive response bias in the bipolar group may have existed.

Our research with depressed patients suggests that adaptive bipolar defense mechanisms provide an additional explanation for the results we obtained. First social desirability (e.g., subscribing to those characteristics that are desirable in our society so as to appear in the most favorable light) and depression are significantly related ($r=-.65$) in

bipolar depressed groups but are not related (r=.07) in unipolar groups (1). In contrast, individuals with high anxiety (e.g., the unipolar compared with the bipolar group [2]), seem to endorse socially undesirable response sets. Behaviorally rated compared with self-reported assessments of unipolar and bipolar groups do not elicit significantly different psychological characteristics (3). Second, bipolar defenses against the stress of depression appear to be considerably more adaptive than those of the unipolar group to the extent that self-reports of dysphoria and anxiety are attenuated in the former group (4). Thus bipolar groups are frequently described as "normal" when, in fact, this designation should be strictly limited to comparisons with unipolar groups. Third, bipolar depressed groups give evidence of their adaptive defenses on tests of self-report through the mechanism of denial (5), a defense long associated with hypomania. It can be inferred, then, that the more successful adaptation to depression in bipolar depressed patients compared with unipolar depressed patients presents a more positive, "normal," and healthy psychological picture.

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Tips from a Chart Browser

SIR: In this modern era, when we discharge psychiatric patients weeks or even days after they have been admitted and worked up, there is a need to assist trainees of all helping professions in writing up rapid case histories. To wit, I offer the following "tips"—apologizing for a term more redolent of off-track betting than of the medical model, although our passion for follow-up studies often makes us more interested in how our patients fare at the end of the race than how they present themselves at the starting-post of our institutions.

The following charting rules of thumb can be applied to all inpatients and to the vast majority of outpatients in a hospital setting:

1. You can always say that the patient is "suffering from low self-esteem." You will be wearing your more analytic hat, however, if you factor out the most successful aspect of your patient's personality functioning and then say that "this area of apparent strength is used to cover over issues of low self-esteem." This sort of statement is 100% safe to make in all cases—almost as safe, in fact, as saying that a patient's condition has "an underlying affective component." This is, after all, an age of intense affects.

2. Every patient should be said to have "constitutional factors contributing to (or predisposing to) the development of" an acute psychotic break, a major depression, et cetera.

3. Attribute your patient's peculiar inability to weather the stresses of life that assault us all either to (but never to both) "deprivation" or "overindulgence," preferably some time during the first two years of life. This follows a long psychiatric tradition of making use of opposites, albeit one at a time, to explain whatever phenomena one happens to be observing.

4. All patients are "ambivalent." Have you ever met anyone, your own analyst included, who didn't manifest "unconscious conflicts," especially around "sexuality and aggression"?

5. If the patient hates all the staff on the unit but likes you, then he is "capable of good object relations, if given the opportunity for a one-to-one relationship." If he likes everybody else but hates you, then he is clearly "splitting." Similarly, if you like the patient but hate your fellow staff, you can blame your patient's regressions on "tensions among staff" that your sensitive patient is alertly "responding to," and wow everyone by referring to these as "Stanton-Schwartz phenomena."

6. In the prognosis section of your case history be sure to make some statement implying that "the prognosis is guarded" if your miraculous treatment plan cannot be established.

7. If at all possible, look for and find: congenital stigmata, records of a low Apgar, soft neurological signs, abnormal reflexes, or a really loaded family psychiatric history. If you can't find these, call in anyone from a neurologist to a geneticist so he or she can find them.

8. For God's (and Bob Spitzer's) sake, never use or refer to or even dream about any diagnostic nomenclature other than the litany, *DSM-III*.

Now hop to it and catch up on that backload of charts you've been putting off for weeks; there's really nothing to it, once you've been around.

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Telephone Contact and Questionnaire Response

SIR: I am writing in response to Alvin A Rosenfeld, M.D., and associates' request (1) for reports of experience with a questionnaire respondent recruitment technique similar to the one they used in their study of Palo Alto families. Dr. Rosenfield and associates telephoned potential respondents to explain the purpose and design of their study before mailing questionnaires. This step increased their response rate greatly over that which is usually reported for mail surveys.

Last year I used a similar method with similar good results. I solicited the participation of 117 clinical psychology graduate students for the first part of a longitudinal study of professional development. Sixty-three were contacted by telephone; the other 54 were sent letters explaining the project with postcards to be mailed back if they were willing to participate. Ninety-eight percent of the graduate students contacted by telephone agreed to participate; 74% of them returned the questionnaires. Fifty-two percent of the graduate students contacted by mail agreed to participate; 79% of them returned the questionnaires. In Dr. Rosenfield and as-

sociates' study 97% of those contacted by telephone agreed to participate, and 86% of them returned the questionnaires.

The percentage of those agreeing to participate when contacted by telephone is virtually identical to the figure reported by Dr. Rosenfield and associates; a far smaller percentage of those contacted by mail were willing to fill out the questionnaire. However, once the decision to participate was made, those initially contacted by mail and by phone returned completed questionnaires at almost the same rate. This suggests that the crucial step in recruitment for such studies is securing the respondent's commitment to complete the questionnaire. Telephone contact is more effective in eliciting this initial commitment, perhaps because it allows the potential participant to ask questions and resolve any doubts he or she may have about the project. Telephone conversations also set up a quasipersonal relationship between researcher and respondent that makes saying no difficult.

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Correction

On page 849 of "Haloperidol Excretion in Human Milk" by Ronald B. Stewart, M.S., Barry Karas, and Philip K. Springer, M.D., in the July 1980 issue, the fourth sentence of the last paragraph should read, ". . . an infant could receive a maximum of 0.0075 mg/day of haloperidol."

BOOK FORUM

NANCY C. ANDREASEN, M.D., EDITOR

The Borderline Syndrome: Constitution, Personality, and Adaptation, *by Michael H. Stone, M.D. New York, N.Y., McGraw-Hill Book Co., 1980, 530 pp., $27.50.*

During the past decade many American psychiatrists have shown considerable interest in the group of disorders subsumed under the borderline rubric. Although competing theoretical formulations abound on the subject, very few empirical studies have been conducted. Some of these formulations, particularly those in the tradition of Melanie Klein, are not intelligible to many nonanalytical clinicians. Further, as Perry and Klerman (1) have cautioned, the concept is being used somewhat indiscriminately for an increasing number of heterogeneous patient populations. Neo-Kraepelinian psychiatrists have generally voiced dissatisfaction with the concept, which they find too nebulous for formal diagnostic purposes. Many dynamically oriented psychiatrists, on the other hand, point to the inherent clinical utility of a broad concept that identifies patients who are borderline in their suitability for psychoanalytic treatment. The views of the dynamic camp have been expressed more flamboyantly, but criticism by the neo-Kraepelinian camp has also sporadically appeared. The split of American psychiatrists on the subject of borderline patients is perhaps best portrayed by the November 1978 issue of the *Journal,* in which the lead article by Shapiro was devoted to an extensive theoretical review of the developmental psychopathology of these patients and the last article was a scathing critique of the concept by Rich.

Dr. Michael Stone has written a critical appraisal of the borderline concept that should satisfy both camps. Indeed, this is a unique book. The author, an analytically trained clinician, attempts to validate a concept developed largely within the analytical tradition by recourse to methods developed by descriptive and biological psychiatry. Because of this methodological advance, Dr. Stone's monograph can be considered a landmark in American psychiatry.

Beginning with Stern, analytical writers were concerned with the early identification of patients who were "falling apart on the couch." Within a linear dynamic logic of decompensation into psychosis, early discussions of borderline patients almost invariably referred to borderline schizophrenia (chapter 1). This was in line with the broader Bleulerian concept of schizophrenia. Eventually, however, Knight and others (table 7-1, pp. 163–164) foresaw the greater affinity of these disorders to the affective psychoses. The major clinical accomplishment of Dr. Stone's work is the convincing argument that psychostructurally defined borderline patients (according to Kernberg's schema) are on the genetic border of the affective rather than the schizophrenic disorders. By undertaking an extensive investigation of morbidity risks for affective and schizophrenic disorders in first-degree biological relatives of borderline probands (chapter 4), the author has shown a significant excess of affective but not of schizophrenic disorders.

Most previous workers have attempted to delineate the genesis of borderline psychopathology in developmental dynamic terms. Dr. Stone further traces this to its *literal* genetic origins:

> The suggestion of a hereditary factor is no new discovery. It was, if anything, taken for granted by the psychiatric and psychoanalytic communities until the second generation of psychoanalysts began to adopt a more linear and purely psychological model of causation. (p. 7)

In his critique of unidimensional models of character formation, Dr. Stone attempts to reformulate dynamic theories within the larger frame of psychiatry. He thereby renders an invaluable service to the discipline of psychoanalysis. Future contributions by the dynamic schools will be more meaningful when evaluated by the same canons of research methodology that psychiatry at large is finally adopting. Dr. Stone's monograph can serve as a touchstone toward such efforts because it is explicitly based on the premise that dynamic concepts—no matter how internally consistent—may eventually lose their heuristic value if not examined in the light of other approaches that can function as external validating criteria. For that matter, without susceptibility to such external validation, *all* psychiatric concepts are tentative at best.

The clinical popularity of borderline diagnoses in recent years has been disproportionate to their nosologic validity (1). Dynamic thinking on the characterological pathology may fascinate clinicians, but, unfortunately, it can also give them a sense of premature certainty about the origin and treatment of this diverse group of "primitive" personality disturbances. In defense of the clinician, it is only fair to mention that until recently borderline cases often could not be classified within traditional diagnostic categories.

Now that operational criteria—largely based on Gunderson's work—have been proposed by *DSM-III,* it is feasible to follow up such cases for eventual nosological clarification. In his review of recent nosological approaches (chapters 8–10), Dr. Stone concludes that only a small minority of borderline patients fit into the schizotypal group (genetically on the border of the schizophrenias), while the majority constitute such early or "dilute" forms of affective disorder as dysthymia, cyclothymia, and bipolar II (genetically on the border with affective disorders). Furthermore, Stone's probands were at high risk for developing affective disorders—both depressive and manic decompensations—during psychoanalytic therapy (chapter 8).

Thus, both phenomenological and genetic approaches seem to converge in placing borderline patients on the bor-

der of affective illness. (Other evidence that has appeared since the publication of this book and that supports this conceptualization is early escape from dexamethasone suppression (2) and shortened REM latency (3) in many borderline patients examined with the use of these biological markers for affective disorders.) The other two conceptualizations offered by the author include conditions borderline to the genetic interphase between schizophrenic and affective illness and an intermediary character formation between neurosis and psychosis. The former viewpoint is at present only a hypothetical possibility because the nosological status of schizoaffective states themselves is unsettled at this time. The latter viewpoint is not acceptable by contemporary nosologic standards (e.g., *DSM-III*), which find no evidence for the existence of a unitary "neurosis" or a "psychosis."

As the schizotypal disorders and the heterogeneous group within the affective spectrum—including the related substance use disorders—are more clearly delineated, the term "borderline" itself may become unnecessary (4). The burden of proof for sustaining the use of borderline as a distinct personality operation (on axis II of *DSM-III*) rests with authors who claim a more or less specific developmental psychopathology *generic* for this heterogeneous group of disorders.

It would have been preferable to address the nosological and phenomenological issues (chapter 8) before the lengthy chapter on the genetics of affective and schizophrenic disorders, which are competently discussed but are abundantly available in other books. Furthermore, case material could have been introduced at appropriate junctures into the main text of the book, instead of being relegated to a terminal chapter. Redundancy could thereby have been avoided, resulting in a text two-thirds of the present size. Fortunately the author's concepts are condensed and illustrated through the medium of diagrams and tables, of which figure 8-9 is perhaps the best.

Despite minor shortcomings in form, this is a very readable book. It should be read, however, at multiple sittings. The author's elegant style assists in achieving conceptual clarity. Indeed, both descriptive and dynamic psychiatrists who may have been mystified by the plethora of publications on the subject will find this monograph both lucid and informative. This very clarification, which may eventually result in the nosologic demise of the borderline concept as is happening with the dual parent concepts of "neurosis" and "psychosis," could paradoxically prevent the book from becoming a classic!

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Fin de Siècle Vienna: Politics and Culture, *by Carl E. Schorske. New York, N.Y., Alfred A. Knopf, 1980, 378 pp., $15.95.*

Books which are both heavy and light reading are a special breed indeed, but this book has both those qualities. It is an intensive exploration of the intellectual, social, and cultural life of Vienna at the turn of the century. It presents a convincing case that, much as tiny fifth-century Athens was the cradle of classical rationalism, *fin de siècle* Vienna was the source from which the tormented and irrational modern era was born. This book describes how that rather traumatic birth occurred. Its content is demanding; but its very richness and density also make it fascinating and exciting reading. New ideas and insights sparkle all through the book.

Its method is that of intellectual and social history, focusing on a small slice of time and exploring it in careful detail so that the grain and texture of the era become immediate and vivid. Schorske reconstructs the Vienna of Freud and his contemporaries as it existed during the final decades of the nineteenth century, showing how in one aspect of life after another an old order of tradition and discipline died and a new one emerged that emphasized individuality and rebellion. Schorske describes *fin de siècle* Vienna as perhaps the last city in Europe to remain small enough for the intellectual elite to know one another well and to exchange and cross-fertilize one another's ideas. One senses the excitement that must have prevailed in this unique environment as revolutionary new ideas were presented in one field after another—in the art of Klimt, the psychology of Freud, and the music of Schoenberg.

The book contains seven chapters, several of which were published initially in historical journals and which received wide recognition and acclaim among Schorske's peers. The first chapter sets the theme of the book by arguing that the crucial characteristic of our modern era is our loss of a sense of history, our lack of a sense of continuity with our past, even our desire to destroy the world that our fathers created. But our fragmentation has not only been longitudinal and chronological; it has also been cross-sectional and spatial. The various intellectual disciplines have become so independent of one another that art, science, and politics no longer aspire to communicate with one another. The age of the synthesizers and universal geniuses is gone, broken apart by an era of analysis and specialization.

Subsequent chapters explore how these changes occurred in various fields. The second chapter examines two literary figures of late nineteenth-century Vienna, Hofmannsthal and Schnitzler, showing how they struggled with the problem of the loss of traditional liberal ideas and solved it by substituting psychological and aesthetic values. The third chapter explores the relationship between architecture and social change. It contains a fascinating description of the development of the Ringstrasse, showing how Viennese taste in public buildings and private apartments mirrored the changes that were occurring in social structure and values. The chapter is liberally illustrated with photographs of the buildings described. It makes one long to walk through the streets, look at the facades of buildings, and see and understand them with a perspective enormously enriched by Schorske's insightful descriptions of how they came into existence.

Subsequent chapters focus on politics, psychology, art, and music, weaving in new themes and ideas. The roots of anti-Semitism, Zionism, and demagoguery are described in

the careers of three politicians of the era: von Schönerer, Lueger, and Herzl. These themes are interwoven with a theme introduced earlier, the death of traditional liberal values. All these themes lead naturally into the career and personal crisis of Sigmund Freud, passed over for academic promotion because of his Jewishness, struggling with his own father's death and preoccupied with the subject of patricide, shocking his contemporaries with his revolutionary ideas about the importance of sexuality. The themes introduced by Freud's crisis move naturally into the artistic revolution created by Gustav Klimt, leader of the Secessionist School, which used his *nuda veritas* as its symbol, creator of a frankly provocative sensuousness in both subject and expression. Yet rebellion had its price, as Schorske points out:

> In "Watersnakes" (Plate II), the woman's sensuality both gains a new concreteness and becomes more threatening. . . . Klimt's women-as-snakes overwhelm the male not so much with the temptation of the Garden as with a sense of his inadequacy in the face of their seemingly inexhaustible capacity for carnal bliss. In his exploration of the erotic, Klimt banished the moral sense of sin that had plagued the righteous fathers. But in its place arose a fear of sex that haunted many of the sensitive sons.

The final chapters explore further the theme of the garden, a traditional symbol of both order and destruction of order through rebellion. These chapters return again to literature through the figures of Stifter and Hofmannsthal and to art through the figure of Kokoschka.

The book begins by viewing Ravel's *La Valse* as symbolic of the dissolution of traditional Vienna, for in it the ordered waltz movement disintegrates before one's ears. It concludes with the music of Schoenberg, who explored the themes of *Liebestod* and *Angst* in his content and who exploded traditional concepts of harmony in the dissonance of the 12-tone scale.

This book is a fine demonstration that synthesis can be achieved indeed in the Age of Analysis. It is beautifully written and illustrated. Its breadth is exciting. Its appeal is broad. Both Freudians and patricides will find something in it to enjoy and to admire.

N.C.A.

Brief Mention

Medicine and Literature, *edited by Enid Rhodes Peschel. New York, N.Y., Neale Watson Academic Publications, 1980, 204 pp., $15.00.*

Medicine is sometimes called a healing art. Much to the dismay of humanistically oriented physicians, in recent years the artful and intuitive aspects of clinical practice have been minimized and the scientific aspects maximized. This book attempts to reunite medicine and the humanities by addressing the subject of literature and medicine.

It contains three types of essays. The first set deals with physician-writers such as Chekhov, Rabelais, William Carlos Williams, and Richard Selzer. The second set of essays examines doctors as portrayed in literature, looking at works by Flaubert, Camus, and Shakespeare. The third set of essays explores the experience of illness as portrayed in literature. Some of these essays are interesting, and some are rather dull. Nearly all are written by literary scholars who appear to have had little direct experience with the practice of medicine, a fact that sometimes gives the book a rather mandarin and esoteric quality.

Biological Aspects of Mental Disorder, *by Solomon H. Snyder, M.D. New York, N.Y., Oxford University Press, 1980, 253 pp., $13.95.*

The title of this book, written by one of psychiatry's most distinguished biological researchers, suggests that it will provide a summary of recent explorations in the biology of psychiatric disorders. In fact, however, the title is misleading. The book provides a relatively simple summary of the entire field of psychiatry, including diagnosis and classification, psychological and biochemical theories concerning etiology, and a variety of treatments including both psychotherapy and drug treatments. Neurochemistry and neuropharmacology, which one would expect to be major strengths in this book, are discussed only briefly, and neurophysiology is scarcely touched on. All types of mental illness are discussed, ranging from the major psychoses to the broad range of sexual disorders. Only the traditional neuroses and personality disorders are left out.

The book is directed at the level of the general reader or medical student. Its style is fresh and clear. Although Snyder is obviously trying hard to be comprehensive and to balance the biological with the psychological, one wishes that he had dealt more fully with his own area of strength and had not been so eclectic and overinclusive.

Experimental Design in Psychiatry: Research Methods for Clinical Practice, *by Walter W. Surwillo, Ph.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1980, 170 pp., $19.50.*

Psychiatrists who value rigorous and objective thinking sometimes find themselves handicapped or embarrassed by their lack of statistical skill. This book was written to remedy such handicaps. It is ostensibly addressed to psychiatrists at the residency level, and it would be quite suitable as a text for trainees who need to learn elementary to intermediate level statistics quickly. It would also be useful reading for psychiatrists who have completed their training and who are dismayed by their statistical illiteracy.

The book is clearly written and includes many illustrative examples that clarify the practical uses and potential abuses of particular statistical techniques. More than half the book is devoted to a discussion of such nonparametric techniques as chi-square, Fisher's exact test, the Mann-Whitney U Test, Friedman two-way analysis of variance (ANOVA), Kruskal-Wallis one-way ANOVA, and the Spearman rank coefficient of correlation. Parametric techniques such as the t test or the Pearson product-moment correlation or linear and multiple regression are also discussed more briefly. The author includes enough mathematics to make these techniques clear, but not so much as to overwhelm or intimidate

the reader. Discussions of experimental design, hypothesis testing, and the appropriateness of particular statistical techniques to various designs or methods of measurement are also included.

N.C.A.

The Uses of Psychiatry in the Law: A Clinical View of Forensic Psychiatry, *by Walter Bromberg. Westport, Conn., Quorum Books, 1979, 430 pp., $25.00.*

Walter Bromberg, M.D., a distinguished elder statesman in his field, has seen it all. One of the most moving passages in his new book, *The Uses of Psychiatry in the Law,* describes the conditions at Manhattan State Hospital on Ward's Island, New York, when Dr. Bromberg first started working there in 1928:

> The buildings that housed the "back wards," including the Inebriate Asylum built in 1854, appeared more antiquated as one moved away from the Administration Building. Here patients of all varieties—cases of dementia praecox, melancholias, manics—wiled away their years. The more disturbed were ordered to sit in straightback chairs, while the more compliant pushed a heavy wooden block across the hall for the obvious purpose of polishing an overpolished floor. There was no communication between patients and little with the nursing staff; some argued with imaginary foes outside the windows; some sat in Rodinesque immobility; some giggled foolishly. During the nonwork hours, the halls looked like an old Hogarth print. Occasionally, a crackling laugh issued from an upstairs window, or a grunt from a restrained manic broke the empty hum.
>
> In the morning, long lines of ragtag patients filed from the halls under attendant-guards, en route to menial jobs in the laundry or on the grounds. The men dressed in ill-fitting drab clothes, a cloth cap set at an idiotic angle, and talked and gesticulated to themselves. The women patients, in clumsy shoes and dun-colored dresses, giggled or mumbled to the morning air. Occasionally, one would depart from the file to emphasize a point to an imaginary persecutor or to lunge at a vexing enemy. The scene was dreary, hopeless, eternal.

Just as he has witnessed the enormous changes in the care of the mentally ill that have taken place since 1928, Dr. Bromberg has also witnessed, at first hand, almost the entire recent development of psychiatry and the law. The great strengths of his book are its comprehensiveness and his ability to illustrate his descriptions with so many examples from his long clinical experience, which includes being one of the medical experts to examine Jack Ruby (we are treated to a lengthy account of the latter's mental state). *The Uses of Psychiatry in the Law* is not so much a theoretical work as an overview of almost every topic conceivable. It discusses, quite well, the shifting development of the insanity defense, the concept of diminished responsibility, competency to stand trial, the psychopathic personality and the law (emphasizing the treatment difficulties that practically everyone experiences), and the relationship of drug addiction and violent crime. Perhaps because I have had some challenging cases in this area in my own private practice, I was especially intrigued by Dr. Bromberg's chapter on sexual crimes and sexual psychopathy. Often the general public is so revolted by such crimes and judges are under such public and political pressure to put those unfortunates who commit them "away" for long or indeterminate periods that it is no easy task for an examining psychiatrist to put into perspective—for judge and jury—the dynamics behind the offense.

The topics noted so far represent only part of the book's range. Dr. Bromberg looks at the role of mental illness in divorce and annulment. He touches on custody, including the famous *Painter v. Bannister* case in which prejudice against the father's "Bohemian" lifestyle played more of a role in the decision making than any psychological unfitness to be a good father on his part. Competency to enter into contracts, to make wills, and to testify as a witness is discussed (with an interesting analysis of Whittaker Chambers' testimony against Alger Hiss, especially the failure of the psychiatric expert witness for the defense to be adequately prepared). The increasingly strict safeguards that are required before an individual can be civilly committed are examined. Dr. Bromberg is candid about psychiatry's inability to predict "dangerousness" to self or others and how lawyers and the legal system hound psychiatrists into trying to make such predictions. The book considers psychiatrists' roles in testifying in personal injury and workman's compensation cases. It concludes with a discussion of the psychiatrist when he or she is involved in a lawsuit—for malpractice, failing to predict a suicide, or violating confidentiality. We learn that, after the California Supreme Court's ruling in the *Tarasoff* case, there is a duty to violate confidentiality to inform a potential victim that he or she may be at risk for harm by a particular patient. Indeed, one of the nice things about *The Uses of Psychiatry in the Law* is the descriptive table of important cases at the end of the volume.

The audience for this book is the general reader, lawyers, and psychiatrists. It is a survey, with a little bit on every topic, and would be a good addition to a medical library. Very few topics, however, are covered in much analytic depth—the book's size simply does not allow for it. But each chapter is a good starting point to get an overview of a particular area, and the footnotes and bibliography point the way to more detailed references.

Rather than having each chapter written by a different expert in the given field, as is *The Harvard Guide to Modern Psychiatry* (1), Dr. Bromberg has attempted the whole task of this book himself, drawing from his many years of practice as a forensic psychiatrist. One might expect that a synthesized, thematic view of the interaction of law and psychiatry might emerge, as it does in Dr. Alan Stone's *Mental Health and Law: A System in Transition* (2), but it does not. Dr. Bromberg chooses, instead, to bring us up to date on the current course of development over a wide range of topics in these two fields. He leaves the working out of the future path of development to us.

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The Literary Freud: Mechanisms of Defense and the Poetic Will. Psychiatry and the Humanities, Vol. 4, *edited by Joseph H. Smith, M.D. New Haven, Conn., Yale University Press, 1980, 379 pp., $27.50.*

It is seldom noted and perhaps not widely known that Freud was awarded the Goethe Prize in 1930. This annual award was founded in 1927 by the City of Frankfurt for annual presentation to "a personality of established achievement whose creative work is worthy of an honour dedicated to Goethe's memory." It is also of interest that the first recipient of this honor was Stefan George (1868–1933), the German poet who led the revolt against realism in German literature through his aristocratic and esoteric lyric verse.

Freud was not well enough to travel to accept the prize ($2,500) and deliver the required address in person, so his daughter, Anna, read his "few sentences." In his brief remarks (1) he defended analysts against the reproach that "they forfeited the right to place themselves under the patronage of Goethe because we have offended against the respect due to him by trying to apply analysis to him himself. I would dispute at once any degradation is intended or implied by this" (1, p. 211).

In the volume of 12 evocative essays under review, there is no parallel apology for the attempt on the part of literary criticism to illuminate or clarify the Freudian text, presumably not open to clarification or amplification by psychoanalytic clinical methodology per se. What therefore emerges is a fascinating literary exegesis of some Freudian metapsychological texts, "in the absence of clinical evidence."

This collection of essays is in line with a whole new concept of applied psychoanalysis, in which Freudian theory is more likely to be employed by nonanalysts in elucidation of their own discipline. These applications in effect basically modify Freudian concepts themselves. These highly provocative essays are introduced by the title paper of the volume, "Freud's Concept of Defense and the Poetic Will" by the eminent literary critic Harold Bloom. His discussion opens with the immediate equation of defense and troping, or the use of figures of speech. (This sets the style of the paper, which is saturated with figures of speech and undoubtedly will send many readers to their dictionaries.) Bloom leads us through a discussion of Shelley's defense of poetry against Thomas Love Peacock's characterization of it as "rubbish of departed ignorance" to an elaborate chapter-by-chapter analysis of Freud's "Beyond the Pleasure Principle" (2), which he seeks to interpret as "a dialectical lyric—indeed, as a post-Romantic crisis lyric" (p. 11). It may be gratuitous to remind the reader that in "Beyond the Pleasure Principle" Freud introduced his most controversial and least accepted metapsychological concept, that of a death instinct, "the urge inherent in organic life to restore an earlier state of things" (2, p. 36).

For example, chapter one of "Beyond the Pleasure Principle" is an allegory or irony in that it says the pleasure principle has priority over the constancy principle only to relieve the analyst of allegiance to nineteenth-century biology and physics. Bloom terms chapter two a "tessera" (a token or password), "an antithetical completion that fails to complete." In this chapter Freud "tacitly and perhaps unknowingly . . . lets us understand that his own earlier synechdoches were incomplete" (p. 14). The synechdoche Bloom refers to at this point is what he referred to earlier as "Freud's highly characteristic synechdoche of neurosis as mutilated part and psychic health as macrocosmic unity" (p. 14). Chapters one and two are discontinuous; the latter is disjunctive in itself because it considers traumatic neurosis and children's play, which are antithetical. Chapter three is labeled Freud's "kenosis" or "emptying himself," as Christ did on becoming a man. Bloom suggests that "we can observe Freud defending his psychoanalytic strength by isolating too rigorously a crucial element in his praxis: the transference."

Chapter four is an "indeliberate exercise in the grotesque, with much of its rhetoric a curious litotes [understatement], and with its argument colored by images of depth"(p. 16). This chapter also reveals Freud's defense against biologism, "a repressed movement from scientism to speculation" (p. 16).

Chapter five is an "askess" or sublimation of the problematic theory of drives with Freud's new metaphor that drives are but "circuitous paths to death." Chapter six is "that final movement I have named by the ratio of apophades, which in rhetoric is the modes of transumption and in poetry is manifested through images of earliness and lateness" (p. 18). Chapter seven is an extension that reveals "the hidden Freudian metalepsis," for example, the literal meaning being death while the figurative one is Eros. Bloom at this point terms the death instinct formulation "outrageously speculative" although of unsettling originality, and he concludes that the drives and defenses are modeled on poetic rhetoric whether or not one believes with Lacan that the unconscious is structured like a language. At one point in this literary and highly "speculative" exposition, Bloom lets the cat out of the bag, to use a trope. He writes, "Granting that I merely seem to be playing with figurations, permit me to extend the play for a space" (p. 21). These examples from the paper have been multiplied to give an adequate sense of its flavor, so to speak. Indeed, one cannot deny the intricacy, the wealth of allusions, the literary cleverness of the essay, but one can muse or be bemused over its clinical or scientific relevance. Of course, this in no way reflects on its merit as literary critique of a famous psychoanalytic text as literature.

The 11 other eminent contributors from major universities and psychiatric centers of learning range over a wide area of related topics from Blake and Freud to Freud and Hart to "The Articulation of the Ego in the English Renaissance." Many reveal the current great interest in the revisionist French psychoanalyst Jacques Lacan. S.H. Brisman and L. Brisman, in their essay "Lies Against Solitude: Symbolic, Imaginary and Real," advance a revisionary idea of their own, namely, that Lacan's notion of the unconscious is structured like a language be restated that the unconscious is structured by language.

Marshall Edelston, in "Two Questions About Psychoanalysis and Poetry," suggests that analysts should read poetry because the same mind that produces dreams, parapraxes, and symptoms also writes poetry. Thus psychoanalysis must propound a theory of mind that also explains its poetic proclivities.

There is no end to the interesting points these 12 essays offer the interested reader, replete as they are with literary, psychological, and psychoanalytic allusions and insights. I can only strongly recommend it as a literary and intellectual feast indeed. To do justice to all of the essays would require a lengthy review of each.

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Individual and Family Therapy: Toward an Integration, *by Fred M. Sander, M.D. New York, N.Y., Jason Aronson, 1979, 235 pp., $20.00.*

In *The Structure of Scientific Revolutions* Kuhn (1) remarked that a new method of observation can initiate a paradigm shift without a crisis having developed in "normal science." Naturalistic observation of patients in their families is a very different vantage point from that of the psychoanalytic situation. The new data it produced led to new concepts, new theories, and new approaches to therapy. Early observers of families adopted contemporary models from neighboring hard sciences, as Freud had done in his era. The models had changed to those of ecology, information exchange, and contextual interactions: the systems paradigm. Family therapists are developing new concepts to convey the newly described phenomena they are working with.

Fred Sander's book is an excellent introduction to the process of integrating the family perspective into general psychiatry for those therapists trained in traditional psychoanalytically oriented psychotherapy. Dr. Sander's talents as a teacher and scholar illuminate the essential assumptions of psychoanalysis and family therapy, which he illustrates with discussions of several dramatic plays. He uses excerpts from *Hamlet* to characterize the psychoanalytic approach and to begin reflection on the paradigm shift involved in thinking about life from a family systems perspective:

> There is general agreement that Hamlet's internal oedipal conflict was complicated by a family situation that *in reality* directly mirrored his unconscious fantasies. Although, as we mentioned earlier, his conflicts were fairly well internalized, he was also embroiled in a rather severe ongoing pathological family system marked by denial, externalization, projection, and acting out. (p. 15)

Dr. Sander remarks that psychoanalysis has "chosen not to deal directly with such external realities except to acknowledge that psychoanalytic treatment is usually not indicated at such times" (p. 16).

In a fascinating chapter organized around Eliot's *The Cocktail Party* (in which he uses action from the play to illustrate various family therapy ideas) Dr. Sander traces the emergence of the major trends in family therapy during the 1950s and 1960s. Albee's *Who's Afraid of Virginia Woolf* and Eliot's *The Family Reunion* serve to bind together some ideas about "Childlessness and the Family Life Cycle (chapter three) and "Schizophrenia and the Family" (chapter four).

Dr. Sander is a classically trained psychoanalyst who also has long experience in family therapy, and he is eminently qualified to survey both areas. He spends 70 pages in a scholarly exegesis of Freud's position on marriage and the family and of recent psychoanalytic views, noting the lack of any systematic study of the problems of marriage or the interplay of the marital relationship with child development in the vast psychoanalytic literature. He then gives a too brief description of the scope of the eclectic family therapy literature course he and Chris Beals originally taught at the Albert Einstein College of Medicine. The clinical practices of the leading family therapists are touched on. In a set of clinical examples, Dr. Sander gives his views on the usefulness of both the individual and the family paradigms. His concluding chapter uses both to analyze Wilde's *Salome*.

The underlying premise of the book is

> that the polarization of individual/intrapsychic and family/interpersonal approaches is an artificial one. We need to understand in what ways the individual approach affects individuals and family systems as we need to know how the family systems approaches affect change in families and individuals. (p. 181)

Dr. Sander's style is clear and readable, and his bibliography is an excellent guide to the literature. His book is a valuable introductory text.

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Psychiatric Factors in Drug Abuse, *edited by Roy W. Pickens, Ph.D., and Leonard L. Heston, M.D. New York, N.Y., Grune & Stratton, 1979, 372 pp., $19.50.*

One impressive feature of this book is the rapidity of its publication. It represents the proceedings of a conference held at the University of Minnesota in March 1979. Grune & Stratton, through a process called Rapid Manuscript Reproduction, have brought us a book more up-to-date than most.

There was a pleasant irony in the fact that I was asked to review this book. The conference, like many of the best continuing medical education (CME) courses at the annual meeting of the American Psychiatric Association, attracted many people and was filled early. My application arrived late and was turned down.

Thirty-five authors have contributed 17 review papers ranging from basic psychopathology to more esoteric subjects, such as alcoholism in anorexia nervosa. Each chapter has a good bibliography, the longest of which is Ellinwood's, with 122 citations on drug-induced psychosis. A computer search of the world literature on this subject alone would have cost more than the entire book, would have been much less selective, and probably would have been less complete.

Although the book has a special interest for researchers and teachers, there is much that is relevant to the practicing clinician. Jaffe and Kanzler's chapter on "Smoking as a Psychiatric Disorder" provides a reasoned perspective to the acrimonious debate that took place when tobacco use disorders were first proposed for inclusion in *DSM-III*. They begin with Sir Humphrey Ralston's 1926 statement that "to regard tobacco as a drug of addiction may be all very well in

a humorous sense, but it is hardly accurate." Their review of the psychological, pharmacological, biological, and treatment data provides a beautiful conceptual overview of the main issues in drug abuse.

The opening chapter by Schuster, Renault, and Blaine on the "Relationship of Psychopathology to Non-Medical Drug Use" is a classic. It should be read by every physician whose medical education created the impression that alcoholism or drug dependence always represents severe psychopathology. The authors present a balanced, well-documented review of both the animal and human literature to point out the continuum of nonmedical drug use that exists from normality through psychosis. They conclude that "drug taking is biologically normal and society must learn to live with that fact and to develop the necessary constraints to prevent unregulated drug use." Their material has provided a new introduction to my substance abuse course for second-year medical students.

Of similar basic medical educational value is Schaefer's article on "Ethnic Differences in Response to Alcohol." His review of the biocultural data effectively illustrates the complexity of the nature-nurture argument. In spite of the fact that "seven of twelve comparisons indicate American Indians have *faster* rates of ethanol disappearance," the "firewater myth" continues to be used as a form of ethnic one-upmanship to prove that Indians cannot hold their liquor.

There is a wealth of other useful information in this book, which illustrates both the depth and respectability of psychiatric interest in drug abuse. There is little direct information on treatment, but the material provided will add knowledge and perspective to the efforts of those of us who treat alcohol and drug abuse.

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Delirium: Acute Brain Failure in Man, *by Z.J. Lipowski, M.D. Springfield, Ill., Charles C Thomas, 1980, 548 pp., $39.75.*

Surprisingly, as the author points out, before this work no book about delirium has been published in English. "Delirium" derives from the Latin word for "furrow," making the literal meaning close to the modern idiom "off the track." The author defines delirium as a "psychiatric syndrome characterized by a transient disorganization of a wide range of cognitive functions due to widespread derangement of cerebral metabolism."

The volume consists of two main sections. The first deals with clinical features and psychopathology of delirium and is followed by a chapter on etiology. This chapter provides the framework for the second part of the volume. A separate chapter addresses pathophysiology and provides an extensive and current review of cerebral blood flow, electroencephalography, and contemporary physiologic concepts of arousal and hypnogenic systems. The remaining chapters in this section deal with the diagnosis and management of delirium. The second part of the volume deals with the various organic causes of delirium and provides an extraordinarily complete review of the wide range of diseases producing delirium.

This is an excellent book that deserves a large audience. Residents and house officers will benefit from its systematic approach to diagnosis and differential diagnosis. Family practitioners, internists, and surgeons will find the second section of the book useful because of its encyclopedic review of the causes of delirium.

Neurologists and organically oriented psychiatrists will find much to learn in the review of pathophysiology and pathogenesis. The bibliography is extensive and current, with citations as recent as 1978. The merits of this book should give it a long half-life and justify the enormous effort that Dr. Lipowski has put into it.

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Exhibitionism: Description, Assessment, and Treatment, *edited by Daniel J. Cox and Reid J. Daitzman. New York, N.Y., Garland STPM Press, 1980, 392 pp., $27.50.*

The title of this volume is somewhat misleading. It does deal solely with exhibitionism, but it would more aptly be titled *The Behavioral Assessment and Treatment of Exhibitionism*. Although there is one chapter each on psychodynamic group therapy, psychoanalytic theory and treatment, and the integration of behavioral and psychodynamic approaches, the remaining 10 chapters are entirely from a behavioral perspective, with two on forensic considerations.

The foregoing is not meant to detract from the book's value. It contains some of the best written, fully described, well-documented, and carefully followed up expositions of behavior therapy for exhibitionists I have seen. With respect to assessment, several of the chapters, especially those by Brownell and by Murphy and associates, are explicit and informative about the various aspects of exhibitionism that must be separately assessed, and how these distinguishable facets variably affect treatment.

Various individual and combined forms of behavior modification therapy, along with available follow-up data, are described in separate chapters. Although there is some inevitable unevenness, in general they meet the essential test of leaving even the nonbehaviorist reader with the confident sense of having understood the procedure and the rationale. An outstanding chapter is Maletsky's lengthy, elegantly written summary of about a decade of clinical research and therapy, including detailed descriptions of techniques, with 186 exhibitionists. Even more impressive is the careful follow-up indicating a failure rate of only 12.4%.

The two chapters on legal considerations are concise, practical, and reasonably comprehensive. The chapter by Rhoads on psychodynamic and behavioral integration is admirably well balanced but, unfortunately, far too simplified. He offers some useful suggestions and illustrations of a combined approach. Mathis' chapter on group therapy is solid and practical. Allen's psychoanalytic chapter is a sound review of psychoanalytic theory and the psychodynamics of exhibitionism, but it is badly obscured by the jargon of psychoanalytic metatheory, which invariably compromises both comprehension and credibility.

There are three matters with which I take issue. First, the chapter on exhibitionists' victims seems carelessly reasoned. Exhibitionism is a common behavior, and the very small percentage of women who report being significantly disturbed by the experience translates into a sizable number. The fact that some women are psychologically vulnerable to

this experience is conceptually very different from regarding the act as intrinsically damaging. This is not to dismiss the offensiveness of exhibitionism but to emphasize that the real victim of this perversion is almost always the exhibitionist.

Second, the point is repeatedly made that exhibitionists seldom escalate to more serious or violent sexual offenses. True, but the fact that many violent sexual criminals report exhibitionism before becoming sexually violent is ignored. These are not statistically inconsistent facts, nor is the matter academic. It is crucial to explore carefully and explicitly for violent sexual fantasies, dreams, or thoughts when one is evaluating an exhibitionist, to help differentiate those few who may need far more careful management and surveillance and whose treatment plans may need to be especially tailored to prevent violence.

Finally, the description of exhibitionists is thin to the point of uselessness in understanding these people when it fails to include a description of what is going on intrapsychically, dynamically, and developmentally.

The data presented are convincing that behavioral therapy is the treatment of choice, by any criterion, for symptomatic relief in the great majority of exhibitionists. Within its scope, this is a good book, to be confidently recommended. One might wish only that its scope were broader. The ultimate relief of a pathological condition is its prevention, which requires exquisite understanding of its etiology and development. Behaviorism cannot suffice in this task regarding exhibitionism as well as it can in devising effective therapy.

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Health and the Family, *edited by Clive Wood. London, England, Academic Press, 1979, 235 pp., $27.00.*

This slim volume reports the proceedings of a conference sponsored jointly by the Royal Society of Medicine and the Royal Society of Medicine Foundation held at the University of Cincinnati Medical Center in 1978. The contributors include eight distinguished scientists and educators from the United Kingdom as well as 19 notable scientists, educators, and administrators from the United States. There is a broad spectrum of presentations dealing comprehensively with the changing role of the family in contemporary life, some of the dramatic advances in medicine in modern times, and the interface of the two phenomena. Is the family disintegrating in a setting of greater social, sexual, and economic freedom for women, along with a mounting divorce rate? Are new or other forms of human relationships superseding the role of the family? Are the health professions providing adequate services to individuals and families? Can the health professions enunciate more clearly the role of intrafamilial stress in social and physiological disequilibrium, and can they intervene in a preventive or therapeutic fashion? What are the strengths and weaknesses of current training programs for physicians?

These are some of the issues to which the contributors address themselves. A well-known sociologist once noted that he was much more interested in raising the right questions than in trying to formulate the correct answers. The present volume provides even-handed responses from both sides of the Atlantic, but in the sense of searching for solutions rather than providing final answers.

Many of the presentations are most relevant to health educators, primary care physicians, and public health specialists. Also, there is important information for health care students in terms of "the shape of things to come."

In view of the continuing neglect of the behavioral sciences in medical school and residency training, psychiatrists may be increasingly called on to round out the trainees' views of individuals and families. From that perspective, they would derive significant benefit from the whole book, which cannot be reviewed in detail here.

I have chosen to focus on a few areas of unusual stress that are common in many families and may be of particular interest to psychiatrists. Christopher F. Clulow of the Institute of Marital Studies, Tavistock Institute of Human Relations, London, and his colleagues have been engaged in a study of the first baby and stress. The changes attendant on the coming of the first child can so disrupt a couple's patterns of living as to be a major life crisis. During pregnancy and following birth, the father may feel neglected and seek solace with another woman, the mother may become depressed, marital satisfactions may diminish, and there may be deterioration of family income and housing accommodations. Either spouse may develop somatic symptoms, and sexual problems often occur. Antenatal and subsequent classes for parents usually focus on the physical and superficial social aspects of the situation. The wife may feel the sole responsibility for the husband's perception that he is being neglected. Actually, little attention is given to the effect of the advent of the baby on the marriage until one spouse or the couple reach the breaking point.

Dr. Clulow and his associates initiated a two-pronged project to study and possibly rectify some of these deficiencies. Small groups of couples were seen by two project workers and a visiting nurse for a period beginning late in the pregnancy and continuing for six sessions, terminating about 6 months after the baby was born. Attention was directed to the couple's talking about their preoccupations, their becoming aware of feelings about the changes required by pregnancy and birth, and discussion of means of coping with these. The rationale for this approach was that by making time available for such reflection, the couples would be better prepared to face anxiety and contain stress.

In addition, project members initiated workshops for visiting nurses to help them perceive and evaluate the effect of the first baby on the marriage relationship and to delineate the role of the visiting nurse. This 18-month project had not been entirely completed at the time of the presentation, but on the surface it seems to have a great deal of merit.

Family-centered birth is presented in considerable detail by Edward J. Quilligan, Professor of Obstetrics and Gynecology, University of Southern California, Los Angeles. Of interest to psychiatrists is the finding that family-centered birth in a hospital setting can improve the relationship between the spouses and increase the process of bonding between mother and infant and father and infant.

Donald G. Langsley, Professor of Psychiatry, University of Cincinnati, reviews the literature on the physician and family crises. Death of a spouse has been rated as the most severe stress, followed by divorce and then by marital separation. According to current trends, 35%–40% of all marriages end in divorce, and this does not include those who have formal or informal separation. Of people seeking psychotherapy, 50% complain of marital difficulties and another 25% report problems related to marriage. Despite these find-

ings and despite the fact that 60% of patients with emotional problems are cared for by primary physicians, the latter often deny that marriage problems are in their area of responsibility.

Langsley reviews several classifications of marriages from a dynamic as well as from a developmental point of view. The axes in these configurations include power, intimacy, and boundaries. He refers to the work of Cuber and Harroff (1), which focused on intimacy. In the conflict-habituated marriage, there is constant tension and the marriage persists only through fear of loneliness. In the devitalized marriage, overt problems are barely evident and numbness and apathy hold sway. In the passive congenial marriage, there is some sharing of interests, but a lack of intense emotional involvement. The vital marriage is described as exciting and rewarding and marked by enthusiasm. Finally, the total marriage has even greater intensity and involvement and each partner sees the other as indispensable.

Langsley goes on to invite the primary physician to use some of the classifications to define the problems in a given troubled marriage, including communication problems and role performance. Then he speaks of developing a therapeutic alliance in brief-treatment marriage therapy. He encourages the primary physician to engage in this kind of treatment rather than referring couples to specialists. This can facilitate continuity of care and promote the diminution of stress and chronic crisis. I am confident that some physicians may be able to function well in this role. However, I wonder whether ongoing workshops as described by Clulow for visiting nurses might be relevant with primary physicians. In addition, psychiatrists to whom couples are referred for treatment might profit by Langsley's suggestions and by greater familiarity with the studies he mentions.

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Object Relations and the Developing Ego in Therapy, *by Althea J. Horner. New York, N.Y., Jason Aronson, 1979, 341 pp., $20.00.*

The increasing psychoanalytic interest in preoedipal psychopathology and the development of object relations theory has led to a burgeoning literature that attempts to relate early childhood development to adult phenomena. In her careful descriptions of the subphases of the separation-individuation process, Margaret Mahler has offered clinicians a way to help their patients put into words their often inchoate anxiety, which interferes with the development of trusting interchanges in the therapeutic process. In this book Dr. Althea Horner attempts to bring together Mahler's ideas, object relations theory, and attachment theory with elements of self-psychology and Piagetian thinking in a formulation of the origins of preoedipal difficulties and their reflections in the intensive psychotherapy of adults. This integrative task is a difficult one; with some reservations, I think she succeeds rather well.

Dr. Horner reviews the early formation of attachment bonds between mother and infant and the process of gradual separation and formation of a cohesive self, suggesting very specific correlates of these stages in the clinical material of her adult patients. Although her certainty about her correlations is somewhat troublesome, she does take clear positions, and her clinical material is rich, varied, and well selected to illustrate the sources of her formulations in the transference observations. She makes complex and clinically useful distinctions between the pathology of schizoid, psychopathic, masochistic, false-self, borderline, narcissistic, and "preneurotic" personality disorders.

There is considerable controversy in the literature about the origin of narcissistic and borderline phenomena in infancy. Using Kohut's formulations of the origins of narcissistic transferences in the childhood phases of the grandiose self and the idealized object, Horner relates these structures to specific subphases: the grandiose self to the elation of the "practicing" period and the idealized object to the panic and dependency of the "rapprochement crisis." Her complex descriptions of the interpersonal, intrapsychic, and affective details of these stages and of the patients' defensive use of the related intrapsychic structures provide useful guidelines for therapeutic intervention.

Horner suggests that both borderline and narcissistic patients experience major trauma during the rapprochement crisis and that borderline patients retreat to their preexisitng self-fragmentation, derived from the symbiotic phase, while narcissistic patients, with more cohesive selves, can use the grandiose self of the practicing phase. She differentiates certain patients' use of grandiosity as a regressive defense against the shame of oedipal failure (in the preneurotic patient) from the more primitive narcissistic patients' use of grandiosity to defend against the anxiety and shame of dependency when there is a less differentiated self and the absence of object constancy.

The book requires work and sophistication on the part of the reader. The rather spare index is not helpful to the reader who might be confused by the welter of concepts. Metapsychological concepts are freely used and tend to be reified. One statement, for instance, reads,

> A significant aspect of being the designated good object is to function as proxy for the rage that the good self must not experience lest it become the bad self and therefore subject to the hate directed at the bad object with whom it is still symbiotically bound and identified.

Despite my own familiarity with these concepts, my mind begins to fade and lose touch with the clinical experience when faced with such abstruse language. Too often, I think, the patient's experience appears to be colored by these theoretical constructs (e.g., one patient reports, "I never had the correct merger experience as a child"), and it is not always clear whether the patient's experience is being correctly understood or whether a new language is being taught. Dr. Horner is, however, quite sensitive to the dangers of the therapist's "knowing too much" and impinging on the patient's privacy and autonomy. Her descriptions of the therapeutic task with these patients and of the patient's need to use the therapist as "mediator of organization" are clear and concise.

Despite the drawbacks of the book, Dr. Horner presents rich and useful ideas for formulating and working dynamically with severely limited patients. Her integrative formulation

of preoedipal pathology is both useful and practical, and I can recommend the book (with the above reservations) for the practicing clinician.

EDWARD R. SHAPIRO, M.D.
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And They Took Themselves Wives: The Emergence of Patriarchy in Western Civilization, *by David Bakan. San Francisco, Calif., Harper & Row, 1979, 179 pp., $10.00.*

In this book David Bakan has produced a succinct, scholarly, and rather timely work. Based on the author's 1976 Terry lectures at Yale University, the book is both an explication of the historical development of Judeo-Christian patriarchy and a defense of the familial values established by that tradition. Leaving room for some disagreement, Dr. Bakan, a professor of psychology and a Talmudic scholar, maintains that the Bible is associated with "what is perhaps the most important step toward the reduction of male-female sex-role differences in the history of civilization." The step alluded to refers to a transition from an ancient Semitic matriarchal society, characterized by the absence of a patrilineal incest taboo and organized geographically by matrilineal clan, to the relatively recent patriarchal structure. Why did this change occur? According to Bakan the ideology of patrilineality was necessary for both the stability of family life, since it established male responsibility for children, and for the survival of the greater society, since it allowed unification of geographically dispersed tribes. Christianity is seen as the natural sociologic outgrowth of patrilineality, since it extends unification still further in allowing brotherhood to exist through faith. The book's scholarship contributes in part to one of its faults. Requiring extensive reference to primary Biblical sources and frequent use of a consonantal text, the book becomes difficult to read. Still there are diversions. Vance Packard is quoted in an analysis of cake symbolism and Kate Millett is in the index along with Ovid, Abraham, and Punch and Judy.

Elaine Pagels' recent book *The Gnostic Gospels* (1) suggests that one of the most striking differences between the orthodox sources used by Bakan and the heretical ones is that the latter often described God in both feminine and masculine but still specifically Christian terms. One wonders what Dr. Bakan would make of this, since the unorthodox texts would support the existence of a viable extended kinship structure organized along more than simply patrilineal lines. This points to an additional concern for the reader. As noted in the previous quotation and as is summarized in the book's conclusion, the author uses Judeo-Christian history to discuss the nature of the male-female role differences, the future of the family, and "the welfare of the total society" of man. It is difficult to rationalize engaging in these broader statements without reference to the contributions of non-Western civilizations. Similarly, a study of family structure in primitive societies creates some doubt as to the logic or universality of the progression that the author outlines. Ernest Crawley, in his classic work *The Mystic Rose* (2), pointed out that even in the most primitive tribes studied, paternal descent is nearly as common as maternal. These are small points, however, and should not distract the reader from the real worth of this book. It is thoughtful, has a sense of history, and should interest a broad audience of readers.

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VICTOR I. REUS, M.D.
San Francisco, Calif.

Psychedelic Drugs Reconsidered, *by Lester Grinspoon and James B. Bakalar. New York, N.Y., Basic Books, 1979, 334 pp., $15.95.*

This book is the third of what appear to be the authors' attempts to provide the nonspecialist with information necessary to informed public policy concerning "drugs of abuse." (Previous volumes dealt with marijuana and cocaine.) After a first semitechnical chapter concerning sources, botanical and otherwise, chemical structure, dose, mode of action, and effects, Grinspoon and Bakalar go on to describe psychedelic use in pre-industrial society and its more recent history from Priestley (nitrous oxide) through Weir Mitchell, William James, Klüver, Hofman, Leary, and finally to legal controls and seeming disinterest. Other chapters present the phenomenology of the response to psychedelic drugs in exquisite detail, the infrequent adverse reactions, and the drugs' questionable therapeutic effects. Final chapters deal with implications of the drug-induced response and a plea for continued or, more accurately, renewed research. An appendix describes the current legal situation regarding psychedelic drug use; the book ends with an excellent, interesting, but not exhaustive bibliography and an index.

Although as a former active participant in some of the controversies concerning their free use in the community I may not bring objectivity, I certainly bring interest and, presumably, knowledge. The facts seem accurate, the semifacts accurately stated. Curiously, even for an interested reader, as William James (and the authors) pointed out, the phenomenology of "phantasms" is "intolerably dull." It is not easy to pay close and critical attention to somebody else's ineffable or ecstatic experience. To my mind, the authors (despite their own caveats) take too literally the reports of birth experiences as possibly reflective of real, specific memory traces rather than personal or cultural retrospective interpretation of something of the commonality of being human. They potentially mistake metaphoric for material connections. The surface similarity of some psychedelically induced mystical experiences and the Heisenberg uncertainty principle does not suggest a possible underlying world order; while analogous, they are unlikely to be homologous.

Our science does, however, need to pay attention to these phenomena. The ineffable needs to be accounted for rather than dismissed. William James said, "No account of the universe in its totality can be final which leaves these other forms of consciousness quite disregarded. . . They forbid a premature closing of our accounts with reality" (1, p. 388). Now that the flower children have faded and the political uproar is past its peak, Grinspoon and Bakalar do us a service by redirecting our attention to important unresolved issues: what do these drugs do, how do they do it, and how may their effects be used as a tool of science, as treatment, and as a mechanism to help us enlarge our view of our humanity?

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WILLIAM A. FROSCH, M.D.
New York, N.Y.

Sexual Excitement: Dynamics of Erotic Life, *by Robert J. Stoller, M.D. New York, N.Y., Pantheon Books, 1979, 271 pp., $11.95.*

In this, his fifth book on sexuality, Dr. Stoller serves the reader a rich and highly seasoned feast for the mind. Like most psychoanalytic writing, the book fits into no neat category but falls somewhere between the technical and the popular, the scientific and the poetic. Unlike much analytic writing, however, it is free of the turgidity, circumlocution, and jargon that afflict so many contributions to that troubled field. Even when he is discursive and tangential, and in this book he sometimes is, Dr. Stoller maintains a vigorous style that carries the reader with him. In fact, the self-confident, direct, even exuberant tone of the book, which gives it its engaging quality, sometimes loosens its argument.

What we have here are two or three books in one: 1) an erudite discourse on sexual arousal and gender identity, reflecting Dr. Stoller's own clear expertise in these matters, 2) an analytic case study of a high-functioning hysterical patient called Belle and of her sadomasochistic fantasy, and 3) an essay on mind, self, consciousness, will, and morality.

This is a book that is chock-full of ideas. Even when the writing wanders far afield, it remains rich in content and stimulating in style. One finds insights, observations, and vignettes strewn like nuggets throughout the text, the footnotes, and the appendixes.

Dr. Stoller's central thesis is that fantasy is an essential component of most people's erotic life and that hostility is a ubiquitous element in most people's erotic fantasy. The hostility is not inborn, nor is it inevitable. It is a device to undo childhood humiliation and to turn defeat into triumph. It is so common because childhood trauma is so common. The essence of therapeutic healing is achievement of such self-understanding and acceptance that one can give up the need for revenge and remove the element of hostility from one's cherished relationships. Rather than a defense of or an apologia for hostility in sexual activity, the book is an elaborate explanation of it and describes at least one method, psychoanalysis, to overcome it. Stoller is *not* a Sade in analytic clothing.

Belle, the subject of the book, is vividly portrayed as a woman of much grace, beauty, and wit. Obviously, Stoller likes her—and so will the reader. The evolution of her personality and her sexual life and the course of her therapy are well documented and flow with forceful logic. Through detailed illustration and description one comes to see her as the paradigm of all neurotic patients, maybe of all people, and as a three-layered creature: an outward shell of rationality that conceals a middle layer of irrational, primitive, and vindictive fantasy, which in turn encloses an inner core of sanity and integrity. The emergence of that inner core into the full light of day is the aim of successful therapy and of successful personal development.

As mentioned above, this is really more than one book. The reader with limited time and endurance is advised to skip part three, Theories of the Mind, and proceed from part two, Data: Belle, to part four, Conclusion. In doing so, he or she would get the full account of Belle's analysis and a great deal more about sexuality and therapy (the discussion of sadomasochism is particularly outstanding and illuminating). What he or she will miss is a lively, even bold discussion of certain profound psychological-philosophic-existential issues. The author does not shrink from tackling head-on such knotty matters as the meaning of "I," the reality of the self, the mind-brain dichotomy, the role of conflict in personality development, the primacy of aggression, and the question of free will. In doing so, he takes the unusual step of inviting commentaries on these matters from two people who have given much thought to them: M.F. Basch and K.M. Colby. Their contributions appear as Appendix A—a gem of lucid discourse on extremely complex subjects. In fact, part three of the text and this appendix, along with references and annotations pertaining to them, can be viewed as a separate and worthy document in themselves.

Some of the style and content of the book can best be conveyed through quotations:

> Psychoanalysis is the subjective study of subjectivity struggling to become the objective study of subjectivity. (p. 193)

> Analysis, with astonishing speed, went from revolution to respectability to outdated mythology. I do not think that a free society can easily bear the loss. (p. 223)

> Considering how appalling existence is for most humans, we in our society are lucky to be able to wring our hands over flawed sexual pleasure. (p. 4)

> My theory makes sexual excitement just one more example of what others have said for millennia: that humans are not a very loving species—especially when they make love. Too bad. (p. 35)

> Although it is old-fashioned to say that they exist, it is no secret that there are women with richly textured and complex femininity who meet anyone's criteria for psychic health. Their femininity is a product of a solid core of gender identity, permanent and non-conflict-laden identifications with feminine women, and successful grappling with and mastery of oedipal conflicts in which they participated with their mothers and fathers. We need not doubt it; they unthinkingly, comfortably accept having female bodies that are sources of physical pleasure. Should they marry and have children, they draw from their femininity a capacity for mothering in which there is so little hostility that, with the help of their masculine husbands, they raise their sons to be masculine and their daughters feminine. (pp. 55–56)

> Let us call her Belle, for that suggests how she felt she was when analysis began: old-fashioned femininity; a touch of exhibitionism; gentle masochism; a slightly addled yet refreshing innocence; soft, round, dreamy erotism; an unbounded focus on males, romance, silken garments, flowers and beads, bosoms, bare behinds and babies. (p. 59)

> More and more I think success in analysis depends on the patient's need to be honest. (p. 65)

> Sadomasochism is, I think, a central feature of most sexual excitement. My hunch is that the desire to hurt

others in retaliation for having been hurt is essential for most people's sexual excitement all the time but not for all people's excitement all the time. (p. 113)

Perhaps the perversion of masochism is a hungry search for skin and mucuous-membrane stimulation in people who got too little in infancy and who have decided, as have "moral masochists," that any attention, even if painful, is better than none. . . . Bak makes the suggestion that sadomasochistic fantasies, especially those of being immobilized, represent the wish not to be separated from mother. . . . I find the involved and unconscious dynamics of sadomasochism more clever, more intelligent, and more sly than is suggested by the sacrificial ego model: we inflict punishment on ourselves in order to avoid having to change. . . . Guilt is not the price paid for being bad but the price paid for the privilege of continuing to be bad. The megalomania of guilt: I fool everyone. . . . I do not think people become masochistic because they truly believe they deserve to be punished. Rather, I think they trick themselves and us into believing that that is what they do, while secretly, they are busy with their foxy little sadisms. (pp. 118–124)

With what, would Colby ask, does consciousness provide us that could not be accomplished without consciousness? My answer is the same, I think, as Freud's. The value of consciousness is pleasure and pain, an effective motivating system: the present models for behavior, such as computers and their programs, will be closer approximations when they can itch as well as scratch. (p. 181)

We still go on living convinced at bottom that we make our own choices. No one, not even a madman, knows he is just a machine. No scientist really believes in the determinism he reveals in his laboratory. . . . In these gentle and liberal times, it is the style in some circles to call off the moralists and to see us all as victims free from the accusation of sin because our behavior is determined: choice is an illusion. But take the battering father. We know it was predetermined he would batter his child, in part, because he too was battered when little. Yet even at the hottest point of his rage, when he lifts his arm to bash in his baby's skull, there is a moment when, were he not indulging himself, he would hold back his hand. Most parents take advantage of their children because the poor things are little. . . . In other words, though it may nowadays be in bad taste, it may nonetheless be scientifically accurate to go back to—even assign responsibility to—parents for what they do to their children. (pp. 200–201)

When one considers how often the subject of sexuality is given such dull treatment in the literature, it is both fortunate and appropriate to find an exciting book on sexual excitement. That it is two books in one need not detract from its value to the reader who is duly warned; in fact, he or she can look upon it as a bonus and a bargain in these inflated times. Good printing, an eye-catching dust jacket, references, generous annotations, and an index round out a happy publishing event.

JOHN RACY, M.D.
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Electroshock: Its Brain-Disabling Effects, *by Peter Roger Breggin. New York, Springer Publishing Co., 1979, 237 pp., $17.95.*

In this book Dr. Breggin sets out to prove that ECT is not therapeutically effective and that, instead, it produces severe brain damage and irreversible mental dysfunction in most, if not all, of those who have received it. He bases his "proof" on what the dust jacket correctly describes as an "original" analysis of the ECT literature along with some anecdotal reports of dissatisfied patients. After admitting that his conclusions are not shared by most others in the psychiatric community (1), Dr. Breggin attempts to explain this discrepancy in terms of some sort of vague conspiracy on the part of "ECT advocates," who he insinuates have misled both patients and colleagues for some presumably nefarious purpose that is left unclear to the reader. Unfortunately, a true resolution of these differences is beyond the scope of Dr. Breggin's book, since, both quantitatively and qualitatively, factual inaccuracies and notable absences ensure an exceedingly biased presentation.

In actuality, the issue of persistent CNS side effects with ECT, currently under active investigation in this country and abroad, is certainly not as simplistic as Dr. Breggin would have it. Although there is some evidence that persistent subtle deficits, chiefly in the area of personal memory, may in fact occasionally occur in individuals following ECT, the vast bulk of both clinical and experimental evidence suggests that the picture of profound, irreversible impairment presented by Dr. Breggin is clearly not a common occurrence. For a more accurate view of what ECT does and does not do, the interested reader is referred to the report on ECT by the American Psychiatric Association (1), the proceedings of an NIMH-sponsored multidisciplinary conference (2), and the encyclopedic text by Fink (3).

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RICHARD D. WEINER, M.D., PH.D.
Durham, N.C.

Understanding and Helping the Schizophrenic: A Guide for Family and Friends, *by Silvano Arieti, M.D. New York, N.Y., Basic Books, 1979, 228 pp., $10.95.*

This clearly written and sensible short book from the pen of an eminent writer, clinician, and teacher is admirably suited to its purpose. The families and friends of schizophrenic patients will find here the facts, perspective, and tempered optimism they need while striving for balance in the face of psychosis. Some patients will profit from reading it also.

Too often treatment plans for schizophrenic illnesses minimize the importance of family support and education. Dr. Arieti has produced a therapeutically useful tool in this book, readily understandable by any intelligent person with

a high school education. The clinician may recommend it to patients' families and friends with confidence. It will not take the place of case work or family therapy, but it will provide useful orientation.

The major clinical types, patterns of breakdown, and schizophrenic logic and language are all described in such a way as to invite empathy with the patient's experience while acknowledging the helpless distress of those close to him or her. There are vivid clinical examples throughout the book that throw a kindly and instructive light. The interplay of genetic, developmental, family, and social factors in producing a schizophrenic breakdown is presented fairly and lucidly. Dr. Arieti is sympathetic to the problems of the "nuclear family" in an urban society, writing nothing here that will needlessly burden already overburdened mothers and fathers. He nevertheless acknowledges the importance of inheritance and parenting.

One chapter on treatment emphasizes the combined use of psychotherapy and psychoactive drugs. Dr. Arieti explains what happens in the former and discusses the uses and limits of the latter. There is another chapter on hospital care in which the importance of a fully supportive milieu is emphasized. The indications for and against hospitalization are reviewed.

One might wish for a fuller discussion of ECT than Dr. Arieti gives us. Although it is not usually employed, it is sometimes lifesaving. Popular hostility to this measure would make a more explicit mention of its indications and safety desirable. I was also troubled by a statement on page 190: "I support the abolition of psychosurgery." Is there any other effective treatment for the rare but wretched individual completely imprisoned in a compulsive-obsessional system, unresponsive to psychotherapy, drugs, shock, or anything else?

The importance of early case finding and intervention is appropriately emphasized throughout, and supportive counseling for prospective parents is recommended when schizophrenia is present in a grandparent. There is an interesting chapter on "special situations" in which crises are discussed. Because it is all too true that "many psychiatrists are very reluctant to make home visits" the discussion mainly addresses the use of hospital emergency rooms and police. Dr. Arieti implies the importance of the therapist's immediate availability to the family when suicide or assault seems imminent, but he does not spell it out. This chapter also includes a discussion of the religious cult problem.

No book of such scope reduced to this length could satisfy every clinician, but this book is for families and friends. It should be useful to them and thereby helpful to schizophrenic patients and their psychiatrists.

JOHN T. MALTSBERGER, M.D.
Belmont, Mass.

The Psychodynamic Approach to Drug Therapy, *edited by Mortimer Ostow, M.D., Med.Sc.D. Riverdale, N.Y., Psychoanalytic Research & Development Fund, 1979, 344 pp., $18.50.*

In the introduction of this book the authors state, "Psychoanalysis can no longer afford to ignore organic treatment modalities, especially drug therapy." I agree. The contributors to this interesting volume have succeeded in describing how drug therapy can be effectively combined with in-depth psychoanalytic approaches in dealing with many difficult clinical problems. In the first part of the book an overview of clinical pharmacology, psychoanalytic psychiatry, and problems with the combination of the two are condensed and reviewed on the basis of the five cases that are presented in the remainder of the book.

The editor of the book, the individual participants, and a staff assistant have managed to condense into about 300 pages a monumental amount of case material and discussion without diluting or compromising their aims. Eight to twelve experts joined together eight or nine times per year for four years to review process case material of patients in psychoanalysis or psychoanalytic psychotherapy who also received psychoactive drugs. The case material is reported with day-to-day summaries and excerpts, and the group discussion of the participants is interspersed with the case material. In reading the material, I obtained a sufficient sense of the dynamics, the drug effects, and their interaction with each other to allow me to make observations of my own about the cases and to speculate on what was happening and how I might have intervened—psychotherapeutically and/or psychopharmacologically. The cases and discussion also allowed me to make valuable comparisons with my own clinical experiences.

This book suffers at least one serious shortcoming or disadvantage. The majority of the selected case material was taken from the early 1960s, when there was a limited experience with and selection of psychopharmacologically active drugs. Thus it is probably significant that it is not until the fifth and last treated case (spanning 1969–1971) that a more precise identification of target symptoms and corresponding selection of drug therapy is evident. The enabling power of a sophisticated, multimodality approach is most obvious in this case, where effective drug therapy combined with skillful ward management and sensitive psychoanalytic psychotherapy produced a remarkable and salutary treatment outcome.

All of the reported cases are about very sick patients. My reactions alternated between profound respect for the clinician's heroic efforts in treating such patients and uneasiness and impatience with what at times seemed more like trial and error approaches that produced more complications and side effects for the patient than benefits. In either event, this is a compelling book because the reader is afforded a very graphic opportunity to follow, over time, evocative and compelling case material. The reader also has a chance to witness the thoughtful (but at times, highly speculative) exchanges and discussions of a group of knowledgeable psychoanalyst/clinicians who are experienced in both psychoanalysis and psychopharmacology.

There is a tendency in this book to emphasize and focus on the unconscious meaning of the effects of drugs, motivational states, and the influences of psychoactive drugs on instinctual drives and to pay relatively little attention to the influences of ego and self structures and/or impairments to account for psychopathology and the effects of drug therapy. It is not surprising, then, that some of the participants in one of the group discussions provocatively (and I believe erroneously) conclude that one of the unsalutary consequences of symptom relief with drug therapy is that the patient often abandons treatment because he or she feels better. They offer that perhaps this is desirable from the public health/case reduction point of view but undesirable if one wishes to work

longer psychotherapeutically with such patients. I believe it is on a similar basis that they also conclude that drug therapy has little to offer in the management of personality disorders. I disagree that drugs reduce motivation for psychotherapy, and I also disagree that drug therapy has no place in the treatment of personality disorders.

When it is argued that drug treatment might cause premature termination of treatment, it could be argued equally strongly that our sicker patients' fears and distress about human contact and closeness drive them away from psychotherapy and that the permissive effect of psychotropic drugs often helps to keep affects optimal and in turn makes psychotherapeutic work more possible. Similarly, many of the observed behavioral problems associated with personality disorders are related to warded-off, painful affect states. As therapeutic interventions begin to contain behavioral expressions of personality disorders, it is not uncommon to observe the surfacing of depression, anxiety, and panic states, which are treatable with drug therapy, which may then further facilitate psychotherapy.

Although I disagree and find myself at variance with some of the emphases and conclusions of this book, I believe it represents a very important contribution and approach. The contributors to this volume have effectively drawn attention to and instructively described how drug therapy can be combined with psychoanalytic approaches. I would recommend and I believe others would welcome similar future efforts by the contributors to this book and others, with the expectation that even more meaningful insights and practical clinical help would be forthcoming given recent advances in psychoanalytic theory and technique and a more sophisticated appreciation of the action, use, and side effects of the many psychoactive agents we currently use in clinical practice.

E. J. KHANTZIAN, M.D.
Cambridge, Mass.

Temperament Styles in Adult Interaction: Applications in Psychotherapy, *by Jane Burks, Ph.D., and Melvin Rubenstein, M.D. New York, N.Y., Brunner/Mazel, 1979, 205 pp., $15.00.*

Burks and Rubenstein present an approach to psychotherapy that is based on identification by the patient of his or her temperamental style and of its effects on his or her relationships. Their thesis, arising from the 20-year longitudinal study of temperament in children by Thomas, Chess, and Birch, is straightforward: temperament, a "biopsychological sub-system," is an inborn dimension of "reactivity and activity"; although influenced by family and social forces, it persists essentially unchanged throughout life and is an important determinant of behavior.

In their work with adult patients, the authors have identified six temperamental styles based on such characteristics as level of activity, mood, responsiveness, and adaptability. Thus the withdrawer is cautious, thorough, and realistic; the persister is intolerant of interruption and proud of his perfectionism; the intenser is forceful in his feelings and the way he expresses them; the approacher is quick to respond and adapt to new situations; the adapter is a pleasing person and easy to get along with; and the doer is highly active and preoccupied with achievement and success.

In therapy, the patient is made aware of his temperament through a process of self-identification (this is achieved in part by his completion of a simple questionnaire) and of its manifestations in various forms of relationship such as with parents, spouse, and workmates. The patient then comes to accept that his temperament is inherent and learns how to use it in his relationships, particularly by making it known to others. According to the authors' experience these goals are better achieved in group therapy than in individual therapy. The therapeutic approach also has application in family and marital therapy as well as in nonclinical settings.

This form of treatment, and the theory underlying it, are certainly replete with good common sense, but many therapists will no doubt find it overly schematic and some will even find it facile. The authors seem to concede this by emphasizing that temperament is only one aspect of personality and then not even the most important; yet their approach in practice appears to deny these additional aspects.

The book is irritatingly cluttered with jargon: manipulative intentionality, individuate, oppositionality, and occupational system are but a few examples. Although it is less than 200 pages long I also found it rather repetitive; for example, we read at several points about the immutability of temperament and about therapeutic goals.

SIDNEY BLOCH, PH.D.
Oxford, England

The Family: Evaluation and Treatment, *edited by Charles K. Hofling, M.D., and Jerry M. Lewis, M.D. New York, N.Y., Brunner/Mazel, 1980, 313 pp., $17.50.*

This book contains the presentations given at the 1979 meeting of the American College of Psychiatrists; as such it is a mixed bag. In it are a number of absolutely first-rate papers containing material not available elsewhere and reflecting original thinking of a high caliber. Jerry M. Lewis' chapter, titled "The Family Matrix in Health and Disease," is a fine review of the systems approach to families and of his and his colleagues' own work. The chapter by David Reiss, "Pathways to Assessing the Family: Some Choice Points and a Sample Route," is outstanding both in its review of the current state of the diagnostic assessment of families and its discussion of the work of his group. For those who are not familiar with the sociobiological perspective on the family, the chapter by David P. Barash, "Evolutionary Aspects of the Family," is a good introduction.

Also in the book are a number of useful and sensible contributions that do not add to the field but review aspects of it. Theodore Lidz's summary of his thinking is one such, as is the sensible discussion of "family interviewing" as opposed to "family therapy" contributed by Saul L. Brown. Brown emphasizes that family interviews can contribute to a treatment program even if they are not the central therapeutic modality and how this can be accomplished. Similarly, the discussion of "The Family as a System" by James Grier Miller and Jessie L. Miller is a competent introduction to this approach to families.

Finally, there are in this volume a number of slighter contributions that may have been interesting as an introduction to family therapy and how to do it for the audience at the meeting. For instance, Marshall D. Schechter and Harold I. Lief discuss a case involving both family and marital treat-

ment that shows a high level of clinical competence. This chapter, "Indications and Contraindications for Family and Marital Therapy: An Illustrative Case," and several others like it do not add to our knowledge and will probably be of interest only to those who have read little or nothing in the field of the family.

The paper by Yrjö O. Alanen titled "In Search of the Interactional Origin of Schizophrenia" deserves separate comment. Alanen is a major contributor to our understanding of schizophrenic patients and their families. This chapter does not do his work justice, probably because he did not have enough time and space and because the issues in this field, where family influences and genetic endowment interact, are so complicated that they are not easily summarized. Alanen's chapter stands alone, somewhat out of place in the book because the current state of this area deserves both a comprehensive review and a clear exposition of Alanen's own work and views. This chapter accomplishes neither task while attempting both.

It is unclear for whom this book, and many like it, are intended. It is appropriate for neither the beginning student of the family nor for the advanced practitioner. Perhaps it belongs in libraries. The chapters by Lewis and Reiss added to my personal understanding and knowledge, and some of the chapters, such as the one by Brown, would be useful to give to residents beginning their work on an inpatient ward. It is unclear, however, that there are readers who will find most of this volume useful or interesting. Much of the book is redundant or available in better form elsewhere. For instance, Lidz should be read at greater length than the 20-some pages his chapter occupies, and Donald Bloch has much more to say than he does here.

In conclusion, editors of collections must be more exacting in whom they will and will not include in the final proceedings of a meeting because much of what sounds interesting in person is not valuable in print. They will also have to consider more carefully for whom the volume is intended.

HENRY GRUNEBAUM, M.D.
Cambridge, Mass.

Methods of Biobehavioral Research, *edited by E.A. Serafetinides, M.D., Ph.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1979, 213 pp., $19.50.*

Research in psychiatry suffers from many ills, not the least of which is the relative lack of emphasis on rigorous research training. The Seminars in Psychiatry series attempts to stimulate interest in biobehavioral research by publishing reviews and updates of the technical advances that are being made in various areas. As a volume in this series, *Methods of Biobehavioral Research* is intended to be an introduction to research methodology, a basic primer emphasizing definitions of terms and techniques and/or describing the rationale for a dozen different research areas. There is no special organization or logical sequence in the material offered (or for this particular selection), but the reader will have no difficulty discriminating between the "hard" and "soft" research areas—not only in terms of the useful information provided but in the extent to which they fulfill the ostensible goals of the volume.

The two opening chapters on neurobiochemistry by A. Yuwiler cover the basic methods of research in biochemistry and then provide an overview of neurochemical approaches in the diagnosis, treatment, and etiology of disease. W.J. Dixon deals with issues in the design of experiments and analysis of research data in a well-organized introductory lecture describing the content of a course in statistics. Dixon provides an excellent reading list covering general texts, mathematical statistics, data analysis, statistics in medical research, and publications in psychiatric research.

Oldenfeld contributes a highly readable review of the pros and cons of several neurodiagnostic procedures for evaluating organicity, emphasizing computerized tomography (CT scanning), the newest of the imaging techniques used for the investigation of brain function. "Methods in Electrophysiological Research" by R. Coger, A. Dymond, and E.A. Serafetinides discusses ongoing electrical activity in the CNS and peripheral measures of autonomic and skeletal muscle activity and includes a section on instrumentation in electrophysiology.

Although it is not a description of methods, Colby's chapter is an excellent statement of the goals, limitations, underlying concepts, and assumptions of computer simulation approaches to research in psychiatry. The chapter on psychopharmacology by Van Putten, May, and Jarvik is also not a discourse on methodology so much as a discussion that ranges from issues in the design of clinical pharmacologic research to the interpersonal interactions that are required to implement such interdisciplinary research. Rounding out the "hard" chapters is an introductory lecture by D. Shapiro dealing with the definitions of relevant features of psychophysiology and biofeedback research and their background in the study of conditioning phenomena.

The chapter on clinical and psychotherapeutic methods of research by M. Straker is a description of the problems of research and researchers in psychiatry. The author does, however, define and illustrate a variety of approaches, pointing out their strengths and weaknesses. The perspective of a biopsychosocial approach to research is offered by J. Cohen, and McGuire's chapter on ethological methods is actually devoted to a definition and justification of ethology. The chapters on family therapy by M. Lansky and nursing research by J. Messick contain some substantive material but also fail to address methodology as such.

In terms of subject matter and content, *Methods of Biobehavioral Research* is a heterogeneous collection of "introductory chapters." The editorial rationale for attempting to cover so much of biobehavioral research rather than devoting a volume to a single area of research is not clear. Personally, I would have preferred the latter. In some areas, of course, such introductory texts are available. There would seem to be a need for more extensive coverage in other areas, and, based on the content of some of these chapters, there are still other areas where there is not yet enough accumulated experience to have developed and evaluated methods and techniques appropriate to the phenomena of primary concern. Be that as it may, several of these chapters are excellent introductions to their respective subjects and are aimed, I would judge, at the novice or undergraduate student. They would make comprehensive introductory lectures summarizing the content of a course or seminar—and, I suspect, many of them are just that. However, there is often a need for just that! The professional investigator often finds it difficult to return to the most basic tenets of his or her approach. The serious beginner is likely to be sent someplace else for the basics in the methodology of the field in

which he or she may be interested. However, if this small volume does not provide everything, it may contain something you wanted to know about a particular area but were afraid to ask.

ROBERT ADER, PH.D.
Rochester, N.Y.

Mental Health Issues in Grief Counseling. Summary of Proceedings: National Conference on Mental Health Issues Related to Sudden Infant Death Syndrome, *edited by Stanley E. Weinstein, Ph.D. Rockville, Md., U.S. Department of Health, Education, and Welfare, 1979, 133 pp., no price listed (paper).*

Sudden infant death syndrome is a confrontation with the absurd. Most people have at least some cognitive awareness of their vulnerability, lack of control, and finitude, but at the same time, as parents they maintain a comfortable illusion that their infants can be loved, cradled, and protected from the world—and from death. If babies are not safe, who is? Sudden infant death syndrome is a violent cognitive and affective assault on the immortality fantasy of the parents. I recommend this collection of papers for all those who work with grief and loss in any form, and for both those who have an inkling of their own finitude and those who do not.

The conference on which this book is based offered experiences that stimulated both the minds and the feelings of the participants. George Engel involved his audience at the conference in a film made by the British Broadcasting Company on the Aberfan slag heap disaster in which 120 people were killed, most of them children. His chapter, titled "Teaching and Learning About Grief," is an affect-laden and instructive exchange between the conference participants and himself after they watched the film together. This and other contributions remind us that the catastrophe of sudden loss inspires anger and helplessness in those whose efforts to be of help seem puny when contrasted to the event.

Albert Cain contributes an excellent chapter on "The Impact of Sudden Infant Death on Families," and Lois Murphy offers another on "Early Attachments and the Effect of SIDS on Parents," particularly the mother. The severity of grief reactions and their enduring nature coupled with the ripple effect on other family members, even through the generations, is emphasized. Cain offers the timely reminder that the verbal, open, shared, and tearful expression of grief is not the one and only true way, and that territoriality often leads to helping people believe that they are the only ones with the knowledge and know-how required to be of help. Murphy points out that sudden infant death syndrome usually occurs in the first six months of life in a "honeymoon phase" during which many parents find their babies "pure joy." The parents experience the bewildering traumatic loss as loss of part of self. The loss of self-confidence, physical distress, guilt, anger, denial, emptiness, aloneness, and loss of meaning of life are all well described. Cain also emphasizes the need to give attention to siblings as well as to both parents.

The role of the counselor and the impact of the work on her or him receives attention in several papers, particularly those of Arthur Rosenbaum and George Engel. There is need for support from colleagues and for awareness that burnout will occur if there is a great imbalance in the counselor's life between giving and receiving. Rosenbaum gives many useful observations about the tasks of counseling, but his is not a "how to" chapter, which would be appropriate only if counseling were a technology rather than an art.

Donald Klein offers wisdom in "Selecting the Mental Health Consultant," and Michael Rothenberg summarizes the conference with great sensitivity. The spiritual dimension receives very little attention and needs to be included in any review of matters of life and death.

I wish that I had been at the conference to experience the affective components, but the 10 contributors have done a great service to us all in sharing their wisdom through the written word.

N. MICHAEL MURPHY, M.D.
Albany, N.Y.

Specialized Techniques in Individual Psychotherapy, *edited by Toksoz B. Karasu, M.D., and Leopold Bel'ak, M.D. New York, N.Y., Brunner/Mazel, 1980, 490 pp., $25.00.*

Twenty-two authors contribute 25 chapters focused on clinical case material that is bound to capture the interest of a wide variety of psychotherapists. Although each chapter is the product of an authority in the field, the final product reveals an uneven quality of excellence. The title led me to anticipate that the focus would be on the details of various strategies in psychotherapeutic activity. What is offered instead is a feast of interesting clinical situations, clinical problems, and diagnostic entities that provide opportunity to discuss recommended specific styles of psychotherapeutic intervention. The clinical entities include ambulatory schizophrenia, borderline personality, obsessive states, alcoholism, stress response syndrome, pathological mourning, sexual dysfunction, psychotherapy of the depressed patient, and others. Marmor (1) has pointed out that we must continue to strive toward a unified science of psychotherapy that will enable us to fit the patient, the therapist, and the technique together in a way that will most effectively, economically, and humanely achieve the desired objectives of mental health.

In the introductory overview, Dr. Karasu states that the task for the future is the achievement of greater specificity for particular kinds of intervention. In reviewing the current plethora of psychotherapies, he suggests three major topographies for clustering in relation to basic themes. These include the dynamic, behavioral, or experiential themes. He describes psychotherapy as a mutual therapist-patient endeavor to investigate and understand the nature of the patient's distress and for treatment of same. He points out that the therapeutic relationship is the dynamic balance between real object relationship, the therapeutic alliance, and transference, and he comments that a good clinician calibrates the use and the timing of the therapeutic techniques. Dr. Karasu also contributes a chapter dealing with the psychotherapy of the physically ill patient. He discusses the dynamic interactions between external stressors, characterological defenses, and coping problems and cautions that countertransference responses, which are reflected by premature confrontation, or interpretations about the patient's use of denial or affective blocking, are a major contributor to therapeutic failure in this clinical situation.

I found Dr. Kernberg's chapter on the psychotherapy of

borderline patients both interesting and helpful. The contribution by Dr. Horowitz on brief treatment of stress response syndromes is also excellent. The chapters on hypnotherapy, sexual therapy, the suicidal patient, the elderly patient, and ambulatory patients with severe anxiety are less successful. The latter article recommends the use of neuroleptics for severe neurotic anxiety and ambulatory ECT under related circumstances. These comments are not congruent with current concepts about somatic management of anxiety states.

Several chapters are unusual in that the topics concern common clinical conditions that somehow escape discussion. These include racial issues in psychotherapy (Hugh Butts, M.D.), family and marital therapy combined with individual therapy (Stephen Fleck, M.D.), and combined psychotherapy and pharmacotherapy (Gerald Sarwer-Foner, M.D.).

Throughout the book the major psychotherapeutic stance is provided by psychoanalytic and psychodynamic formulations. The flexible adaptive variations of this basic hypothesis are useful in providing an underlying theoretical and structural support for psychotherapeutic work in a wide variety of clinical settings. However, this book shares the shortcoming of many others on psychotherapy in that process is described but data on outcomes are lacking.

Whether the practitioner's interest lies in private practice, the general hospital, community clinic, or institutional psychiatry, this book provides good reading and useful clinical clues and can be a guide to better psychotherapy. The editors' hopes and objectives are fulfilled in this volume.

REFERENCE

1. Marmor J: Recent trends in psychotherapy. Am J Psychiatry 137:409–416, 1980

M. STRAKER, M.D.
Los Angeles, Calif.

Nourishing the Humanistic in Medicine: Interactions with the Social Sciences, *edited by William R. Rogers and David Barnard. Pittsburgh, Pa., University of Pittsburgh Press in cooperation with the Institute on Human Values in Medicine of the Society for Health and Human Values, 1979, 325 pp., $10.95.*

We are in a time when the pertinence of the medical model to psychiatry is passionately defended and attacked. This volume usefully expands the definition of that model in directions that should somewhat mute the controversy over its applicability in psychiatry. Those who want psychiatrists to reidentify themselves with other physicians and medical practices will find it easier to persuade psychiatrists who see their role and skills as encompassing the pathogenic and therapeutic elements in society at large. Those who promote a broader, more varied view of illness, health, and the physician will find their dimensions more comfortably capacious as herein tailored.

The book insistently and correctly turns our attention to values, an area sadly neglected in medical and psychiatric education. Values, in my view, are the specific human adaptation device. That which works (to some end or other) becomes valued. Values become organized into value systems. Value systems then develop momentum and inertia, which may then foster or frustrate all human endeavor, especially the understanding of health and unhealth and provision for their care.

The tendency of both social scientists and physicians to subdivide their concerns and approaches into hyperspecialisms that ignore the needs of whole human beings is diligently and successfully countered by this book.

This group of essays by individual authors grew out of dialogues initiated by the Institute on Human Values in Medicine, partly funded by the National Endowment for the Humanities. The participants from medicine and social sciences were provided five two-day retreats over the course of two years in which to reflect, reveal, and stimulate one another's thinking, so the results are illumined by these efforts and opportunities for the contributions to affect one another before reaching outside readers. Particularly impressive to me in this imaginative plan to humanize was the exchange among participants about their personal and professional "pilgrimages," including encounters with tragedy, illness, medical care, and healing. This process, plus the debate and discussion by the group at preliminary presentations, lend the finished chapters a degree of integration and mutual honing uncommon in contributions to an edited volume.

Theologian/psychologist William B. Rogers competently reviews and interprets for medical educators the dual factors of helplessness and power as they are experienced, taught, and learned by doctors, patients, and society.

Psychiatrist Robert J. Lifton urges the replacement of what he calls "psychic numbing" and the "technicist" model of professional intervention with a renewed emphasis on value advocacy and personal openness in dealing with critical ethical questions in medical education.

Anthropologist Anthony Oliver-Smith sensitively examines the role of the fit between the basic assumptions of two parties in a "crisis dyad," trying to construct a helpful context of meaning for suffering.

These three papers contributed most to my understanding, but historian Barbara Sicherman's analysis of the cyclical nature of the "progressive" medicine propounded by Richard Putnam and Henry Cabot is also valuable, as are the other eight chapters.

All this being said, I found the book lacking in a credible unifying view of humankind that would optimize the utility of other concepts presented. The role of intraspecific cooperation in the survival and well-being of all species, particularly our own, should have been prominently underscored. Some such humanizing formulation as that of Ashley Montagu should have been incorporated: the purpose of human life is to live as though to live and love were the same thing.

That the authors have nevertheless been faithful latently to such understanding is revealed by my wish to have been present in order to more explicitly give it tongue.

RODERIC GORNEY, M.D., PH.D.
Los Angeles, Calif.

Adaptation in Schizophrenia: The Theory of Segmental Set, *by David Shakow, Ph.D. New York, N.Y., Wiley-Interscience (John Wiley & Sons), 1979, 232 pp., $19.95.*

Ever since his early days as Chief Psychologist and Director of Psychological Research at the Worcester State Hospital, David Shakow has been interested in schizophrenia as a

clinician and as a researcher. When he writes on the subject, therefore, it behooves us to listen. The present volume sets forth his theory of segmental set. Generalized set is described as "the kind of adjustment (conscious, preconscious, and unconscious) that disposes a person to perceive a situation objectively and to respond equally objectively, in an autonomous fashion." Segmental set typical of schizophrenia "is disclosed by an inconsistency within an aspect of the response (as in cognitive dispersal) or in the incongruous combination of the cognitive, affective, and conative aspects into a spontaneous unit." The result is schizophrenic thinking.

Shakow draws heavily on his earlier work at Worcester and later at NIMH. As he states in his preface, he eschews the ideal task of integrating the enormously complex body of clinical and experimental literature bearing on the subject. The result is a somewhat disappointing volume that gives the impression at times of being thrown together hastily without adequate reference to the other literature, particularly of the later decades.

The book is introduced by a well-deserved tribute to Shakow from his colleague David Rosenthal. The beginning chapters on set theory and on the stimulus-response process that serves as context for it are clear and explicit, although one regrets Shakow's frank avoidance of any detailed integration of set theory with modern psychopharmacology, linguistics, decision theory, and cognitive psychology. There follow, however, a series of brief 3–5-page chapters on research, developmental aspects, coping behavior, symptoms, and diagnosis that promise more than they fulfill. The 3-page treatment of developmental aspects (actually about 2 pages of text) might better have been left out. The 5-page chapter on the therapeutic aspects of segmental set has little that is new and specific to set theory.

The relative neglect of the background of the set concept in history is corrected by an appendix by Ann Masten on "Set and Schema Theory in Twentieth Century American and British Psychology," which, however, relates to Shakow's work only incidentally. The appendix also has a 21-page description of the Worcester State Classification System for Schizophrenics, which although interesting in the light of the appearance of *DSM-III* is of little pertinence to the explanation of set theory. In summary, Shakow has much to offer in his clinical-descriptive treatment of segmental set but is disappointing in his theoretical and integrative development of it.

The volume has a detailed author and subject index and an adequate bibliography.

WILLIAM A. HUNT, PH.D.
Chicago, Ill.

Basic Intensive Psychotherapy, *by William H. Reid, M.D. New York, N.Y., Brunner/Mazel, 1980, 126 pp., $12.00.*

This succinct, clearly written book is specifically aimed at all beginning psychotherapists and their supervisors, not only residents in psychiatry. It has a strong psychoanalytic bias (for instance, the phenomena of psychic energy and ego defense mechanisms are repeatedly dealt with).

In his preface Dr. Reid explicitly hopes to follow in the tradition of Dr. Kenneth Colby's *Primer for Psychotherapists* (1). He succeeds admirably in this ambitious task.

The book deals with everyday realities and problems encountered in intensive psychotherapy, both those of the patient and those of the therapist. Most of the book deals with three distinct phases of therapy: 1) the opening phase, including building the therapeutic alliance, 2) the middle phase, dealing with resistance, working through, transference, countertransference, and specific psychotherapeutic techniques, and 3) the third phase, termination.

The longest section is that on the crucial middle phase of therapy. It deals with the actual, often painful and difficult work done to minimize the patient's symptoms. Dr. Reid discusses "the two basic forms" the transference may take—"a source of nourishment and love . . . or a figure of considerable authority." Working with important resistances, such as repression, projection, and denial, is discussed briefly and with a minimum of jargon.

The section on termination is especially helpful in its ideas about different types of termination—natural, forced, and premature. Trainees need much help in dealing with these issues and the guilt often stirred up within the therapist when he or she rotates onto another service and must terminate or transfer patients.

Scattered throughout the book are brief clinical vignettes dealing with the process of treatment and various therapeutic techniques. The book might be strengthened by increasing the frequency and length of excerpts from interviews, along with more examples and discussion of poor or inappropriate therapeutic interventions.

Overall, however, this book achieves its goal of briefly presenting basic concepts of psychodynamic, psychoanalytically oriented psychotherapy. The author's implicit skills as a therapist and supervisor make it a welcome addition to the literature. I recommend it to beginning therapists and their supervisors as a valuable aid in learning how to approach this subtle and fascinating form of psychotherapy.

REFERENCE

1. Colby KM: A Primer for Psychotherapists. New York, Ronald Press, 1951

CHARLES A. LEVIE, M.D.
Springfield, Ill.

Schizophrenia: A Biopsychosocial Perspective, *by Andrew Crider. Hillsdale, N.J., Lawrence Erlbaum Associates (New York, N.Y., Halsted Press, John Wiley & Sons, distributor), 1979, 194 pp., $16.50.*

This small volume is of interest to psychiatrists because it condenses and evaluates major up-to-date research and concepts of schizophrenia. Most valuable is the use of bridging concepts between descriptive psychiatry, biochemistry, genetics, and previous life history to arrive at a plausible amalgamation.

The book opens with a brief overview of changing concepts of schizophrenia from Bleuler and Kraepelin through *DSM-III*, including the problems that still remain in making a reliable clinical diagnosis of schizophrenia.

The second chapter, "The Schizophrenic Psychosis," amply illustrates the author's view of the development of the schizophrenic psychosis from its onset, marked by a mood shift toward euphoria, to early schizophrenia, marked by in-

creased perplexity because of the inability to control attention and ideation. This is followed by the schizophrenic psychosis, complete with primary and secondary delusions, hallucinations, a language disorder, and a variety of dissociative phenomena characteristic of the full-blown psychosis. The author supports these three phases by a much needed summary and synthesis of well-known research studies on reaction time and attention dysfunction.

The third chapter, on major biochemical hypotheses, is well suited for students of psychiatry and includes a parsimonious presentation of the way neurotransmitters function at synapses. The transmethylation hypothesis, the dopamine hypothesis, and the amphetamine hypothesis are clearly presented, as is the evidence for and against each. The section on the orthomolecular hypothesis at best provides a basis for refuting the outrageous pseudoscientific claims made for this approach. It is unfortunately given too much space for its importance. More important, Crider makes it abundantly clear that any single biochemical model at this time cannot be definitive because of the vast complexity of the brain and the possibility that there are myriad unknown and only partially known biochemical processes remaining to be studied.

In the economical review of genetic factors in schizophrenia, the fourth chapter, the author tends to prefer the polygenic over the modified dominance inheritance theory. He concludes that inherited predisposition is necessary but not sufficient to produce schizophrenia and reasons that genetic studies provide the best single source of evidence for the role of environmental factors in its etiology.

The chapter on life history contains a tightly reasoned review of three major retrospective developmental studies and illuminates the scientific limitations to their findings as having significance in the etiology of schizophrenia. In a similar way, Crider finds that predictive studies on high-risk populations are faced with very complex problems of selection of subjects, diagnostic criteria, and the number of decades necessary to do such a study.

In the section on developmental studies the author concludes that both uncontrolled clinical observations and controlled laboratory studies of interactions and communication patterns of families with schizophrenic children fail to demonstrate a specific developmental or life history event that characterizes preschizophrenic children or that can be isolated as being necessary for later psychosis.

The chapter on patienthood deals with the social impact of schizophrenia as demonstrated by epidemiological studies and includes the significant demographic correlates of socioeconomic status, age, and race. The next section sketches the decline of the mental hospital as the primary place to treat psychotic patients. The section on deinstitutionalization and patient care summarizes the mental health legislation, the litigation regarding human rights of mental patients, and the role of antipsychotic medications in the development of patient care in the community.

The book closes with a knowledgeable survey of the unfulfilled promises of deinstitutionalization and the myth of rehabilitation, the myth of reintegration, and the myth of continuity of care. Except for a few small sections meant for students this is an excellent review of schizophrenia fo psychiatrists.

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Books Received

This listing acknowledges the receipt of recent books. Books of particular interest to the readers of this journal will be reviewed as space permits, and copies of the reviews will be sent to the publishers. Books cannot be returned to the publishers.

Symlog: Case Study Kit with Instructions for a Group Self Study, *by Robert F. Bales. New York, N.Y., Free Press (Macmillan Publishing Co.), 1980, 17 pp., $5.95 (paper).*

Doing Psychotherapy, *by Michael Franz Basch. New York, N.Y., Basic Books, 1980, 181 pp., $15.00.*

Friendship: How to Give It, How to Get It, *by Joel D. Block, Ph.D. New York, N.Y., Macmillan Publishing Co., 1980, 226 pp., $10.95.*

Competence and Coping During Adulthood, *edited by Lynne A. Bond and James C. Rosen. Hanover, N.H., University Press of New England, 1980, 365 pp., $20.00.*

Drug-Free Therapeutic Community: An Evaluation, *by Robert C. Brook, Ed.D., and Paul C. Whitehead, Ph.D. New York, N.Y., Human Sciences Press, 1980, 149 pp., $14.95.*

Self-Disclosure: Origins, Patterns, and Implications of Openness in Interpersonal Relationships, *by Gordon J. Chelune and associates. San Francisco, Calif., Jossey-Bass, 1979, 374 pp., $16.95.*

Freud: The Man and the Cause, *by Ronald W. Clark. New York, N.Y., Random House, 1980, 617 pp., $19.95.*

The Evolution of Human Consciousness, *by John Hurrell Crook. Oxford, England, Clarendon Press (Oxford University Press), 1980, 431 pp., $39.00.*

Psychology and Psychiatry in Courts and Corrections: Controversy and Change, *by Ellsworth A. Fersh, Jr., Ph.D., J.D. New York, N.Y., John Wiley & Sons, 1980, 353 pp., $23.95.*

Toxicological and Pathological Studies on Psychoactive Drug-Involved Deaths, *by Louis A. Gottschalk, M.D., and Robert H. Cravey. Davis, Calif., Biomedical Publications, 1980, 467 pp., $42.00.*

Tradeoffs: Executive, Family and Organizational Life, *by Barrie S. Greiff, M.D., and Preston K. Munter, M.D. New York, N.Y., New American Library, 1980, 201 pp., $10.00.*

Psychotherapy Supervision: Theory, Research and Practice, *edited by Allen K. Hess. New York, N.Y., John Wiley & Sons, 1980, 530 pp., $33.50.*

The Medical Casebook of Adolf Hitler: His Illnesses, Doctors and Drugs, *by Leonard L. Heston, M.D., and Renate Heston, R.N. New York, N.Y., Stein and Day, 1979, 176 pp., $12.95.*

Assessing Schizophrenic Thinking: A Clinical and Research Instrument for Measuring Thought Disorder, *by Mary Hollis Johnston and Philip S. Holzman. San Francisco, Calif., Jossey-Bass, 1979, 301 pp., $15.95.*

Helping People Change: A Textbook of Methods, 2nd ed., *edited by Frederick H. Kanfer and Arnold P. Goldstein. New York, N.Y., Pergamon Press, 1980, 578 pp., $27.50; $13.95 (paper).*

Freud and His Patients: Downstate Psychoanalytic Institute Twenty-Fifth Anniversary Series, Vol. II, *edited by Mark Kanzer, M.D., and Jules Glenn, M.D. New York, N.Y., Jason Aronson, 1980, 444 pp., $25.00.*

Raising the Young Blind Child: A Guide for Parents and Educators, *by Shulamith Kastein, Isabelle Spaulding, and Battia Scharf. New York, N.Y., Human Sciences Press, 1980, 198 pp., no price listed.*

Gregory Bateson: The Legacy of a Scientist, *by David Lipset. Englewood Cliffs, N.J., Prentice-Hall, 1980, 354 pp., $16.95.*

Burglary and Theft, *by John M. Macdonald, M.D. Springfield, Ill., Charles C Thomas, 1980, 270 pp., $19.75.*

Principles and Practice of Biodynamic Psychotherapy: An Integration. Serial Handbook of Modern Psychiatry, Vol. IV, *by Jules H. Masserman, M.D. New York, N.Y., Thieme-Stratton, 1980, 206 pp., $20.00.*

Noncatecholoc Phenylethylamines, Part 2: Phenylethanolamine, Tyramines, and Octopamine, *edited by Aron D. Mosnaim and Marion E. Wolf. New York, N.Y., Marcel Dekker, 1980, 339 pp., $55.00.*

Becoming a Mother, *by Ann Oakley. New York, N.Y., Schocken Books, 1980, 328 pp., $18.95.*

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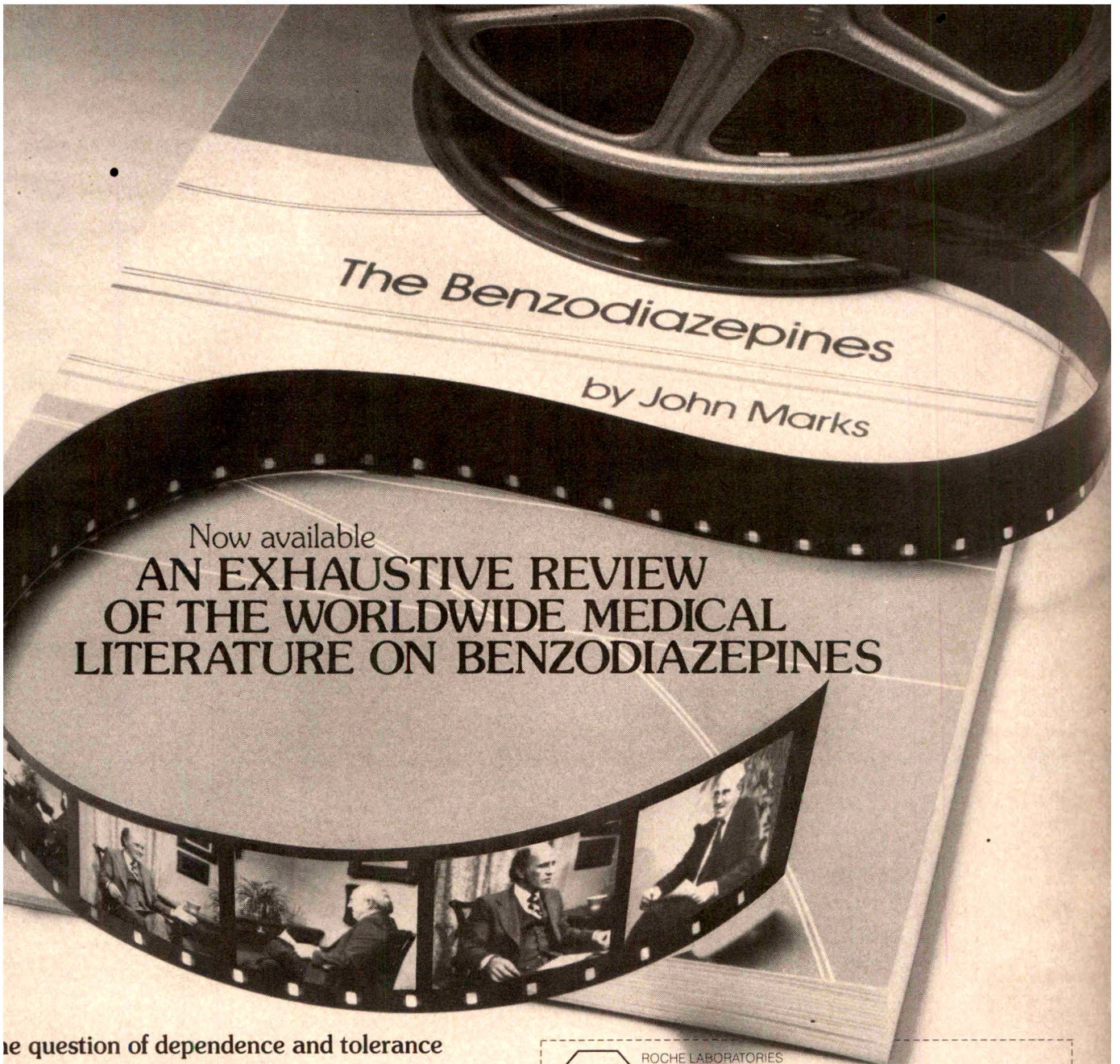
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Please send me_____copies of the monograph *The Benzodiazepines: Use, overuse, misuse, abuse* by Dr. John Marks.

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Brief Summary of Prescribing Information

Indications and Usage: Symptomatic relief of anxiety, tension, agitation, irritability and insomnia associated with anxiety neuroses and transient situational disturbances; anxiety associated with depressive symptoms and as a treatment of symptoms of anxiety if such symptoms are a significant feature of functional or organic disorders, particularly gastrointestinal or cardiovascular.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients on lorazepam not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation.

Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions.

Observe usual precautions with impaired renal or hepatic function.

Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component.

Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day. No effect dose was 1.25mg/kg/day (approximately 6 times the maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatric patients requires caution and frequent monitoring for symptoms of upper G.I. disease.

Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associateo with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug.

In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Overdosage: In management of overdosage with any drug, bear in mind that multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring of vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

Ativan® IV for (lorazepam) Anxiety

Dosage: **Individualize for maximum beneficial effects. Increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; insomnia due to anxiety or transient situational stress, 2-4mg h.s.**

How Supplied: **0.5, 1.0 and 2.0mg tablets.**

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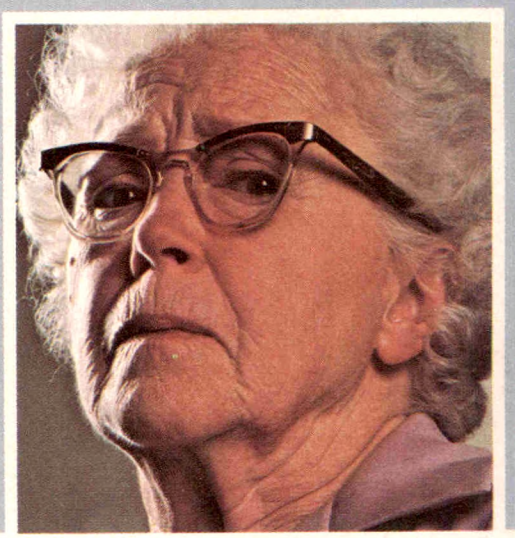
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An age for anxiety.

The aging process all too frequently is accompanied by anxiety states. When anxious older patients do not respond to your sympathetic counseling, a benzodiazepine may be the next logical step.

But which one? Here, more than in many other patient groups, the metabolites of the available compounds may make a decisive difference. Unlike diazepam, clorazepate and chlordiazepoxide, Ativan (lorazepam) has no long-acting metabolites, and its simple metabolism is less readily impaired with advancing age. Steady-state serum levels are rapidly achieved and disappear rapidly once you decide to discontinue therapy. For these reasons, residual effects are not likely to present problems. (The pharmacokinetic profile of a drug can define such characteristics as its absorption, distribution, metabolism and elimination but cannot, at present, be directly related to its therapeutic effectiveness.)

Proved effective in many nationwide, double-blind studies (including patients in older age groups), Ativan is compatible with a wide range of commonly prescribed drugs. (The benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.) Patients who have trouble swallowing solid medication appreciate the small Ativan tablet, which is tasteless and disintegrates within seconds in water or fruit juice.



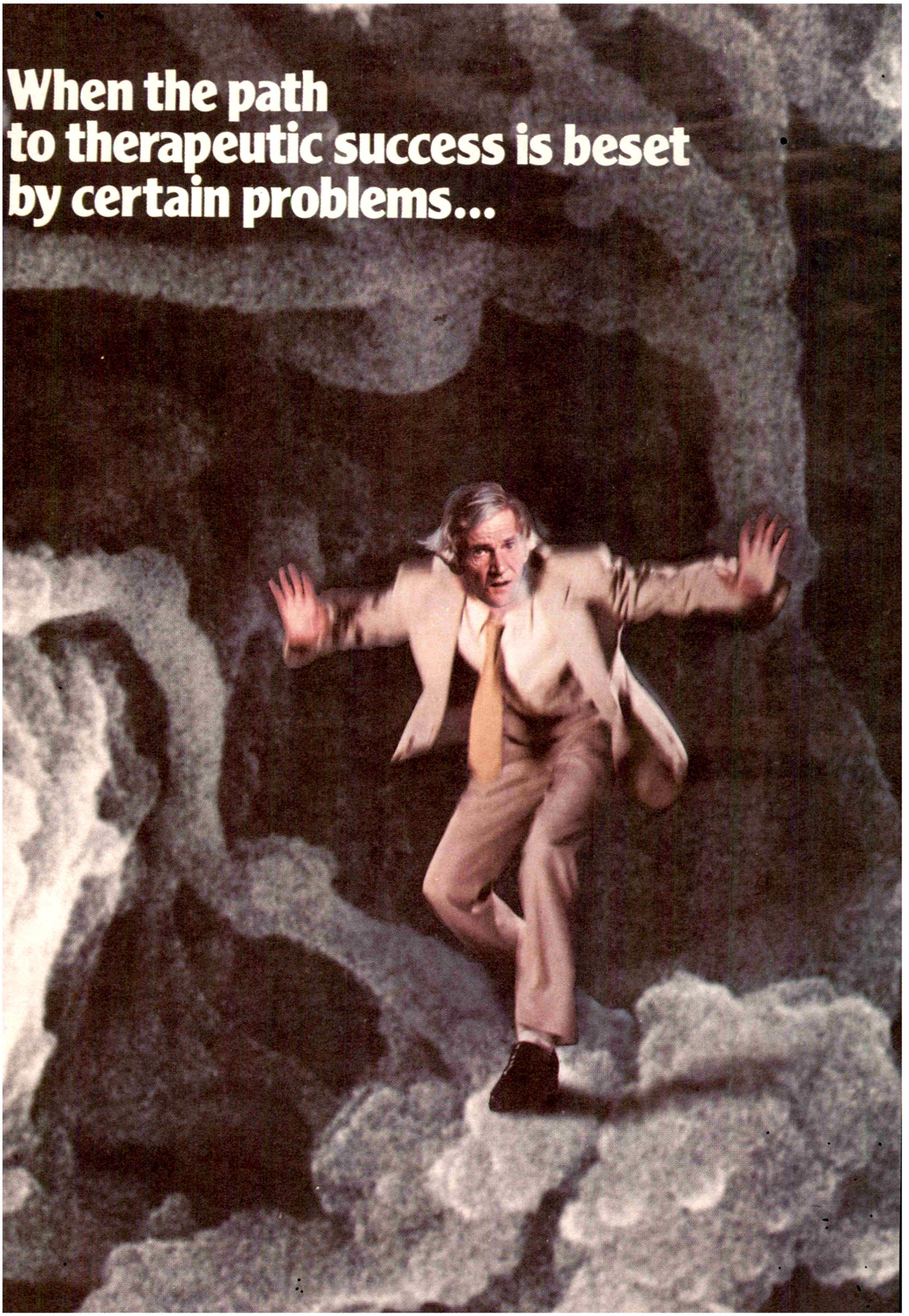
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Ativan® (lorazepam) C IV

0.5 mg tablet



for Anxiety


When the path
to therapeutic success is beset
by certain problems...

Start antidepressant therapy with

Norpramin®

(desipramine hydrochloride tablets NF)

25, 50, 75, 100, 150 mg.

to ease the patient's path

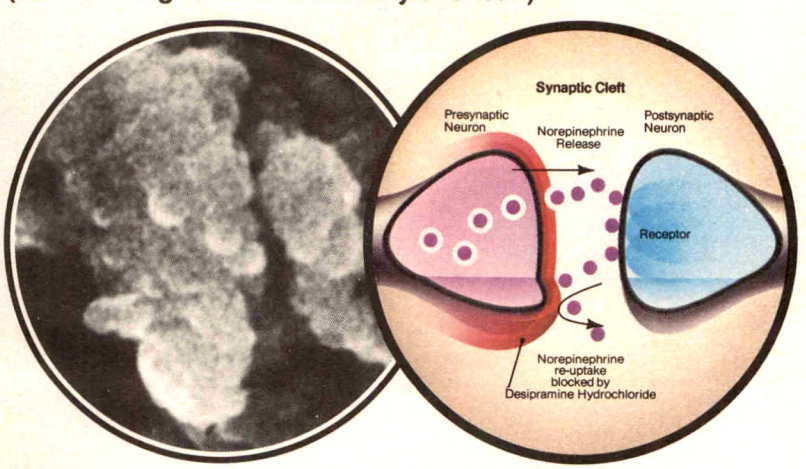
Helps relieve the sleep disturbances that often accompany depression.

- Improvement in sleep patterns may begin within one week in some patients.[1]
- As the depression is relieved, difficulty in falling asleep, restlessness, and early morning awakening diminish.
- The patient is helped to remain calm yet active, although the physician should caution the patient about driving or operating machinery if drowsiness occurs. (See Warnings, Precautions, and Adverse Reactions overleaf.)

Less anticholinergic activity

- Studies of animal tissues[2] and normal human subjects[3,4] have shown that desipramine has less anticholinergic activity than amitriptyline or doxepin.
- The single-bond side chain of desipramine may be associated with less anticholinergic activity than is the double-bond side chain of amitriptyline or doxepin.
- This may mean less dry mouth, less blurred vision, less urinary retention, less constipation.

(See Warnings in Brief Summary overleaf.)



Electron micrograph of human synaptic cleft, postulated site of tricyclic antidepressant activity.

Laboratory evidence suggests that desipramine blocks the re-uptake of norepinephrine at the presynaptic nerve terminal,[7] ensuring increased cleft and receptor site neurotransmitter concentration.

Potent blocker of norepinephrine re-uptake

Current evidence[5,6] suggests depression results from inadequate levels of norepinephrine or serotonin here, at the synaptic cleft.

If your patient is unresponsive to treatment with one tricyclic, consider switching to a tricyclic that blocks the re-uptake of a different neurotransmitter.

(Please read the Brief Summary overleaf before prescribing Norpramin.)

Merrell

MNQ-264

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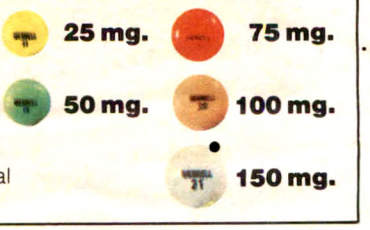
☐ Norpramin offers improved sleep patterns beginning within one week in some patients.

☐ Norpramin provides less anticholinergic activity than amitriptyline or doxepin, as shown in studies of animal tissues and normal human subjects. (See Warnings section of Brief Summary.)

☐ Norpramin is a potent blocker of norepinephrine re-uptake.

Dosage Flexibility

Convenient choice of 5 tablet strengths and once daily or divided dosage schedule allows titration to individual response.



Norpramin®
(desipramine hydrochloride tablets NF)

AVAILABLE ONLY ON PRESCRIPTION

Brief Summary

MECHANISM OF ACTION: Available evidence suggests that many depressions have a biochemical basis in the form of a relative deficiency of neurotransmitters such as norepinephrine and serotonin. Norepinephrine deficiency may be associated with relatively low urinary 3-methoxy-4-hydroxyphenyl glycol (MHPG) levels, while serotonin deficiencies may be associated with low spinal fluid levels of 5-hydroxyindolacetic acid.

While the precise mechanism of action of the tricyclic antidepressants is unknown, a leading theory suggests that they restore normal levels of neurotransmitters by blocking the re-uptake of these substances from the synapse in the central nervous system.

Evidence indicates that the secondary amine tricyclic antidepressants, including Norpramin, may have greater activity in blocking the re-uptake of norepinephrine. Tertiary amine tricyclic antidepressants, such as amitriptyline, may have greater effect on serotonin re-uptake.

Norpramin (desipramine hydrochloride) is not a monoamine oxidase (MAO) inhibitor and does not act primarily as a central nervous system stimulant. It has been found in some studies to have a more rapid onset of action than imipramine. Earliest therapeutic effects may occasionally be seen in 2 to 5 days, but full treatment benefit usually requires 2 to 3 weeks to obtain.

INDICATIONS: Norpramin (desipramine hydrochloride) is indicated for relief of symptoms in various depressive syndromes, especially endogenous depression.

CONTRAINDICATIONS: Desipramine hydrochloride should not be given in conjunction with, or within 2 weeks of, treatment with an MAO inhibitor drug; hyperpyretic crises, severe convulsions, and death have occurred in patients taking MAO inhibitors and tricyclic antidepressants. When Norpramin (desipramine hydrochloride) is substituted for an MAO inhibitor, at least 2 weeks should elapse between treatments. Norpramin should then be started cautiously and should be increased gradually.

The drug is contraindicated in the acute recovery period following myocardial infarction. It should not be used in those who have shown prior hypersensitivity to the drug. Cross sensitivity between this and other dibenzazepines is a possibility.

WARNINGS: **1.** Extreme caution should be used when this drug is given in the following situations: a. In patients with cardiovascular disease, because of the possibility of conduction defects, arrhythmias, tachycardias, strokes, and acute myocardial infarction. b. In patients with a history of urinary retention or glaucoma, because of the anticholinergic properties of the drug. c. In patients with thyroid disease or those taking thyroid medication, because of the possibility of cardiovascular toxicity, including arrhythmias. d. In patients with a history of seizure disorder, because this drug has been shown to lower the seizure threshold. **2.** This drug is capable of blocking the antihypertensive effect of guanethidine and similarly acting compounds. **3.** USE IN PREGNANCY: Safe use of desipramine hydrochloride during pregnancy and lactation has not been established; therefore, if it is to be given to pregnant patients, nursing mothers, or women of childbearing potential, the possible benefits must be weighed against the possible hazards to mother and child. Animal reproductive studies have been inconclusive. **4.** USE IN CHILDREN: Norpramin (desipramine hydrochloride) is not recommended for use in children since safety and effectiveness in the pediatric age group have not been established. **5.** The patient should be cautioned that this drug may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery. **6.** In patients who may use alcohol excessively, it should be borne in mind that the potentiation may increase the danger inherent in any suicide attempt or overdosage.

PRECAUTIONS: **1.** It is important that this drug be dispensed in the least possible quantities to depressed outpatients, since suicide has been accomplished with this class of drug. Ordinary prudence requires that children not have access to this drug or to potent drugs of any kind; if possible this drug should be dispensed in containers with child-resistant safety closures. Storage of this drug in the home must be supervised responsibly. **2.** If serious adverse effects occur, dosage should be reduced or treatment should be altered. **3.** Norpramin (desipramine hydrochloride) therapy in patients with manic-depressive illness may induce a hypomanic state after the depressive phase terminates. **4.** The drug may cause exacerbation of psychosis in schizophrenic patients. **5.** Close supervision and careful adjustment of dosage are required when this drug is given concomitantly with anticholinergic or sympathomimetic drugs. **6.** Patients should be warned that while taking this drug their response to alcoholic beverages may be exaggerated. **7.** Clinical experience in the concurrent administration of ECT and antidepressant drugs is limited. Thus, if such treatment is essential, the possibility of increased risk relative to benefits should be considered. **8.** The sedative effects of Norpramin and benzodiazepines (*e.g.*, chlordiazepoxide or diazepam) are additive. Both the sedative and anticholinergic effects of the major tranquilizers are additive to those of Norpramin. **9.** This drug should be discontinued as soon as possible prior to elective surgery because of the possible cardiovascular effects. Hypertensive episodes have been observed during surgery in patients taking desipramine hydrochloride. **10.** Both elevation and lowering of blood sugar levels have been reported. **11.** Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathologic neutrophil depression. **12.** Norpramin 25, 50, 75, and 100 mg. tablets contain FD&C Yellow No. 5 (tartrazine), which may cause allergic-type reactions (including bronchial asthma) in certain susceptible individuals. Although the overall incidence of FD&C Yellow No. 5 (tartrazine) sensitivity in the general population is low, it is frequently seen in patients who also have aspirin hypersensitivity.

ADVERSE REACTIONS: NOTE: Included in the following listing are a few adverse reactions that have not been reported with this specific drug. However, the pharmacologic similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when Norpramin (desipramine hydrochloride) is given.

Cardiovascular: hypotension, hypertension, tachycardia, palpitation, arrhythmias, heart block, myocardial infarction, stroke.

Psychiatric: confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurologic: numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures; alteration in EEG patterns; tinnitus.

Anticholinergic: dry mouth, and rarely associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis, increased intraocular pressure; constipation, paralytic ileus; urinary retention, delayed micturition, dilatation of urinary tract.

Allergic: skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight), edema (of face and tongue or general), drug fever, cross sensitivity with other tricyclic drugs.

Hematologic: bone marrow depressions including agranulocytosis, eosinophilia, purpura, thrombocytopenia.

Gastrointestinal: anorexia, nausea and vomiting, epigastric distress, peculiar taste, abdominal cramps, diarrhea, stomatitis, black tongue.

Endocrine: gynecomastia in the male, breast enlargement and galactorrhea in the female; increased or decreased libido, impotence, testicular swelling; elevation or depression of blood sugar levels.

Other: jaundice (simulating obstructive), altered liver function; weight gain or loss; perspiration, flushing; urinary frequency, nocturia; parotid swelling; drowsiness, dizziness, weakness and fatigue, headache; alopecia.

Withdrawal Symptoms: Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache, and malaise.

DOSAGE AND ADMINISTRATION: Not recommended for use in children. Lower dosages are recommended for elderly patients and adolescents. Lower dosages are also recommended for outpatients compared to hospitalized patients, who are closely supervised. Dosage should be initiated at a low level and increased according to clinical response and any evidence of intolerance. Following remission, maintenance medication may be required for a period of time and should be at the lowest dose that will maintain remission.

Usual Adult Dose: The usual adult dose is 100 to 200 mg. per day. In more severely ill patients, dosage may be further increased gradually to 300 mg./day if necessary. Dosages above 300 mg./day are not recommended.

Dosage should be initiated at a lower level and increased according to tolerance and clinical response.

Treatment of patients requiring as much as 300 mg. should generally be initiated in hospitals, where regular visits by the physician, skilled nursing care, and frequent electrocardiograms (ECG's) are available.

The best available evidence of impending toxicity from very high doses of Norpramin is prolongation of the QRS or QT intervals on the ECG. Prolongation of the PR interval is also significant, but less closely correlated with plasma levels. Clinical symptoms of intolerance, especially drowsiness, dizziness, and postural hypotension, should also alert the physician to the need for reduction in dosage. Plasma desipramine measurement would constitute the optimal guide to dosage monitoring.

Initial therapy may be administered in divided doses or a single daily dose.

Maintenance therapy may be given on a once-daily schedule for patient convenience and compliance.

Adolescent and Geriatric Dose: The usual adolescent and geriatric dose is 25 to 100 mg. daily.

Dosage should be initiated at a lower level and increased according to tolerance and clinical response to a usual maximum of 100 mg. daily. In more severely ill patients, dosage may be further increased to 150 mg./day. Doses above 150 mg./day are not recommended in these age groups.

Initial therapy may be administered in divided doses or a single daily dose.

Maintenance therapy may be given on a once-daily schedule for patient convenience and compliance.

OVERDOSAGE: See prescribing information for a discussion of symptoms and treatment of overdose.

Product Information as of July, 1979

References:

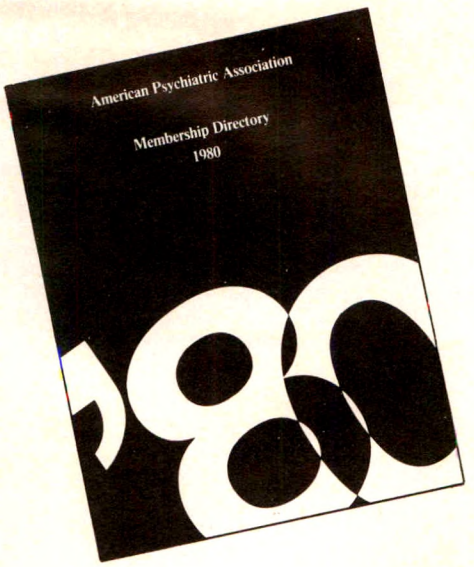
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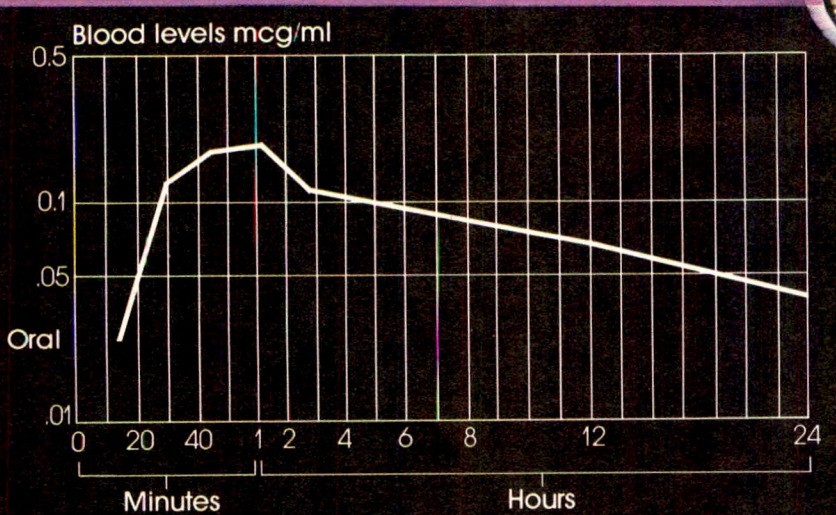
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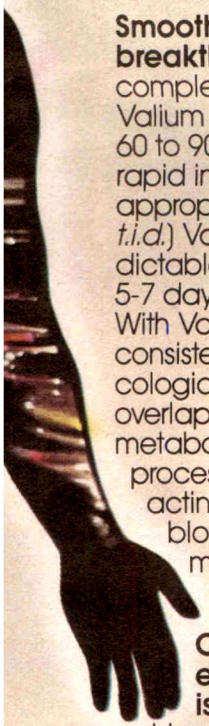
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Fast-in



24-hour blood level curve of 10-mg oral dose of diazepam in a single healthy subject.

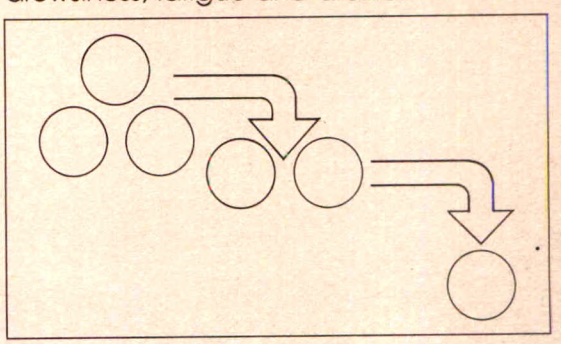
Smooth-out



Smooth: Avoids sudden symptom breakthrough Because of its fast and complete absorption, peak blood levels of Valium (diazepam/Roche) are achieved in 60 to 90 minutes after a single dose for a rapid initial calming effect. Typically, with an appropriate dosage regimen (e.g., 5 mg *t.i.d.*) Valium builds in a uniform and predictable way to a steady-state level within 5-7 days, without accumulating beyond. With Valium the excretion rate is similarly consistent and predictable. When pharmacologic support is no longer needed, the overlapping half-lives of diazepam and its metabolites insure a gradual elimination process. Because metabolites of short-acting benzodiazepines are inactive, blood levels of these drugs decrease more rapidly without benefit of a tapering effect.

Consistent therapeutic effect even when an occasional dose is omitted Once steady-state is achieved, sudden symptom breakthrough is highly unlikely, even if a dose is forgotten or skipped. That's because the half-life of Valium—27-37 hours—provides the advantage of self-tapering action, complementing your gradual reduction of the patient's dosage and smoothing his transition to independent coping.

To discontinue smoothly... Gradual discontinuation of medication—while rarely necessary after short-term therapy—*is* good medical practice in patients who have received high doses for extended periods. When dosage is tapered, untoward side effects can be avoided. Throughout therapy, in fact, side effects are infrequent with Valium and are seldom more serious than drowsiness, fatigue and ataxia.



Before prescribing, please see summary of product information on following page. ROCHE®

2-mg, 5-mg, 10-mg scored tablets

Valium®

Smooth transition to independent coping

diazepam/Roche®

Valium®
diazepam/Roche

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Management of anxiety disorders, or short-term relief of symptoms of anxiety; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).
The effectiveness of Valium in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension; changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Anxiety disorders, symptoms of anxiety, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

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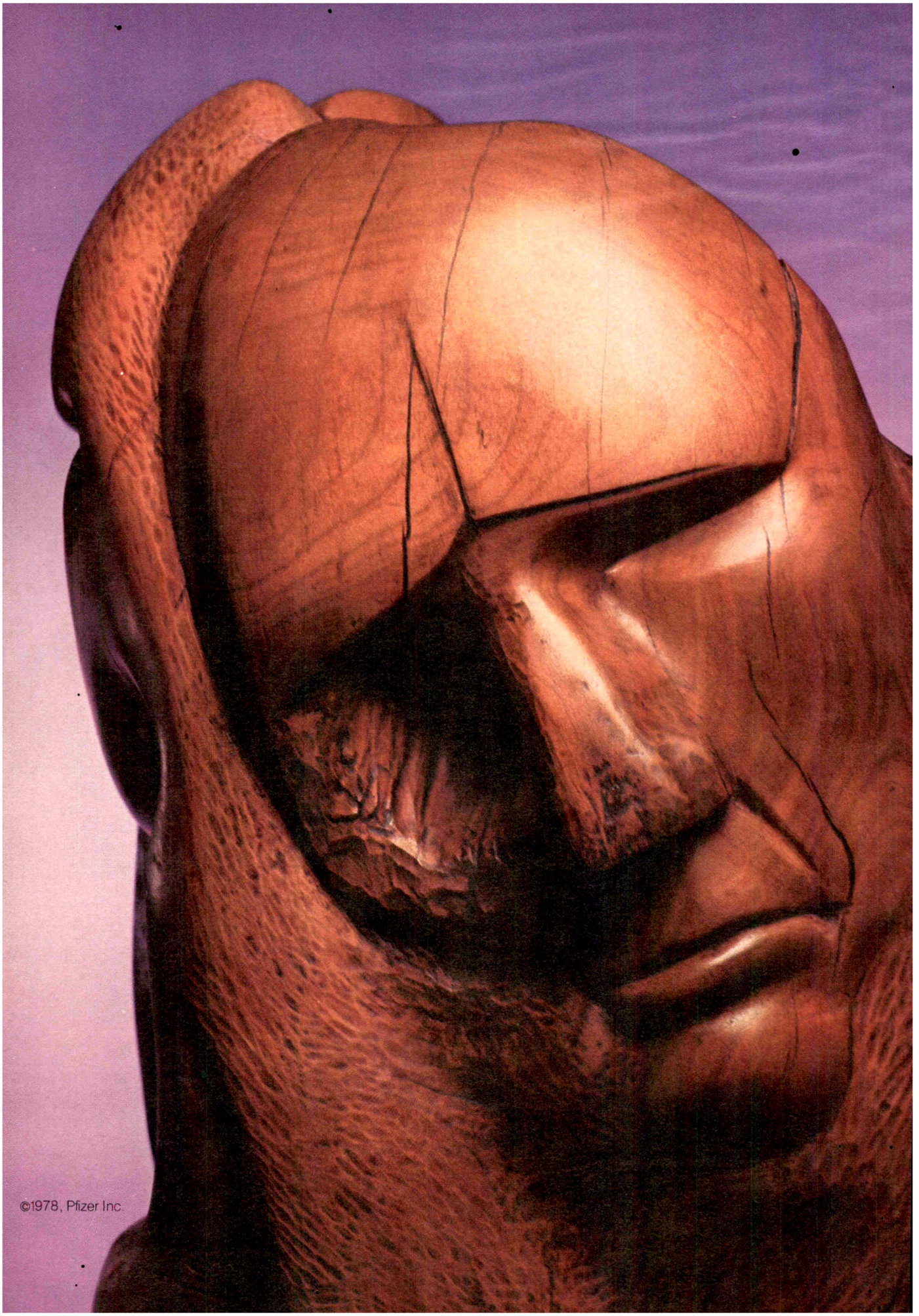
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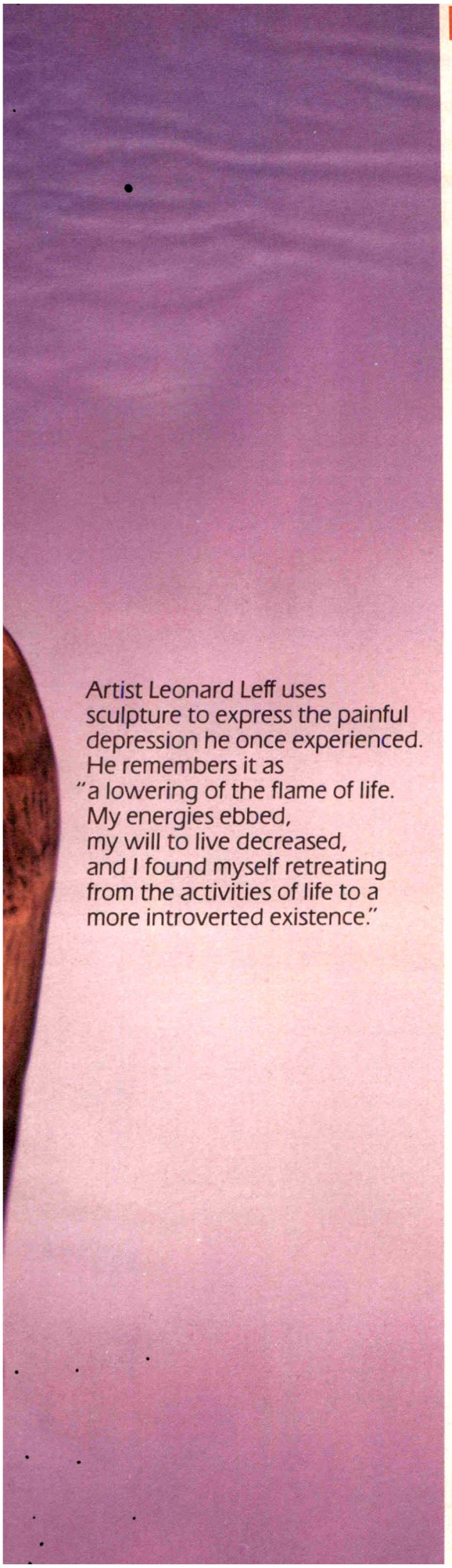
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† The 150-mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.

See Brief Summary on next page for information on contraindications, warnings, precautions and adverse reactions.

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which may improve patient compliance. The total daily dosage, up to 150 mg per day, may be given on a once-a-day schedule without loss of effectiveness. Sinequan may also be given on a divided dosage schedule, up to 300 mg per day.

PROMINENT SEDATIVE EFFECT

which may help to relieve the difficulty in falling and staying asleep, and the early-morning awakening often associated with depression.

ESTABLISHED ANTIANXIETY ACTIVITY

to help alleviate the anxiety which often accompanies clinical depression.

USUALLY WELL TOLERATED

At doses up to 150 mg per day, Sinequan does not generally affect the antihypertensive activity of guanethidine and related compounds. Tachycardia and hypotension have been reported occasionally. Drowsiness is the most commonly observed side effect. Dry mouth, blurred vision, constipation and urinary retention have been reported.

EXTENDED RANGE OF DOSAGE STRENGTHS

for flexibility in individualizing therapy.

*The 150-mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.

BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate

Contraindications. Contraindicated in individuals who have shown hypersensitivity to the drug, and in patients with glaucoma or a tendency to urinary retention. These disorders should be ruled out, particularly in older patients. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind.

Warnings. The once-a-day dosage regimen of SINEQUAN (doxepin HCl) in patients with intercurrent illness or patients taking other medications should be carefully adjusted. This is especially important in patients receiving other medications with anticholinergic effects.

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies performed in animals have shown no evidence of harm to the animal fetus. Since there is no experience in pregnant women receiving this drug, safety in pregnancy has not been established. There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.

Usage in Children: Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors. Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug. The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered and the dosage involved.

Usage with Alcohol: It should be borne in mind that alcohol ingestion may increase the danger inherent in any intentional or unintentional SINEQUAN overdosage. This is especially important in patients who may use alcohol excessively.

Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug.

Patients should also be cautioned that their response to alcohol may be potentiated.

Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy. Prescriptions should be written for the smallest feasible amount.

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.

Adverse Reactions. NOTE: Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN.

Anticholinergic Effects: Dry mouth, blurred vision, constipation, and urinary retention have been reported. If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage.

Central Nervous System Effects: Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued. Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, numbness, paresthesias, ataxia, and extrapyramidal symptoms and seizures.

Cardiovascular: Cardiovascular effects including hypotension and tachycardia have been reported occasionally.

Allergic: Skin rash, edema, photosensitization, and pruritus have occasionally occurred.

Hematologic: Eosinophilia has been reported in a few patients. There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura.

Gastrointestinal: Nausea, vomiting, indigestion, taste disturbances, diarrhea, anorexia, and aphthous stomatitis have been reported. (See anticholinergic effects.)

Endocrine: Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.

Other: Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice, alopecia, and headache have been occasionally observed as adverse effects.

Dosage and Administration. For most patients with illness of mild to moderate severity, a starting daily dose of 75 mg is recommended. Dosage may subsequently be increased o decreased at appropriate intervals and according to individual response. The usual optimum dose range is 75 mg/day to 150 mg/day.

In more severely ill patients higher doses may be required with subsequent gradual increase t 300 mg/day if necessary. Additional therapeutic effect is rarely to be obtained by exceeding a dose of 300 mg/day.

In patients with very mild symptomatology or emotional symptoms accompanying organic disease, lower doses may suffice. Some of these patients have been controlled on doses as lov as 25-50 mg/day.

The total daily dosage of SINEQUAN (doxepin HCl) may be given on a divided or once-a-da dosage schedule. If the once-a-day schedule is employed the maximum recommended dose i 150 mg/day. This dose may be given at bedtime. **The 150 mg capsule strength is intended fo maintenance therapy only and is not recommended for initiation of treatment.**

Antianxiety effect is apparent before the antidepressant effect. Optimal antidepressant effec may not be evident for two to three weeks.

Overdosage.

A. Signs and Symptoms

1. Mild: Drowsiness, stupor, blurred vision, excessive dryness of mouth.

2. Severe: Respiratory depression, hypotension, coma, convulsions, cardiac arrhythmias an tachycardias.

Also: urinary retention (bladder atony), decreased gastrointestinal motility (paralytic ileus) hyperthermia (or hypothermia), hypertension, dilated pupils, hyperactive reflexes.

B. Management and Treatment

1. Mild: Observation and supportive therapy is all that is usually necessary.

2. Severe: Medical management of severe SINEQUAN overdosage consists of aggressive supportive therapy. If the patient is conscious, gastric lavage, with appropriate precautions t prevent pulmonary aspiration, should be performed even though SINEQUAN is rapidly absorbed The use of activated charcoal has been recommended, as has been continuous gastric lavage with saline for 24 hours or more. An adequate airway should be established in comatose patient and assisted ventilation used if necessary. EKG monitoring may be required for several days since relapse after apparent recovery has been reported. Arrhythmias should be treated with th appropriate antiarrhythmic agent. It has been reported that many of the cardiovascular and CNS symptoms of tricyclic antidepressant poisoning in adults may be reversed by the slow intra venous administration of 1 mg to 3 mg of physostigmine salicylate. Because physostigmine i rapidly metabolized, the dosage should be repeated as required. Convulsions may respond t standard anticonvulsant therapy; however, barbiturates may potentiate any respiratory depres sion. Dialysis and forced diuresis generally are not of value in the management of overdosag due to high tissue and protein binding of SINEQUAN.

Supply. SINEQUAN is available as capsules containing doxepin HCl equivalent to: 10 mg, 75 mg and 100 mg doxepin: bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's). 25 mg an 50 mg doxepin: bottles of 100, 1000, 5000, and unit-dose packages of 100 (10 x 10's). 150 m doxepin: bottles of 50, 500, and unit-dose packages of 100 (10 x 10's). SINEQUAN Ora Concentrate (10 mg/ml) is available in 120 ml bottles with an accompanying dropper calibrate at 5 mg, 10 mg, 15 mg, 20 mg, and 25 mg. Each ml contains doxepin HCl equivalent to 10 m doxepin. Just prior to administration, SINEQUAN Oral Concentrate should be diluted wit approximately 120 ml of water, whole or skimmed milk, or orange, grapefruit, tomato, prune o pineapple juice. SINEQUAN Oral Concentrate is not physically compatible with a number o carbonated beverages. For those patients requiring antidepressant therapy who are o methadone maintenance, SINEQUAN Oral Concentrate and methadone syrup can be mixe together with Gatorade®, lemonade, orange juice, sugar water, Tang®, or water; but not wit grape juice. Preparation and storage of bulk dilutions is not recommended.

More detailed professional information available on request.

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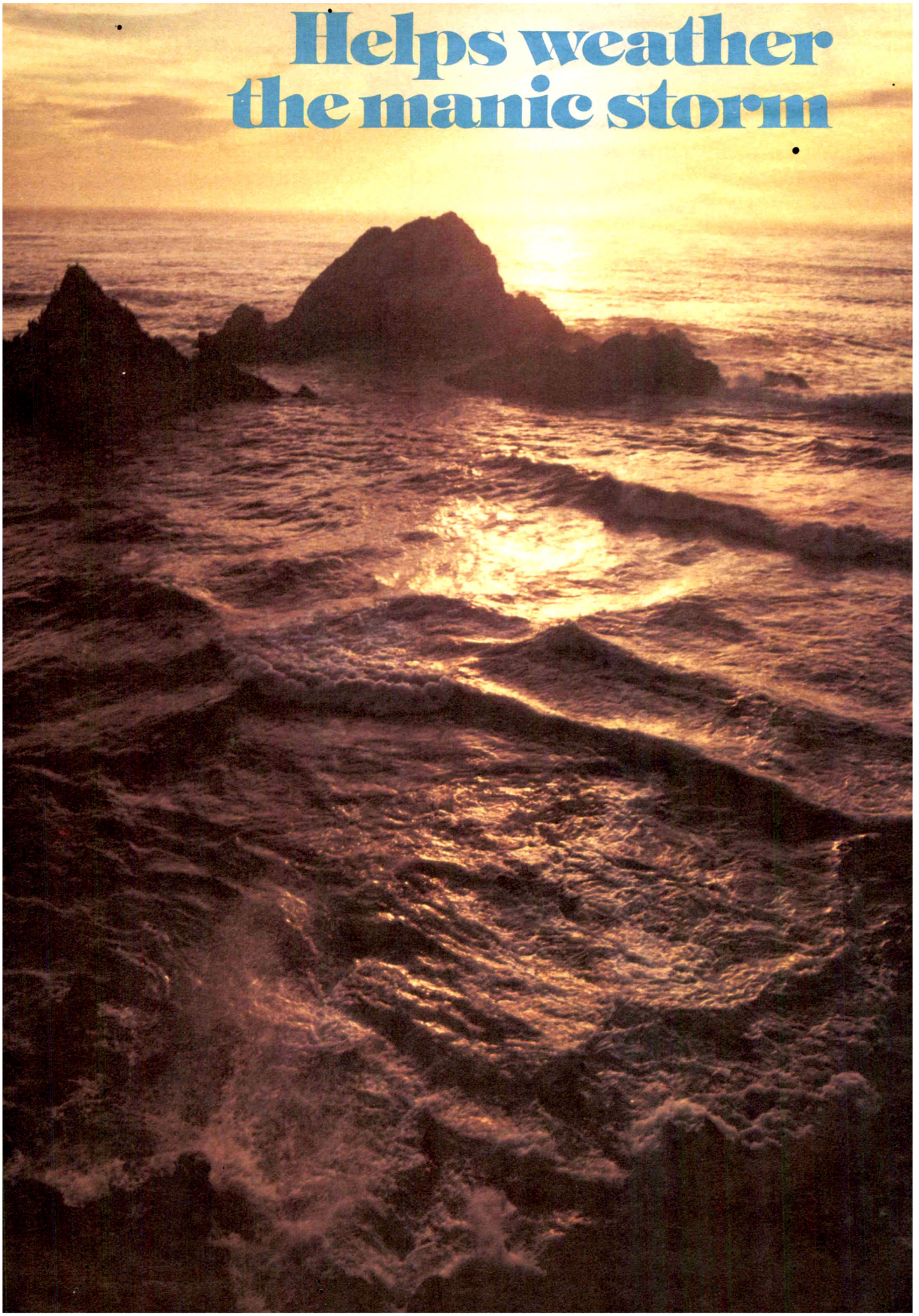
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- **prevents or diminishes the intensity of future episodes**
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- **helps patients function within normal limits**

Before prescribing, see complete prescribing information in SK&F literature or PDR. The following is a brief summary.

> **WARNING**
> Lithium toxicity is closely related to serum lithium levels, and can occur at doses close to therapeutic levels. Facilities for prompt and accurate serum lithium determinations should be available before initiating therapy.

INDICATIONS: Treatment of manic episodes of manic-depressive illness. Maintenance therapy prevents or diminishes the intensity of subsequent episodes in manic-depressive patients with a history of mania.

WARNINGS: Lithium should generally not be given to patients with significant renal or cardiovascular disease, severe debilitation or dehydration, sodium depletion, or to patients receiving diuretics.

Lithium may prolong the effects of neuromuscular blocking agents. Such agents should be given with caution to patients receiving lithium. Lithium therapy has been reported in some cases to be associated with morphologic changes in the kidneys. The relationship between such changes and renal function has not been established.

Caution patient and family to watch for diarrhea, vomiting, tremor, mild ataxia, drowsiness, or muscular weakness as signs of lithium toxicity, and to discontinue therapy and contact a physician should they occur. Patients receiving combined therapy with lithium and an antipsychotic should be monitored closely for early evidence of neurologic toxicity and treatment discontinued promptly if such signs appear. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery).

Lithium should not be used in pregnancy, especially during the first trimester, unless potential benefits outweigh possible hazards. Except in rare and unusual circumstances, nursing should not be undertaken while a patient is on lithium therapy, since lithium is excreted in human milk.

Not recommended for children under 12.

PRECAUTIONS: Lithium tolerance is greater during the acute manic phase and decreases when manic symptoms subside.

Lithium therapy may lead to sodium depletion. Normal diet (including salt) and adequate fluid intake (2500-3000 ml.) must be maintained, at least during initial stabilization period. Protracted sweating or diarrhea can decrease tolerance; in such cases, administer supplemental fluid and salt.

Sweating, diarrhea, and concomitant infection with elevated temperatures may require temporary reduction or cessation of dosage.

Where hypothyroidism exists, thyroid function should be monitored during lithium stabilization and maintenance; where hypothyroidism occurs during stabilization and maintenance, supplemental thyroid treatment may be used.

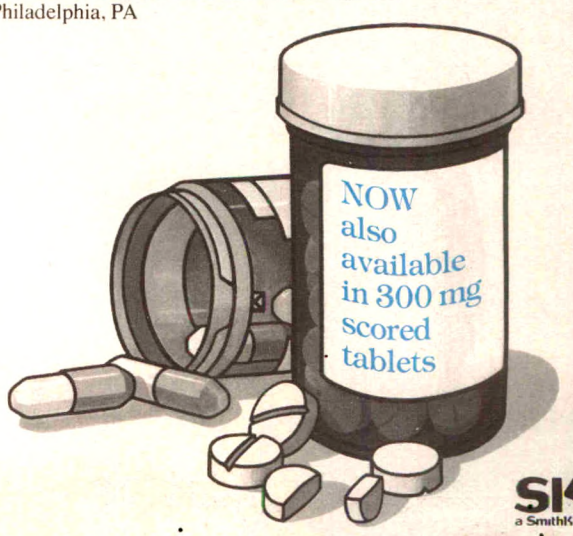
ADVERSE REACTIONS: Mild to moderate toxic reactions may occur at serum lithium levels from 1.5 to 2.5 mEq./1., and moderate to severe reactions at levels from 2.0 to 2.5 mEq./1. Fine hand tremor, polyuria, and mild thirst may occur during initial therapy and persist. Transient and mild nausea and general discomfort may also appear during initial therapy. These effects usually subside with continued treatment or temporary reduction or cessation of dosage. If persistent, discontinue dosage. Diarrhea, vomiting, drowsiness, muscular weakness, and lack of coordination may be early signs of toxicity and may occur at levels below 2.0 mEq./1. At higher levels, ataxia, giddiness, tinnitus, blurred vision, and a large output of dilute urine may be seen. Serum levels above 3.0 mEq./1. may produce a complex clinical picture, involving multiple organs and systems. Serum levels should not exceed 2.0 mEq./1. during acute phase.

The following reactions appear to be related to serum lithium levels, including levels within the therapeutic range: **Neuromuscular**—tremor, muscle hyperirritability (fasciculations, twitching, clonic movements of whole limbs), ataxia, choreo-athetotic movements, hyperactive deep tendon reflex; **Central Nervous System**—blackout spells, epileptiform seizures, slurred speech, dizziness, vertigo, incontinence of urine or feces, somnolence, psychomotor retardation, restlessness, confusion, stupor, coma; **Cardiovascular**—cardiac arrhythmia, hypotension, peripheral circulatory collapse; **Gastrointestinal**—anorexia, nausea, vomiting, diarrhea; **Genitourinary**—albuminuria, oliguria, polyuria, glycosuria; **Dermatologic**—drying and thinning of hair, alopecia, anesthesia of skin, chronic folliculitis, exacerbation of psoriasis, xerosis cutis; **Autonomic**—blurred vision, dry mouth; **Thyroid Abnormalities**—euthyroid goiter and/or hypothyroidism (including myxedema) with lower T_3 and T_4. I^{131} uptake may be elevated; rare cases of hyperthyroidism; **EEG Changes**—diffuse slowing, widening of the frequency spectrum, potentiation and disorganization of background rhythm; **EKG Changes**—reversible flattening, isoelectricity or inversion of T-waves; **Miscellaneous**—fatigue, lethargy, transient scotomata, dehydration, weight loss, tendency to sleep.

Reactions unrelated to dosage include: transient EEG and EKG changes, leukocytosis, headache, diffuse nontoxic goiter with or without hypothyroidism, transient hyperglycemia, generalized pruritus with or without rash, cutaneous ulcers, albuminuria, worsening of organic brain syndromes, excessive weight gain, edematous swelling of ankles or wrists, thirst or polyuria, sometimes resembling diabetes insipidus, and metallic taste. A single case of a syndrome resembling Raynaud's has been reported.

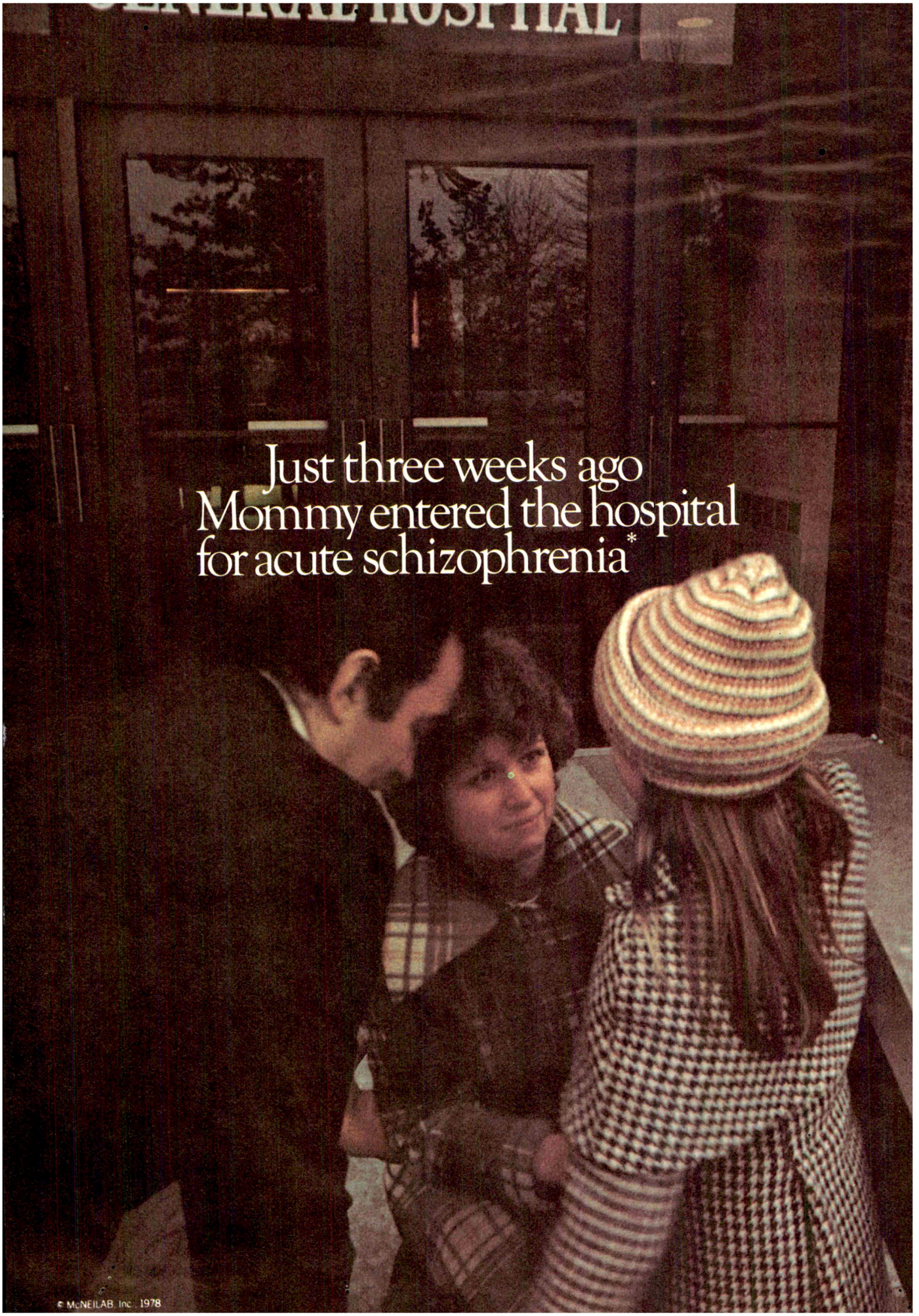
HOW SUPPLIED: 300 mg. capsules in bottles of 100.
300 mg. scored tablets in bottles of 100.

Smith Kline &French Laboratories
Philadelphia, PA



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A61



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Haldol®
(haloperidol)
tablets/concentrate/injection

Promptly controls psychotic symptoms... usually leaving the patient more responsive to therapy

Rapid onset of therapeutic effect in a wide range of psychotic symptoms.[1-4]

Helps avoid long-term hospitalization.[1-3,5]

Minimal risk of hypotension, oversedation, or troublesome anticholinergic effects.[1,4,6,7,9-11]
Transient hypotension occurs rarely; severe orthostatic hypotension has not been reported. Although some instances of drowsiness have been reported, marked sedation is rare.

Permits aggressive titration to effective dosage levels for optimal control.[5,6]

Often leaves patient with improved insight,
with a renewed interest in the environment, and more likely to participate in therapeutic efforts.[1,7]

Common side effects easily controlled.[1,4-6,8,9]
Although extrapyramidal symptoms (EPS) have been reported frequently, they are usually dose-related and readily controlled with dose adjustment or antiparkinson drugs. EPS often diminish spontaneously with continued use of HALDOL haloperidol.

References: 1. Ayd, F.J., Jr.: Med. Sci. *18*:55 (Oct.) 1967. 2. Rapp, M.S.: Can. Psychiatr. Assoc. J. *15*:73 (Feb.) 1970. 3. Rubin, R.: Ala. J. Med. Sci. *8*:414 (Oct.) 1971. 4. Abuzzahab, F.S., Sr.: Psychosomatics *11*:188 (May-June) 1970. 5. Man, P.L.: Dis. Nerv. Syst. *34*:113 (Feb.) 1973. 6. Howard, J.S.: Dis. Nerv. Syst. *35*:458 (Oct.) 1974. 7. Sugerman, A.A., et al.: Am. J. Psychiatry *120*:1190 (June) 1964. 8. Darling, H.F.: Dis. Nerv. Syst. *34*:364 (Oct.-Nov.) 1973. 9. Gerle, B.: Clin. Trials J. *3*:380 (Feb.) 1966. 10. Snyder, S.H., et al.: Science *184*:1243 (June 21) 1974. 11. Stimmel, C.L.: Dis. Nerv. Syst. *34*:219 (Apr.) 1976.

*Not an actual case history, this situation illustrates the action of HALDOL haloperidol as reported in various clinical studies (available on request).

Please turn page for summary of prescribing information.

concentrate

A tasteless, odorless, colorless Liquid Concentrate for better patient acceptability: 2 mg per ml haloperidol (as the lactate).

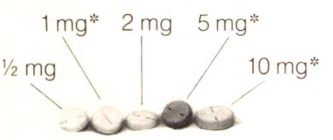
injection

A rapid-acting injection for psychiatric emergencies: 5 mg haloperidol (as the lactate) with 1.8 mg methylparaben and 0.2 mg propylparaben per ml, and lactic acid for pH adjustment to 3.4 ± 0.2.



tablets

5 tablet strengths for convenience in individualizing dosage:



HALDOL® (haloperidol)

*contain FD&C Yellow No. 5 (see Precautions).

tablets/concentrate/injection

A dosage form for every therapeutic need

Summary of Prescribing Information

Contraindications: Severe, toxic CNS depression or comatose states from any cause, hypersensitivity to the drug, Parkinson's disease.

Warnings: ***Usage in Pregnancy:*** Safe use in pregnancy or in women likely to become pregnant has not been established; use only if benefit clearly justifies potential hazards. Infants should not be nursed during drug treatment.

Combined Use With Lithium: Patients receiving lithium plus haloperidol should be monitored closely for early evidence of neurological toxicity.

General: Bronchopneumonia, sometimes fatal, has followed use of major tranquilizers, including haloperidol. Prompt remedial therapy should be instituted if dehydration, hemoconcentration or reduced pulmonary ventilation occurs, especially in the elderly. Decreased serum cholesterol and/or cutaneous and ocular changes have been reported with chemically-related drugs, although not with haloperidol. Mental and/or physical abilities required for hazardous tasks or driving may be impaired. Alcohol should be avoided due to possible additive effects and hypotension.

Precautions: Administer cautiously to patients: (1) with severe cardiovascular disorders, due to the possibility of transient hypotension and/or precipitation of anginal pain (if a vasopressor is required, epinephrine should not be used since HALDOL haloperidol may block its vasopressor activity and paradoxical further lowering of blood pressure may occur); (2) receiving anticonvulsant medication since HALDOL haloperidol may lower the convulsive threshold; (3) with known allergies or a history of allergic reactions to drugs; (4) receiving anticoagulants. Concomitant antiparkinson medication, if required, may have to be continued after HALDOL haloperidol is discontinued because of different excretion rates; if both are discontinued simultaneously, extrapyramidal symptoms may occur. Intraocular pressure may increase when anticholinergic drugs, including antiparkinson drugs, are administered concomitantly with HALDOL haloperidol. When HALDOL haloperidol is used for mania in cyclic disorders, there may be a rapid mood swing to depression. Severe neurotoxicity may occur in patients with thyrotoxicosis receiving antipsychotic medication, including HALDOL haloperidol. FD&C Yellow No. 5 (tartrazine) may cause allergic-type reactions (including bronchial asthma) in certain susceptible individuals, especially in those who have aspirin hypersensitivity.

Adverse Reactions: ***CNS Effects:*** *Extrapyramidal Reactions:* Neuromuscular (extrapyramidal) reactions have been reported frequently, often during the first few days of treatment. Generally they involved Parkinson-like symptoms which were usually mild to moderately severe and usually reversible. Other types of neuromuscular reactions (motor restlessness, dystonia, akathisia, hyperreflexia, opisthotonos, oculogyric crises) have been reported far less frequently, but were often more severe. Severe extrapyramidal reactions have been reported at relatively low doses. Generally, extrapyramidal symptoms are dose-related since they occur at relatively high doses and disappear or become less severe when the dose is reduced. Antiparkinson drugs may be required. Persistent extrapyramidal reactions have been reported and the drug may have to be discontinued in such cases.

Withdrawal Emergent Neurological Signs: Abrupt discontinuation of short term antipsychotic therapy is generally uneventful. However, some patients o maintenance treatment experience transient dyskinetic signs after abrup withdrawal. In certain cases these are indistinguishable from "Persistent Ta dive Dyskinesia" except for duration. It is unknown whether gradual with drawal will reduce the occurrence of these signs, but until further evidence available haloperidol should be gradually withdrawn.

Persistent Tardive Dyskinesia: Although rarely reported with HALDOL halo peridol, tardive dyskinesia may appear during or after long-term therapy. Th risk appears to be greater in elderly patients on high-dose therapy, especial females. Symptoms are persistent and sometimes appear irreversible; there no known effective treatment and all antipsychotic agents should be discon tinued. The syndrome may be masked by reinstitution of drug, increasin dosage, or switching to a different antipsychotic agent.

Other CNS Effects: Insomnia, restlessness, anxiety, euphoria, agitation drowsiness, depression, lethargy, headache, confusion, vertigo, grand m seizures, and exacerbation of psychotic symptoms.

Cardiovascular Effects: Tachycardia and hypotension. ***Hematologi Effects:*** Reports of mild, usually transient leukopenia and leukocytosis, min mal decreases in red blood cell counts, anemia, or a tendency toward lyn phomonocytosis; agranulocytosis rarely reported and only in association wit other medication. ***Liver Effects:*** Impaired liver function and/or jaundic reported. ***Dermatologic Reactions:*** Maculopapular and acneiform reaction isolated cases of photosensitivity, loss of hair. ***Endocrine Disorders:*** Lact tion, breast engorgement, mastalgia, menstrual irregularities, gynecomasti impotence, increased libido, hyperglycemia and hypoglycemia. ***Gastrointe tinal Effects:*** Anorexia, constipation, diarrhea, hypersalivation, dyspepsi nausea and vomiting. ***Autonomic Reactions:*** Dry mouth, blurred vision, u nary retention and diaphoresis. ***Respiratory Effects:*** Laryngospasm, bro chospasm and increased depth of respiration.

The injectable form is intended only for acutely agitated psychotic patien with moderately severe to very severe symptoms.

Caution: Federal law prohibits dispensing without prescription. 08

IMPORTANT: Full directions for use should be read before HALDO haloperidol is administered or prescribed.

HALDOL tablets and concentrate (120 ml) are manufactured by McN Laboratories Co., Dorado, PR 00646. ©McN 198

McNEIL McNeil Laboratories, McNEILAB, INC.
Fort Washington, PA 19034

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SEPTEMBER 1980

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When depressive symptoms come in a cluster of blues...:

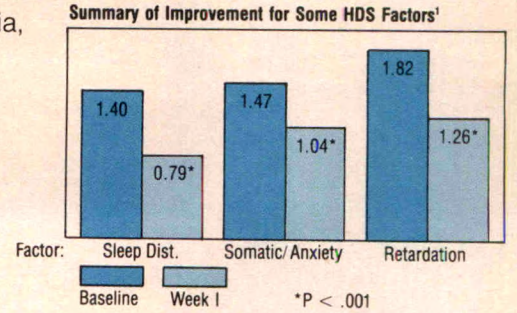


Adapin® (doxepin HCl) helps patients see life in all its colors

Improvements noted at one week

Depressed patients often present with fatigue, anorexia, anxiety, mood swings, loss of interest, *and most frequently, early awakening and insomnia*. In a summation of 9 clinical studies involving 198 patients, significant clinical improvement was noted after the first week of doxepin treatment.[1] However, optimal antidepressant response may not be evident for two to three weeks.

Summary of Improvement for Some HDS Factors[1]



Normalized sleep patterns

In a study of clinically depressed patients with symptoms of insomnia, treatment with doxepin significantly reduced difficulty falling asleep, difficulty staying asleep and frequency of early morning awakening.[2]

Low potential for cardiotoxicity

The incidence of tachycardia and hypotension, which have been observed during treatment with tricyclic antidepressants, has been reported infrequently with doxepin. In 452 patients given doxepin, the incidence of tachycardia was 2.88%; in 495 patients, the incidence of hypotension was 2.62%.[3]

Now available... 75 mg capsules for h.s. dosage

Brief Summary of Prescribing Information ADAPIN® (doxepin HCl) Capsules
Indications–Relief of symptoms of anxiety and depression.
Contraindications–Glaucoma, tendency toward urinary retention or hypersensitivity to doxepin.
Warnings–Adapin has not been evaluated for safety in pregnancy. No evidence of harm to the animal fetus has been shown in reproductive studies. There are no data concerning secretion in human milk, nor on effect in nursing infants.

Usage in children under 12 years of age is not recommended. MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug, as serious side-effects and death have been reported with the concomitant use of certain drugs and MAO inhibitors.

In patients who may use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage.

Precautions–Drowsiness may occur and patients should be cautioned against driving a motor vehicle or operating hazardous machinery. Since suicide is an inherent risk in depressed patients they should be closely supervised while receiving treatment. Although Adapin has shown effective tranquilizing activity, the possibility of activating or unmasking latent psychotic symptoms should be kept in mind. This product contains FD&C Yellow No. 5 (tartrazine) which may cause allergic-type reactions (including bronchial asthma) in certain susceptible individuals. Although the overall incidence of FD&C Yellow No. 5 (tartrazine) sensitivity in the general population is low, it is frequently seen in patients who also have aspirin hypersensitivity.

Adverse Reactions–Dry mouth, blurred vision and constipation have been reported. Drowsiness has also been observed.

Adverse effects occurring infrequently include extrapyramidal symptoms, gastrointestinal reactions, secretory effects such as sweating, tachycardia and hypotension. Weakness, dizziness, fatigue, weight gain, edema, paresthesias, flushing, chills, tinnitus, photophobia, decreased libido, rash and pruritus may also occur.

Dosage and Administration–In mild to moderate anxiety and/or depression: 25 mg t.i.d. Increase or decrease the dosage according to individual response. Daily dosage, up to 150 mg may be taken at bedtime without loss of effectiveness. Usual optimum daily dosage is 75 mg to 150 mg per day not to exceed 300 mg per day.

Antianxiety effect usually precedes the antidepressant effect by two or three weeks.

How Supplied–Each capsule contains doxepin, as the hydrochloride. 10 mg, 25 mg, 50 mg, 75 mg and 100 mg capsules in bottles of 100 and 1000.

For complete prescribing information please see package insert or PDR.

References:

1. Barranco SF, Thrash ML, Hackett E, et al: Early onset of response to doxepin treatment. *J Clin Psychiatry* 40:265-269, 1979 (Sinequan®).
2. Karacan I, Blackburn AB, Thornby JI: The Effect of Doxepin HCl (Sinequan®) on Sleep Patterns and Clinical Symptomatology of Neurotic Depressed Patients with Sleep Disturbance, in Mendels J (ed): *Sinequan®: A monograph of recent clinical studies*, Princeton, NJ, Excerpta Medica, 1977, pp 4-22.
3. Pitts NE: The clinical evaluation of doxepin–A new psychotherapeutic agent. *Psychosomatics* 10:164-171, 1969.

Sinequan® brand of doxepin HCl was the drug used in studies referenced as 1, 2 and 3.

Rx DIVISION
PENNWALT
ROCHESTER, NEW YORK 14623

Adapin® (doxepin HCl) ... effective single-entity drug with antidepressant and antianxiety effects

A-252

CONVENIENCE, COMPLIANCE



One prescription to write, one prescription for the patient to have filled and usually only one tablet per dose to take. Two dosage strengths simplify titration.

Less trouble with treatment dropouts – early relief encourages patients to stay with their regimen. In a nationwide, multicenter study there were fewer dropouts due to side effects or treatment failure with Limbitrol than with amitriptyline alone.*

More than an antidepressant, without a phenothiazine – for *nonpsychotic* anxious depression. Limbitrol combines the antidepressant action of amitriptyline with the *specifically effective* antianxiety action of Librium® (chlordiazepoxide HCl/Roche), for efficacy while minimizing phenothiazine risks.†

Broad symptomatic relief – symptoms likely to respond in the first week of treatment include insomnia, agitation, psychic and somatic anxiety, anorexia and feelings of guilt or worthlessness.

*Data on file, Medical Department, Hoffmann-La Roche Inc.

†The causal relationship between the phenothiazines and extrapyramidal side effects, including tardive dyskinesia, is well established. In contrast the reported incidence of these adverse reactions with Limbitrol or either of its components is rare. (For a complete list of side effects reported with Limbitrol, consult full disclosure.)

ROCHE®

A CLEAR-CUT RATIONALE AND A COMFORTABLE PATIENT



Fundamental reasons to start prescribing

Limbitrol® IV

Tablets 5-12.5 each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)
Tablets 10-25 each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt)



for moderate depression and anxiety

Please see following page for a summary of product information.

LIMBITROL® TABLETS Tranquilizer-Antidepressant

Before prescribing, please consult complete product information, a summary of which follows:
Indications: Relief of moderate to severe depression associated with moderate to severe anxiety.
Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants. Do not use with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Contraindicated during acute recovery phase following myocardial infarction.
Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs. Closely supervise cardiovascular patients. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses. Myocardial infarction and stroke reported with use of this class of drugs.)
***Usage in Pregnancy:* Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage; withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline; symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).
Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Periodic liver function tests and blood counts are recommended during prolonged treatment. Amitriptyline component may block action of guanethidine or similar antihypertensives. Concomitant use with other psychotropic drugs has not been evaluated: sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Limbitrol should not be taken during the nursing period. Not recommended in children under 12.
In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects.
Adverse Reactions: Most frequently reported are those associated with either component alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline. Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely.
This list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs:
Cardiovascular: Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke.
Psychiatric: Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.
Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns.
Anticholinergic: Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract.
Allergic: Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus.
Hematologic: Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia.
Gastrointestinal: Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.
Endocrine: Testicular swelling and gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female and elevation and lowering of blood sugar levels.
Other: Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.
Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. I.V. administration of 1 to 3 mg physostigmine salicylate has been reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.
Dosage: Individualize according to symptom severity and patient response. Reduce to smallest effective dosage when satisfactory response is obtained. Larger portion of daily dose may be taken at bedtime. Single h.s. dose may suffice for some patients. Lower dosages are recommended for the elderly.
Limbitrol 10-25, initial dosage of three to four tablets daily in divided doses, increased up to six tablets or decreased to two tablets daily as required. Limbitrol 5-12.5, initial dosage of three to four tablets daily in divided doses, for patients who do not tolerate higher doses.
How Supplied: White, film-coated tablets, each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) and blue, film-coated tablets, each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt) — bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50.

ROCHE®
ROCHE PRODUCTS INC.
Manati, Puerto Rico 00701

Your guide to patient management... when you decide medication is needed

How to initiate and maintain therapy

Select dosage strength appropriate for each patient
☐ Limbitrol 5-12.5 is recommended to minimize drowsiness and for elderly patients
☐ Limbitrol 10-25 may be indicated for patients who tolerate medication without undue side effects

Specify daily dosage based on symptom severity
☐ An initial dosage of three tablets is recommended
☐ Dosage may be increased to six tablets or decreased to two tablets daily as necessary
☐ Once a satisfactory response is obtained, patients should be continued on the smallest dose required to maintain remission

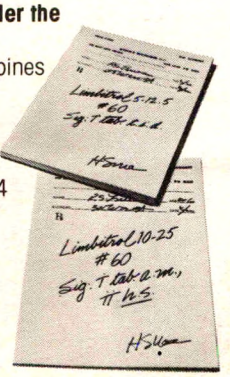
Utilize dosage options to best accommodate individual patient needs
☐ T.I.D. or Q.I.D., familiar regimens most suited for patients who tolerate medication without undue drowsiness
☐ Two tablets one hour before bedtime and one tablet midday may minimize daytime drowsiness and help relieve a common target symptom—insomnia
☐ Entire dosage h.s. to take maximum advantage of the sedative effect

How to make each patient an informed patient

1. Discuss with patients the probability that they will experience drowsiness, especially during the first week.
2. Reassure your patients that drowsiness is one indication that the medication is working and that it may help alleviate their insomnia.
3. Encourage patients to report if drowsiness becomes troublesome so that, if necessary, dosage schedule can be adjusted.
4. Caution patients about the combined effects with alcohol or other CNS depressants. Let them know that the additive effects may produce a harmful level of sedation and CNS depression.
5. Caution patients about activities requiring complete mental alertness, such as operating machinery or driving a car.
6. Warn pregnant patients and patients of childbearing age that the safety of Limbitrol in pregnancy has not yet been established.

Please see complete product disclosure for other pertinent information.

Limbitrol should not be used under the following circumstances:
1. Hypersensitivity to benzodiazepines or tricyclic antidepressants.
2. Concomitantly with an MAO inhibitor. To replace an MAO inhibitor with Limbitrol, discontinue MAO inhibitor for a minimum of 14 days before cautiously initiating Limbitrol therapy.
3. During the acute recovery phase following myocardial infarction.



In moderate depression and anxiety

Limbitrol®

Relief without a phenothiazine

CLORAZEPATE DIPOTASSIUM

Tranxene® CT

Ideal for short-term antianxiety therapy



Acts promptly...tapers gently at discontinuation

Tranxene goes to work in minutes

Nordiazepam uptake is rapid, with pharmacologic effects reported within 30 minutes.[1] Peak serum levels are attained within 1-2 hours after dosing.

Allows post-therapy adjustment time

When administration stops, nordiazepam serum levels taper at a gentle rate. This built-in taper may give your patients more time to adjust to cessation of therapy.[2]

1. Sedation reported in normal volunteers 30 minutes after dosing. Tranxene Drug Monograph 97-0185, p. 9, 1979.
2. The elimination kinetics of an agent can be closely defined, but these cannot at present be directly related to therapeutic or adverse effects.

Abbott Laboratories, North Chicago, IL 60064

0043331

Brief Summary

INDICATIONS — Symptomatic relief of anxiety and tension associated with anxiety disorders, other psychoneurotic disorders, transient situational disturbances, and functional or organic disorders. Symptomatic relief of acute alcohol withdrawal.

Effectiveness in long-term use (over 4 months) not assessed by systematic clinical studies. Physician should periodically reassess usefulness for each patient.

CONTRAINDICATIONS — Known hypersensitivity to the drug. Acute narrow angle glaucoma.

WARNINGS — Not for use in depressive neuroses or psychotic reactions. Caution patients against hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles. Advise against simultaneous use of other CNS depressants, and caution patients that effects of alcohol may be increased. Not recommended for patients under 18. Nervousness, insomnia, irritability, diarrhea, muscle aches, and memory impairment have followed abrupt withdrawal from long-term high dosage. Withdrawal symptoms were reported after abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months. Use caution in patients having psychological potential for drug dependence (dependence has been observed in dogs and rabbits).

Pregnancy and Lactation: Minor tranquilizers should almost always be avoided first trimester. Consider possibility of pregnancy before initiating therapy. Patient should consult physician about discontinuation if she becomes pregnant or plans pregnancy. Do not give to nursing mothers.

PRECAUTIONS — Observe usual precautions in depression accompanying anxiety, or in patients with suicidal tendency, or those with impaired renal or hepatic function. Do periodic blood counts and liver function tests during prolonged therapy. Use small doses and gradual increments in the elderly or debilitated.

ADVERSE REACTIONS — Drowsiness, dizziness, various g.i. complaints, nervousness, blurred vision, dry mouth, headache, mental confusion, insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression, slurred speech, abnormal liver and kidney function, decreased hematocrit, decreased systolic blood pressure.

DOSAGE — ANXIETY—Usual daily dose 30 mg or less (start the elderly or debilitated at 7.5-15 mg). Adjust gradually within 15-60 mg daily range. Capsules and scored tablets: divided doses; or once daily h.s. (start patient at 15 mg). Single Dose Tablets, 22.5 mg (for patients stabilized on 7.5 mg t.i.d.) or 11.25 mg: once daily at any hour. ALCOHOL WITHDRAWAL—In divided doses: 1st day 30 mg initially, then 30-60 mg; 2nd day 45-90 mg; 3rd day 22.5-45 mg; 4th day 15-30 mg. Then taper to 15-7.5 mg daily, and discontinue as soon as stable.

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*Sedation reported in normal volunteers 30 minutes after dosing; Tranxene Drug Monograph, 97-0185, p. 9, 1979. See overleaf for brief summary.

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THE AMERICAN JOURNAL OF PSYCHIATRY

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By Mardi J. Horowitz, Nancy Wilner, Charles Marmar, and Janice Krupnick

Tardive Dyskinesia: Summary of a Task Force Report of the American Psychiatric Association

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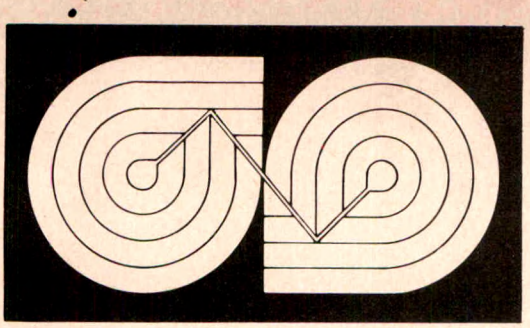
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Manuscripts that report the results of experimental investigation with human subjects must include a statement that informed consent was obtained after the procedure(s) had been fully explained.

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Papers are reviewed by at least two experts to determine the originality, validity, and significance of content and conclusions. Authors will usually be advised within 3–4 months of the decision on their paper, although delays are sometimes unavoidable. Reviewers' comments will be returned with rejected manuscripts if they are judged to be useful to the authors. All reviewers remain anonymous.

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Overview articles attempt to bring together important relevant information on a topic of general interest to psychiatry. They are usually written at the invitation of the Editor; authors who have ideas for overview articles are advised to check with the Editor to ensure that a similar work is not in preparation. Overviews should not exceed 7,500 words **(no more than 25 double-spaced pages—including tables and figures—and 100 references).** All overviews, including those written by invitation, are given the same peer review received by other papers.

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The précis should be up to 100 words for regular articles and Brief Communications. (No précis is used in the Clinical and Research Reports section.) The précis should be a single paragraph using complete, connected sentences, active verbs, and the third person. In most cases, the précis replaces a summary.

Text

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1. Berne E: Principles of Group Treatment. New York, Oxford University Press, 1966, p 26
2. Blackwell B, Marley E, Price J, et al: Hypertensive interactions between monoamine oxidase inhibitors and foodstuffs. Br J Psychiatry 113:349–365, 1967
3. Jones AB: Long-term administration of tricyclic antidepressants: three-year follow-up. Am J Psychiatry (in press)
4. Smythe JCW (ed): Psychiatry and Human Values. Springfield, Ill, Charles C Thomas, 1971
5. Brosin H: Communication systems of the consultation process, in The Psychiatric Consultation. Edited by Mendel W, Solomon P. New York, Grune & Stratton, 1968

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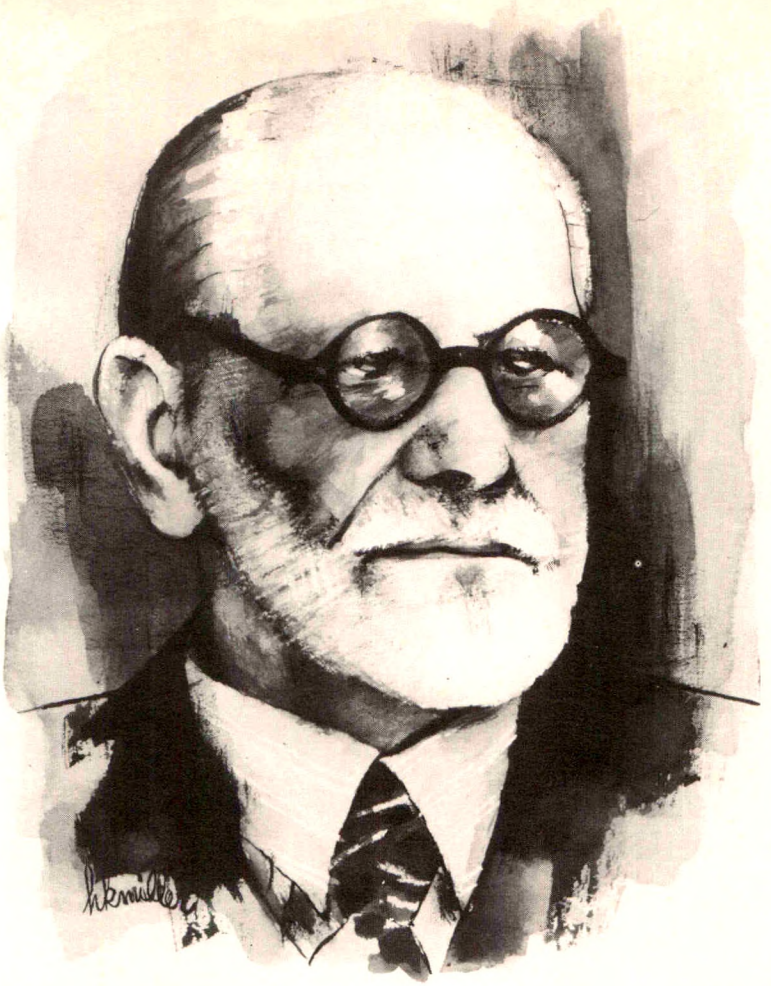
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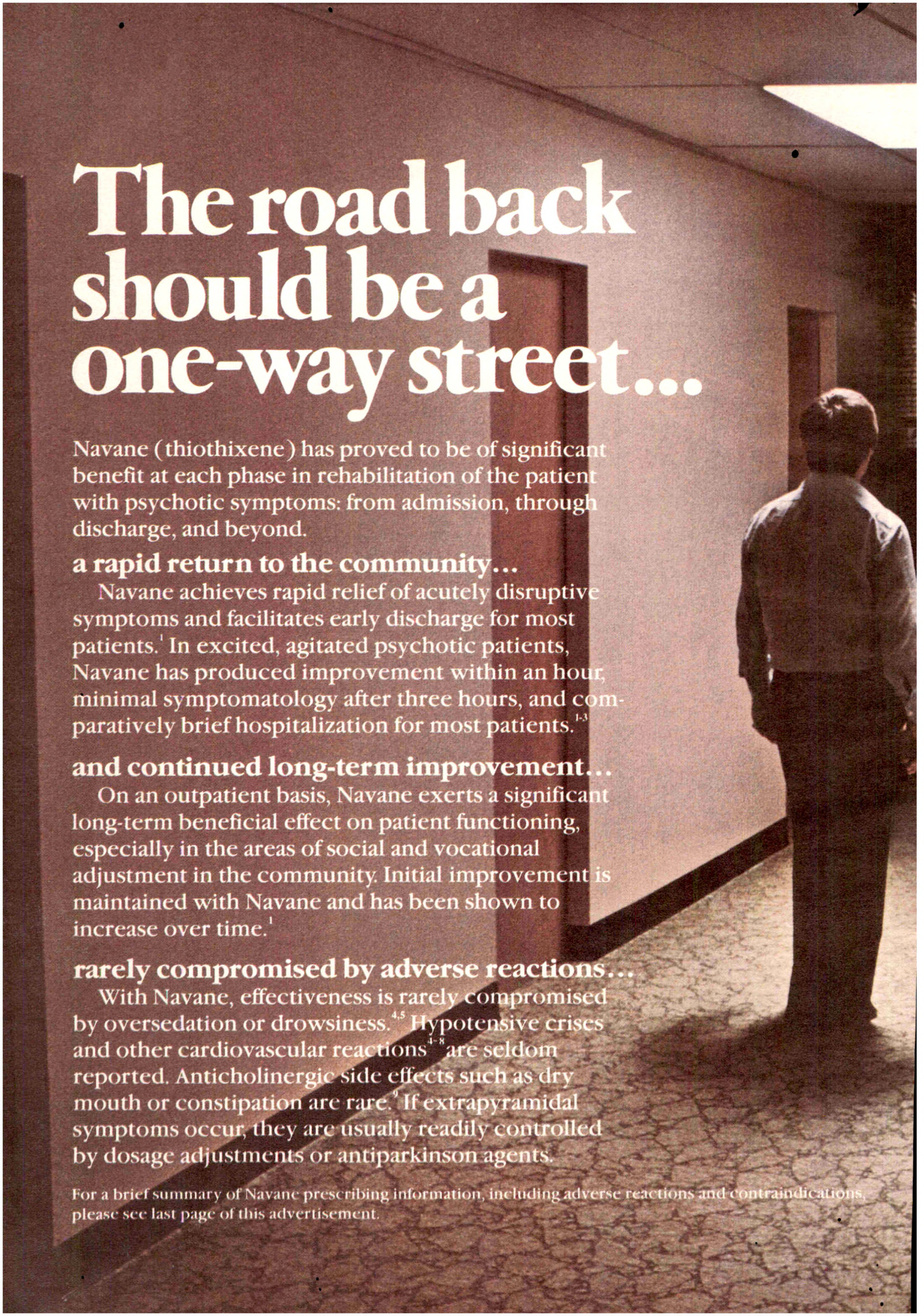
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The road back should be a one-way street...

Navane (thiothixene) has proved to be of significant benefit at each phase in rehabilitation of the patient with psychotic symptoms: from admission, through discharge, and beyond.

a rapid return to the community...

Navane achieves rapid relief of acutely disruptive symptoms and facilitates early discharge for most patients.[1] In excited, agitated psychotic patients, Navane has produced improvement within an hour, minimal symptomatology after three hours, and comparatively brief hospitalization for most patients.[1-3]

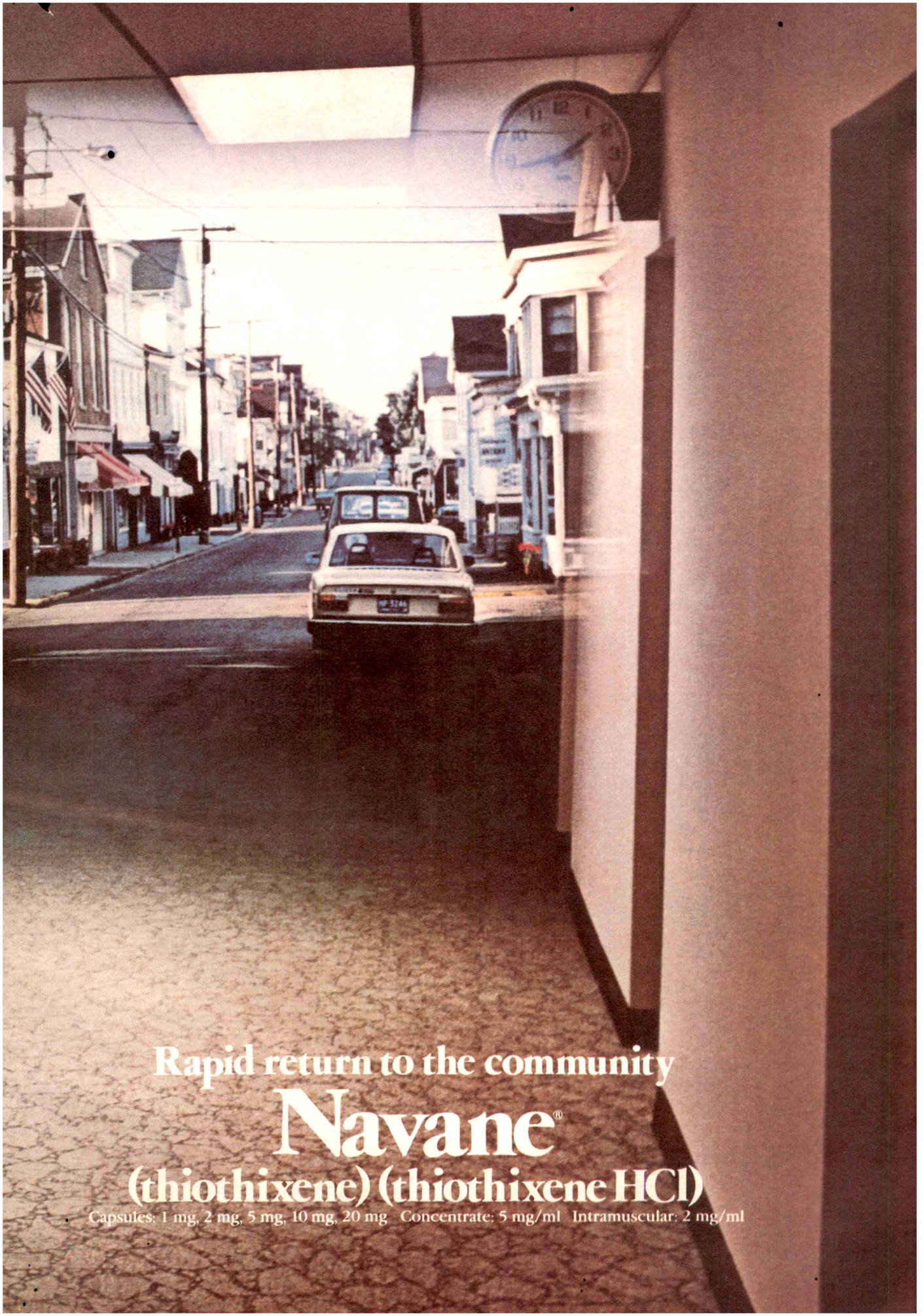
and continued long-term improvement...

On an outpatient basis, Navane exerts a significant long-term beneficial effect on patient functioning, especially in the areas of social and vocational adjustment in the community. Initial improvement is maintained with Navane and has been shown to increase over time.[1]

rarely compromised by adverse reactions...

With Navane, effectiveness is rarely compromised by oversedation or drowsiness.[4,5] Hypotensive crises and other cardiovascular reactions[4-8] are seldom reported. Anticholinergic side effects such as dry mouth or constipation are rare.[9] If extrapyramidal symptoms occur, they are usually readily controlled by dosage adjustments or antiparkinson agents.

For a brief summary of Navane prescribing information, including adverse reactions and contraindications, please see last page of this advertisement.



Rapid return to the community

Navane®

(thiothixene) (thiothixene HCl)

Capsules: 1 mg, 2 mg, 5 mg, 10 mg, 20 mg Concentrate: 5 mg/ml Intramuscular: 2 mg/ml

Rapid return to the community

Navane® (thiothixene) (thiothixene hydrochloride)

Capsules: 1 mg, 2 mg, 5 mg, 10 mg, 20 mg Concentrate: 5 mg/ml Intramuscular: 2 mg/ml

BRIEF SUMMARY OF PRESCRIBING INFORMATION
Navane® (thiothixene)
Capsules: 1 mg, 2 mg, 5 mg, 10 mg, 20 mg (thiothixene hydrochloride) Concentrate: 5 mg/ml, Intramuscular: 2 mg/ml

Contraindications. Navane (thiothixene) is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy*– Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration of Navane to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children– The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration– As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane (thiothixene). Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudo-parkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leukocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution – For Intramuscular Use Only. Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane (thiothixene) Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate– In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Stimulants that may cause convulsions (e.g. picrotoxin or pentylenetetrazol) should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg of thiothixene in bottles of 100, 1,000, and unit-dose pack of 100 (10 x 10's). Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100, 500, and unit-dose pack of 100 (10 x 10's).

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg, and in 30 ml (1 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, and 5 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10 vials. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

References: 1. Rickels K, Norstad N, Downing R: The acutely decompensated schizophrenic patient, presented as a scientific exhibit at the American Psychiatric Association 28th Institute on Hospital and Community Psychiatry, September 20-22, 1976, Atlanta, Ga. 2. Stotsky BA: Relative efficacy of parenteral haloperidol and thiothixene for the emergency treatment of acutely excited and agitated patients. *Dis Nerv Syst* 38:967-973, Dec 1977. 3. Sloan RB, Razani J, Maloney MP, et al: Premorbid adjustment, cognition and outcome in schizophrenia, presented as a scientific exhibit at the 128th Annual Meeting of the American Psychiatric Association, May 5-9, 1975, Anaheim, California. 4. Engelhardt DM, Rudorfer L, Rosen B: Haloperidol and thiothixene in the long-term treatment of chronic schizophrenic outpatients in an urban community: Social and vocational adjustment. *J Clin Psychiatry* 39:834-840, Dec 1978. 5. Itil TM, Unverdi C, Wohlrabe J, et al: Drug therapy of psychosis associated with organic brain syndrome, presented as a scientific exhibit at the American Public Health Association Centennial, Atlantic City, New Jersey, Nov 12-16, 1972. 6. Brauzer B, Goldstein BJ: Comparative effects of intramuscular thiothixene and trifluoperazine in psychotic patients: *J Clin Pharmacol* 8:400-403, Nov-Dec 1968. 7. Dillenkoffer RL, Gallant DM, George RB, et al: Electrocardiographic evaluations of schizophrenic patients, presented as a scientific exhibit at the 125th Annual Meeting of the American Psychiatric Association, Dallas, Texas, May 1-4, 1972. 8. Data on file at Roerig. 9. Goldstein B, Weiner D, Banas F: Clinical evaluation of thiothixene in chronic ambulatory schizophrenic patients, in Lehmann HE, Ban TA (eds): *Modern Problems in Pharmacopsychiatry.* Basel, S Karger, 1969, vol 2, pp 45-52.

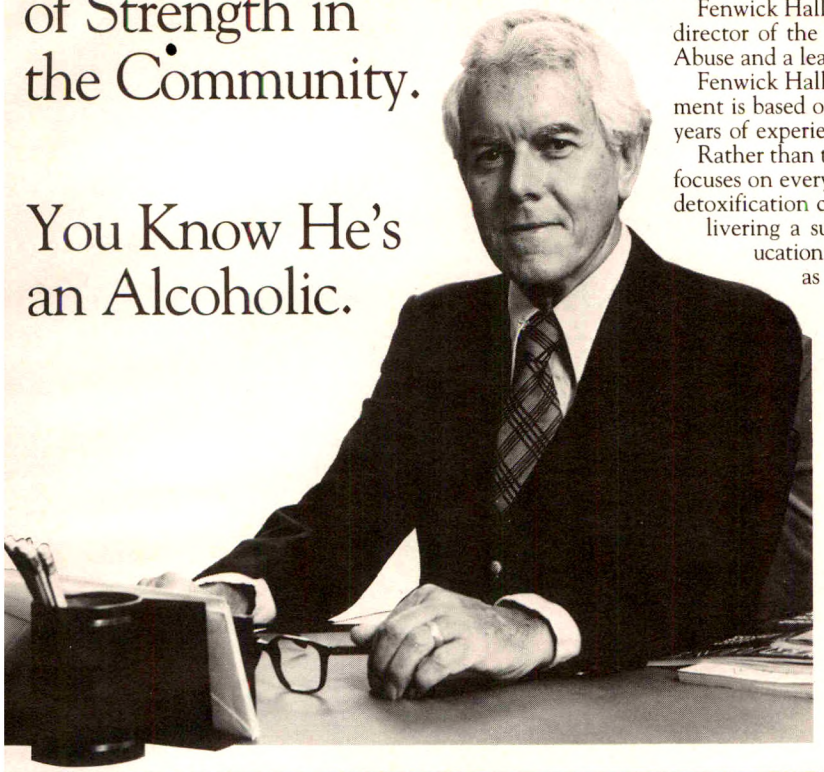
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†The causal relationship between the phenothiazines and extrapyramidal side effects, including tardive dyskinesia, is well established. In contrast the reported incidence of these adverse reactions with Limbitrol or either of its components is rare. (For a complete list of side effects reported with Limbitrol, consult full disclosure.)

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for moderate depression and anxiety

Please see following page for a summary of product information.

LIMBITROL® TABLETS Tranquilizer-Antidepressant

Before prescribing, please consult complete product information, a summary of which follows:
Indications: Relief of moderate to severe depression associated with moderate to severe anxiety.
Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants. Do not use with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Contraindicated during acute recovery phase following myocardial infarction.
Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs. Closely supervise cardiovascular patients. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses. Myocardial infarction and stroke reported with use of this class of drugs.)

***Usage in Pregnancy:* Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage; withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline; symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).
Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Periodic liver function tests and blood counts are recommended during prolonged treatment. Amitriptyline component may block action of guanethidine or similar antihypertensives. Concomitant use with other psychotropic drugs has not been evaluated: sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Limbitrol should not be taken during the nursing period. Not recommended in children under 12.
In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects.
Adverse Reactions: Most frequently reported are those associated with either component alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline. Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely.
This list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs:
Cardiovascular: Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke.
Psychiatric: Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.
Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns.
Anticholinergic: Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract.
Allergic: Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus.
Hematologic: Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia.
Gastrointestinal: Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.
Endocrine: Testicular swelling and gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female and elevation and lowering of blood sugar levels.
Other: Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.
Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. I.V. administration of 1 to 3 mg physostigmine salicylate has been reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.
Dosage: Individualize according to symptom severity and patient response. Reduce to smallest effective dosage when satisfactory response is obtained. Larger portion of daily dose may be taken at bedtime. Single h.s. dose may suffice for some patients. Lower dosages are recommended for the elderly.
Limbitrol 10-25, initial dosage of three to four tablets daily in divided doses, increased up to six tablets or decreased to two tablets daily as required. Limbitrol 5-12.5, initial dosage of three to four tablets daily in divided doses, for patients who do not tolerate higher doses.
How Supplied: White, film-coated tablets, each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) and blue, film-coated tablets, each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50.

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How to initiate and maintain therapy

Select dosage strength appropriate for each patient
☐ Limbitrol 5-12.5 is recommended to minimize drowsiness and for elderly patients
☐ Limbitrol 10-25 may be indicated for patients who tolerate medication without undue side effects

Specify daily dosage based on symptom severity
☐ An initial dosage of three tablets is recommended
☐ Dosage may be increased to six tablets or decreased to two tablets daily as necessary
☐ Once a satisfactory response is obtained, patients should be continued on the smallest dose required to maintain remission

Utilize dosage options to best accommodate individual patient needs
☐ T.I.D. or Q.I.D., familiar regimens most suited for patients who tolerate medication without undue drowsiness
☐ Two tablets one hour before bedtime and one tablet midday may minimize daytime drowsiness and help relieve a common target symptom—insomnia
☐ Entire dosage h.s. to take maximum advantage of the sedative effect

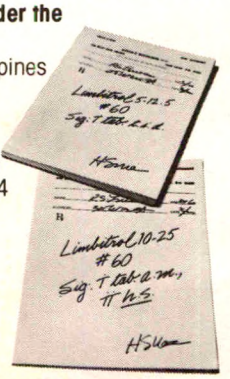
Your guide to patient management... when you decide medication is needed

How to make each patient an informed patient

1. Discuss with patients the probability that they will experience drowsiness, especially during the first week.
2. Reassure your patients that drowsiness is one indication that the medication is working and that it may help alleviate their insomnia.
3. Encourage patients to report if drowsiness becomes troublesome so that, if necessary, dosage schedule can be adjusted.
4. Caution patients about the combined effects with alcohol or other CNS depressants. Let them know that the additive effects may produce a harmful level of sedation and CNS depression.
5. Caution patients about activities requiring complete mental alertness, such as operating machinery or driving a car.
6. Warn pregnant patients and patients of childbearing age that the safety of Limbitrol in pregnancy has not yet been established.

Please see complete product disclosure for other pertinent information.

Limbitrol should not be used under the following circumstances:
1. Hypersensitivity to benzodiazepines or tricyclic antidepressants.
2. Concomitantly with an MAO inhibitor. To replace an MAO inhibitor with Limbitrol, discontinue MAO inhibitor for a minimum of 14 days before cautiously initiating Limbitrol therapy.
3. During the acute recovery phase following myocardial infarction.



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Brief Summary of Prescribing Information

Indications and Usage: Symptomatic relief of anxiety, tension, agitation, irritability ar insomnia associated with anxiety neuroses and transient situational disturbances; anxie associated with depressive symptoms and as a treatment of symptoms of anxiety if such sym toms are a significant feature of functional or organic disorders, particularly gastrointestinal cardiovascular.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by syster atic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CN acting drugs, warn patients on lorazepam not to operate machinery or motor vehicles, and diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with bart turates and alcohol have occurred following abrupt discontinuance of benzodiazepin (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addi tion-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillanc when on benzodiazepines because of their predisposition to habituation and dependenc Withdrawal symptoms have also been reported following abrupt discontinuance of benzod azepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid ov sedation.

Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occ sional convulsions.

Observe usual precautions with impaired renal or hepatic function.

Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepa has not been shown of significant benefit in treating gastrointestinal or cardiovascular comp nent.

Esophageal dilation occurred in rats treated with lorazepam for more than 1 year 6mg/kg/day. No effect dose was 1.25mg/kg/day (approximately 6 times the maximum huma therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn with 2 months of first observation. Clinical significance is unknown; but use of lorazepam for pr longed periods and in geriatric patients requires caution and frequent monitoring for symptor of upper G.I. disease.

Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have h elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tes are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressa effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbi Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschis malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship dosage. Although all these anomalies were not present in the concurrent control group, th have been reported to occur randomly in historical controls. At 40mg/kg and higher, there w evidence of fetal resorption and increased fetal loss in rabbits which was not seen at low doses. Clinical significance of these findings is not known. However, increased risk of conge tal malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam a meprobamate) during first trimester of pregnancy has been suggested in several studi Because use of these drugs is rarely a matter of urgency, use of lorazepam during this peri should almost always be avoided. Possibility that a woman of child-bearing potential may pregnant at institution of therapy should be considered. Advise patients if they become pre nant to communicate with their physician about desirability of discontinuing the drug.

In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepa and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like oth benzodiazepines. As a general rule, nursing should not be undertaken while on a drug sin many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and genera disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxio patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9% weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, na sea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, e function disturbance, various gastrointestinal symptoms and autonomic manifestations. In dence of sedation and unsteadiness increased with age. Small decreases in blood pressu have been noted but are not clinically significant, probably being related to relief of anxiety.

Overdosage: In management of overdosage with any drug, bear in mind that multiple age may have been taken. Manifestations of overdosage include somnolence, confusion and con Induce vomiting and/or undertake gastric lavage followed by general supportive care, monit ing of vital signs and close observation. Hypotension, though unlikely, usually may be control with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

Ativan® IV for Anxiety (lorazepam)

Dosage: **Individualize for maximum beneficial effects. Increase do gradually when needed, giving higher evening dose before increasi daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosa may vary from 1 to 10mg/day in divided doses. For elderly or deb tated, initially 1-2mg/day; insomnia due to anxiety or transient sit tional stress, 2-4mg h.s.**

How Supplied: **0.5, 1.0 and 2.0mg tablets.**

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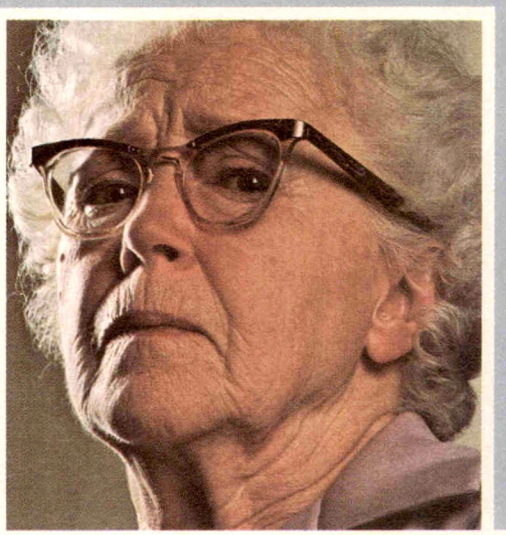
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An age for anxiety.

The aging process all too frequently is accompanied by anxiety states. When anxious older patients do not respond to your sympathetic counseling, a benzodiazepine may be the next logical step.

But which one? Here, more than in many other patient groups, the metabolites of the available compounds may make a decisive difference. Unlike diazepam, clorazepate and chlordiazepoxide, Ativan (lorazepam) has no long-acting metabolites, and its simple metabolism is less readily impaired with advancing age. Steady-state serum levels are rapidly achieved and disappear rapidly once you decide to discontinue therapy. For these reasons, residual effects are not likely to present problems. (The pharmacokinetic profile of a drug can define such characteristics as its absorption, distribution, metabolism and elimination but cannot, at present, be directly related to its therapeutic effectiveness.)

Proved effective in many nationwide, double-blind studies (including patients in older age groups), Ativan is compatible with a wide range of commonly prescribed drugs. (The benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.) Patients who have trouble swallowing solid medication appreciate the small Ativan tablet, which is tasteless and disintegrates within seconds in water or fruit juice.



See important information on preceding page.

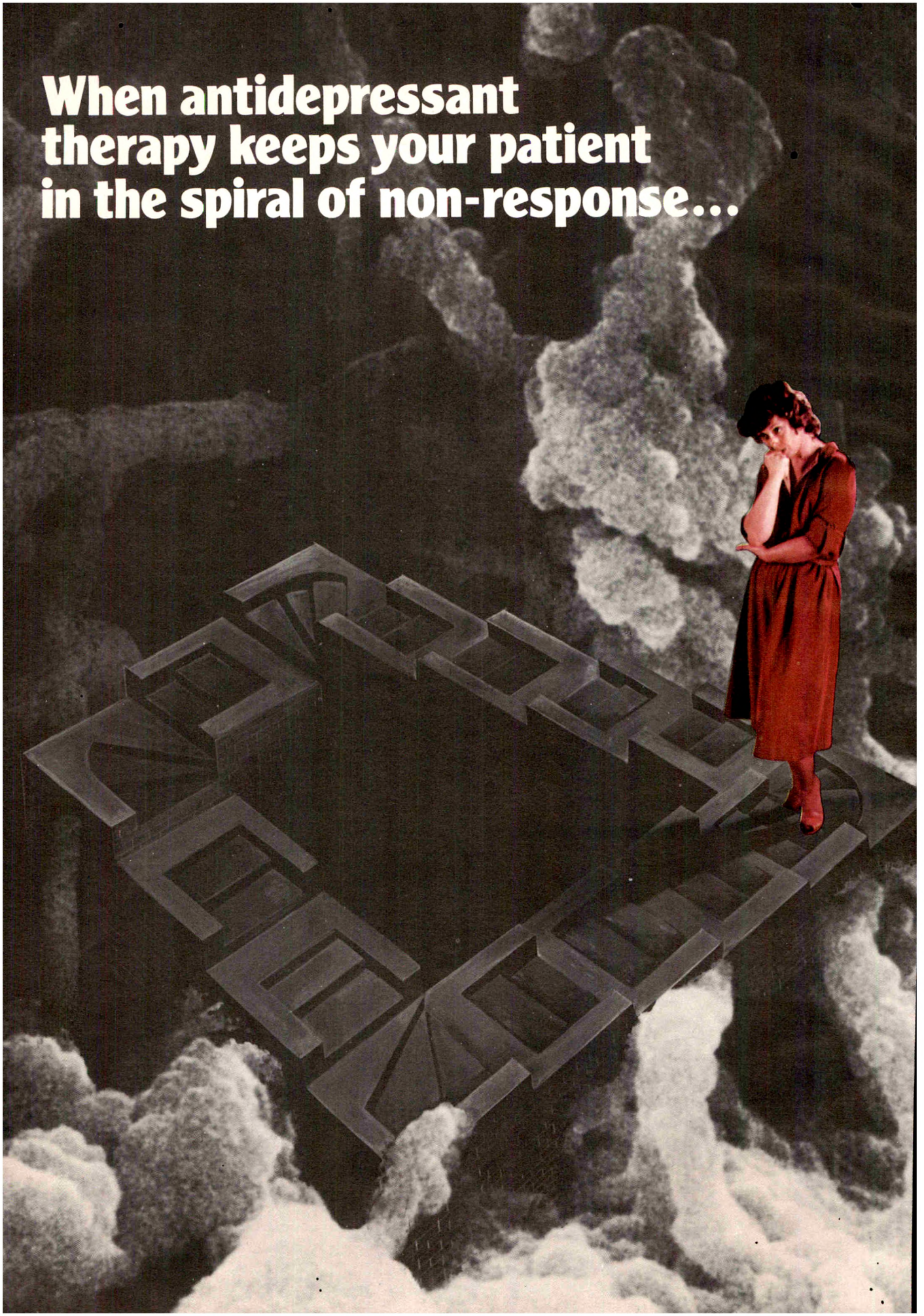
Ativan® (lorazepam)

0.5 mg tablet



for Anxiety

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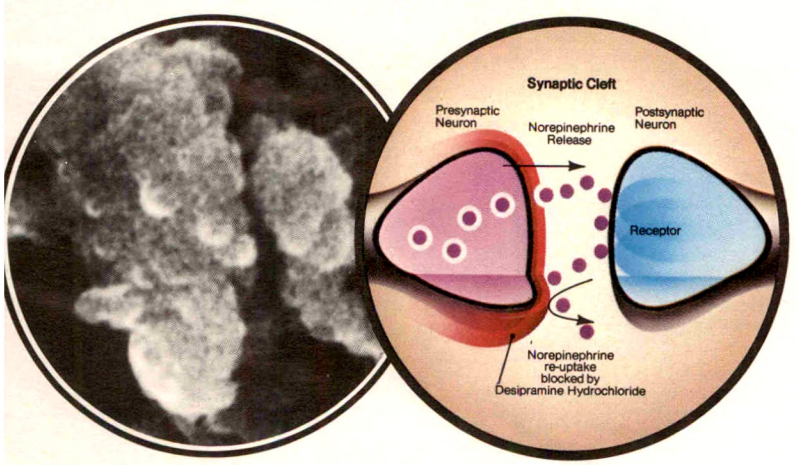
Helps the patient remain calm but active

- Begins to improve sleep patterns within one week in some patients.[1]
- As the depression is relieved, difficulty in falling asleep, restlessness, and early morning awakening diminish.
- Norpramin does not usually inhibit normal activity, although patients should be cautioned against driving or operating machinery if drowsiness occurs. (See Warnings, Precautions, and Adverse Reactions in Brief Summary overleaf.)

Less anticholinergic activity

- Studies of animal tissues[2] and normal human subjects[3,4] have shown that desipramine has less anticholinergic activity than amitriptyline or doxepin.
- The single-bond side chain of desipramine may be associated with less anticholinergic activity than is the double-bond side chain of amitriptyline or doxepin.
- This may mean less dry mouth, less blurred vision, less urinary retention.

(See Warnings in Brief Summary overleaf.)



lectron micrograph of human synaptic left, postulated site of tricyclic ntidepressant activity.

Laboratory evidence suggests that desipramine blocks the re-uptake of norepinephrine at the presynaptic nerve terminal,[7] ensuring increased cleft and receptor site neurotransmitter concentration.

Potent blocker of norepinephrine re-uptake

Current evidence[5,6] suggests depression results from inadequate levels of norepinephrine or serotonin here, at the synaptic cleft.

If your patient is unresponsive to treatment with one tricyclic, consider switching to a tricyclic that blocks the re-uptake of a different neurotransmitter.

(Please read the Brief Summary overleaf before prescribing Norpramin.)

Merrell

MNQ-266

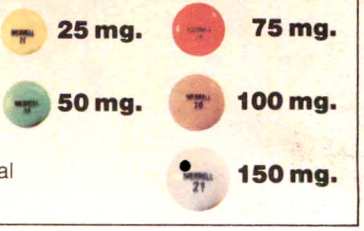
☐ potent blocker of norepinephrine re-uptake

☐ with improved sleep patterns beginning within one week in some patients[1]

☐ with less anticholinergic activity than amitriptyline or doxepin, as shown in studies of animal tissues[2] and normal human subjects[3,4] (See Warnings section of Brief Summary.)

Dosage Flexibility

Convenient choice of 5 tablet strengths and once daily or divided dosage schedule allows titration to individual response.



Norpramin®
(desipramine hydrochloride tablets NF)

AVAILABLE ONLY ON PRESCRIPTION

Brief Summary

MECHANISM OF ACTION: Available evidence suggests that many depressions have a biochemical basis in the form of a relative deficiency of neurotransmitters such as norepinephrine and serotonin. Norepinephrine deficiency may be associated with relatively low urinary 3-methoxy-4-hydroxyphenyl glycol (MHPG) levels, while serotonin deficiencies may be associated with low spinal fluid levels of 5-hydroxyindolacetic acid.

While the precise mechanism of action of the tricyclic antidepressants is unknown, a leading theory suggests that they restore normal levels of neurotransmitters by blocking the re-uptake of these substances from the synapse in the central nervous system.

Evidence indicates that the secondary amine tricyclic antidepressants, including Norpramin, may have greater activity in blocking the re-uptake of norepinephrine. Tertiary amine tricyclic antidepressants, such as amitriptyline, may have greater effect on serotonin re-uptake.

Norpramin (desipramine hydrochloride) is not a monoamine oxidase (MAO) inhibitor and does not act primarily as a central nervous system stimulant. It has been found in some studies to have a more rapid onset of action than imipramine. Earliest therapeutic effects may occasionally be seen in 2 to 5 days, but full treatment benefit usually requires 2 to 3 weeks to obtain.

INDICATIONS: Norpramin (desipramine hydrochloride) is indicated for relief of symptoms in various depressive syndromes, especially endogenous depression.

CONTRAINDICATIONS: Desipramine hydrochloride should not be given in conjunction with, or within 2 weeks of, treatment with an MAO inhibitor drug; hyperpyretic crises, severe convulsions, and death have occurred in patients taking MAO inhibitors and tricyclic antidepressants. When Norpramin (desipramine hydrochloride) is substituted for an MAO inhibitor, at least 2 weeks should elapse between treatments. Norpramin should then be started cautiously and should be increased gradually.

The drug is contraindicated in the acute recovery period following myocardial infarction. It should not be used in those who have shown prior hypersensitivity to the drug. Cross sensitivity between this and other dibenzazepines is a possibility.

WARNINGS: 1. Extreme caution should be used when this drug is given in the following situations: a. In patients with cardiovascular disease, because of the possibility of conduction defects, arrhythmias, tachycardias, strokes, and acute myocardial infarction. b. In patients with a history of urinary retention or glaucoma, because of the anticholinergic properties of the drug. c. In patients with thyroid disease or those taking thyroid medication, because of the possibility of cardiovascular toxicity, including arrhythmias. d. In patients with a history of seizure disorder, because this drug has been shown to lower the seizure threshold. **2.** This drug is capable of blocking the antihypertensive effect of guanethidine and similarly acting compounds. **3.** USE IN PREGNANCY: Safe use of desipramine hydrochloride during pregnancy and lactation has not been established; therefore, if it is to be given to pregnant patients, nursing mothers, or women of childbearing potential, the possible benefits must be weighed against the possible hazards to mother and child. Animal reproductive studies have been inconclusive. **4.** USE IN CHILDREN: Norpramin (desipramine hydrochloride) is not recommended for use in children since safety and effectiveness in the pediatric age group have not been established. **5.** The patient should be cautioned that this drug may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery. **6.** In patients who may use alcohol excessively, it should be borne in mind that the potentiation may increase the danger inherent in any suicide attempt or overdosage.

PRECAUTIONS: 1. It is important that this drug be dispensed in the least possible quantities to depressed outpatients, since suicide has been accomplished with this class of drug. Ordinary prudence requires that children not have access to this drug or to potent drugs of any kind; if possible this drug should be dispensed in containers with child-resistant safety closures. Storage of this drug in the home must be supervised responsibly. **2.** If serious adverse effects occur, dosage should be reduced or treatment should be altered. **3.** Norpramin (desipramine hydrochloride) therapy in patients with manic-depressive illness may induce a hypomanic state after the depressive phase terminates. **4.** The drug may cause exacerbation of psychosis in schizophrenic patients. **5.** Close supervision and careful adjustment of dosage are required when this drug is given concomitantly with anticholinergic or sympathomimetic drugs. **6.** Patients should be warned that while taking this drug their response to alcoholic beverages may be exaggerated. **7.** Clinical experience in the concurrent administration of ECT and antidepressant drugs is limited. Thus, if such treatment is essential, the possibility of increased risk relative to benefits should be considered. **8.** The sedative effects of Norpramin and benzodiazepines (*e.g.*, chlordiazepoxide or diazepam) are additive. Both the sedative and anticholinergic effects of the major tranquilizers are additive to those of Norpramin. **9.** This drug should be discontinued as soon as possible prior to elective surgery because of the possible cardiovascular effects. Hypertensive episodes have been observed during surgery in patients taking desipramine hydrochloride. **10.** Both elevation and lowering of blood sugar levels have been reported. **11.** Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathologic neutrophil depression. **12.** Norpramin 25, 50, 75, and 100 mg. tablets contain FD&C Yellow No. 5 (tartrazine), which may cause allergic-type reactions (including bronchial asthma) in certain susceptible individuals. Although the overall incidence of FD&C Yellow No. 5 (tartrazine) sensitivity in the general population is low, it is frequently seen in patients who also have aspirin hypersensitivity.

ADVERSE REACTIONS: NOTE: Included in the following listing are a few adverse reactions that have not been reported with this specific drug. However, the pharmacologic similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when Norpramin (desipramine hydrochloride) is given.

Cardiovascular: hypotension, hypertension, tachycardia, palpitation, arrhythmias, heart block, myocardial infarction, stroke.

Psychiatric: confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurologic: numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures; alteration in EEG patterns; tinnitus.

Anticholinergic: dry mouth, and rarely associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis, increased intraocular pressure; constipation, paralytic ileus; urinary retention, delayed micturition, dilatation of urinary tract.

Allergic: skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight), edema (of face and tongue or general), drug fever, cross sensitivity with other tricyclic drugs.

Hematologic: bone marrow depressions including agranulocytosis, eosinophilia, purpura, thrombocytopenia.

Gastrointestinal: anorexia, nausea and vomiting, epigastric distress, peculiar taste, abdominal cramps, diarrhea, stomatitis, black tongue.

Endocrine: gynecomastia in the male, breast enlargement and galactorrhea in the female; increased or decreased libido, impotence, testicular swelling; elevation or depression of blood sugar levels.

Other: jaundice (simulating obstructive), altered liver function; weight gain or loss; perspiration, flushing; urinary frequency, nocturia; parotid swelling; drowsiness, dizziness, weakness and fatigue, headache; alopecia.

Withdrawal Symptoms: Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache, and malaise.

DOSAGE AND ADMINISTRATION: Not recommended for use in children. Lower dosages are recommended for elderly patients and adolescents. Lower dosages are also recommended for outpatients compared to hospitalized patients, who are closely supervised. Dosage should be initiated at a low level and increased according to clinical response and any evidence of intolerance. Following remission, maintenance medication may be required for a period of time and should be at the lowest dose that will maintain remission.

Usual Adult Dose: The usual adult dose is 100 to 200 mg. per day. In more severely ill patients, dosage may be further increased gradually to 300 mg./day if necessary. Dosages above 300 mg./day are not recommended.

Dosage should be initiated at a lower level and increased according to tolerance and clinical response.

Treatment of patients requiring as much as 300 mg. should generally be initiated in hospitals, where regular visits by the physician, skilled nursing care, and frequent electrocardiograms (ECG's) are available.

The best available evidence of impending toxicity from very high doses of Norpramin is prolongation of the QRS or QT intervals on the ECG. Prolongation of the PR interval is also significant, but less closely correlated with plasma levels. Clinical symptoms of intolerance, especially drowsiness, dizziness, and postural hypotension, should also alert the physician to the need for reduction in dosage. Plasma desipramine measurement would constitute the optimal guide to dosage monitoring.

Initial therapy may be administered in divided doses or a single daily dose.

Maintenance therapy may be given on a once-daily schedule for patient convenience and compliance.

Adolescent and Geriatric Dose: The usual adolescent and geriatric dose is 25 to 100 mg. daily.

Dosage should be initiated at a lower level and increased according to tolerance and clinical response to a usual maximum of 100 mg. daily. In more severely ill patients, dosage may be further increased to 150 mg./day. Doses above 150 mg./day are not recommended in these age groups.

Initial therapy may be administered in divided doses or a single daily dose.

Maintenance therapy may be given on a once-daily schedule for patient convenience and compliance.

OVERDOSAGE: See prescribing information for a discussion of symptoms and treatment of overdose.

Product Information as of July, 1979

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4. Peterson, G.R., Blackwell, B., Hostetler, R.M., Kuzma, R., and Adolphe, A.: Anticholinergic activity of the tricyclic antidepressants despiramine [*sic*] and doxepin in nondepressed volunteers. Communic. Psychopharmacol. 2:145-150, 1978.

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7. Ross, S.B. and Renyi, A.L.: Tricyclic antidepressant agents. I. Comparison of the inhibition of the uptake of ^{3}H-noradrenaline and ^{14}C-5-hydroxytryptamine in slices and crude synaptosome preparations of the midbrain-hypothalamus region of the rat brain. Acta Pharmacol. (Kobenhavn) 36:382-394, 1975.

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New!

LAWYERS, PSYCHIATRISTS AND CRIMINAL LAW

Cooperation or Chaos?

By **Harlow M. Huckabee,** *Arlington, Virginia. Foreword by* **Fred G. Folsom** *and* **Cono R. Namorato.** This law-psychiatry interface brings together and analyzes the conflicting positions and theories related to criminal responsibility and competency to stand trial.

The first of two main sections focuses on criminal responsibility and the controversy generated by that topic among professionals. Among the areas reviewed are responsibility tests, evidence of mental disorder on *mens rea* the mental state required for the offense), various proposals to substitute *mens rea* for traditional responsibility tests, and the current chaos and need for cooperation among psychiatrists and lawyers. In the concluding chapter of this section, the author suggests the establishment of a research/clearinghouse program that would coordinate efforts to solve problems at both the state and federal levels.

The second section evaluates a number of other problems related to law and psychiatry. Competency to stand trial, the debate on psychiatrists' expertise in matters of criminal law, the briefing of psychiatrists, shopping for psychiatrists, and the use of impartial experts and/or court psychiatric clinics are all analyzed.

This volume points to the factors affecting psychiatric opinions and describes how psychiatrists can arrive at opposite conclusions concerning the same defendant; clarifies the culpability of courts and lawyers, as well as psychiatrists, for the breakdown in the system; and demonstrates the criticality of lawyer-psychiatrist cooperation. Its inclusion of suggestions for further research and action in this area adds to the importance of this work. *'80, 184 pp., $16.75*

New! **PRIMER ON THE RORSCHACH TECHNIQUE: A Method of Administration, Scoring, and Interpretation** *by* **Eugene E. Levitt,** *Indiana Univ. School of Medicine, Indianapolis.* Special features include a step-by-step format, a discussion of methods for correlating the Rorschach with the MMPI, and a chapter on the use of the Rorschach technique with children. Appendices present information on the probabilities of the use of color as a determinant in selected responses, scoring illustrations, and interpretation of individual Rorschach factors. *'80, 116 pp., 2 il., 15 tables, cloth-$14.50, paper-$8.95*

THE AMERICAN ALCOHOLIC: The Nature-Nurture Controversy in Alcoholic Research and Therapy (3rd Ptg.) *by* **William Madsen,** *Univ. of California, Santa Barbara. '80, 272 pp., 4 il., $14.50*

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New! **BODYMIND LIBERATION: Achieving Holistic Health** *by* **Robert Henley Woody,** *Univ. of Nebraska, Omaha.* The author of this text explains how the integration of physical and mental processes determines the overall health of the human organism. He shows how the bodymind operates, how bodymind problems occur, and how the bodymind can be liberated and restored to health through pragmatic techniques. The necessity of controlling anxiety; the significance of self-actualization; the effects of trust, caring, and sharing; the value of physical contact; and related subjects are examined. *'80, 172 pp., $15.50*

New! **GRIEF COUNSELING AND SUDDEN DEATH: A Manual and Guide** *by* **Polly Doyle,** *Contra Costa County Crisis and Suicide Intervention Center, Walnut Creek, California. (8 Contributors)* Not only does this book provide the information needed to understand grief and to counsel bereaved persons, it also details the development and operation of a community-based, volunteer grief counseling program. An extensive section on general counseling techniques and approaches covers such specifics as the anniversary interview, the role of the Coroner's Office, the autopsy, and the funeral. *'80, 336 pp., 1 il., $21.50*

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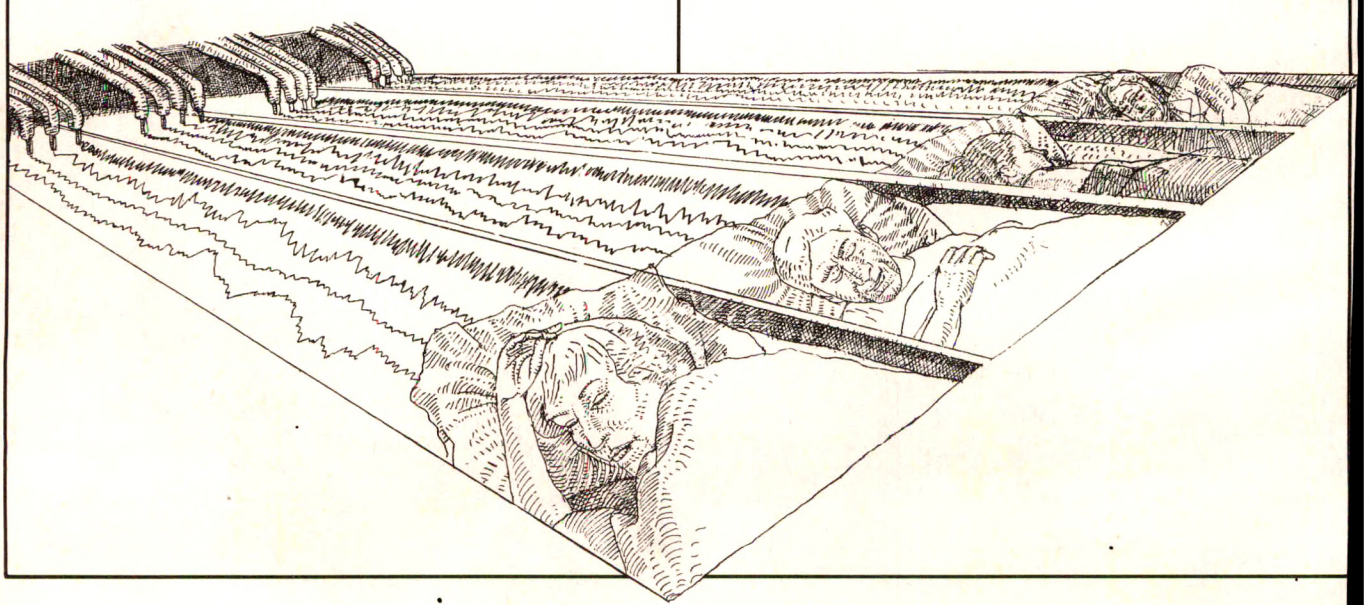
MORE POLYGRAPHIC EVIDENCE

Sleep research laboratory testing of insomnia patients for 995 subject nights demonstrates efficacy of Dalmane (flurazepam HCl/Roche)[1]

In 14 separate studies, subjects given Dalmane were monitored with polygraphic recordings for a total of 995 nights, objectively demonstrating the efficacy of Dalmane.

Efficacy from the first night through 28 nights, without need to increase dosage

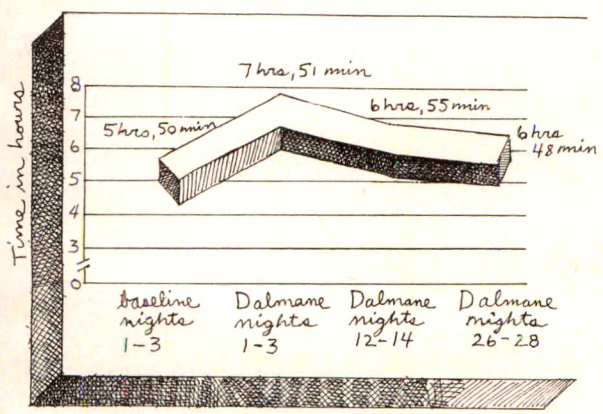
Dalmane (flurazepam HCl/Roche) is the only sleep medication objectively proven effective through 28 consecutive nights of administration in two separate studies in the sleep research laboratory.[2-4] The prolonged use of Dalmane is seldom necessary; should it be, appropriate patient evaluations such as periodic blood counts and liver and kidney function tests are recommended.



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)F EFFICACY FOR INSOMNIA

Dalmane® (flurazepam HCl/Roche) improved sleep during short, intermediate and longer-term use



Improvement shown represents mean improvement, in hours, of total sleep time. n=5

Adapted from Dement WC, *et al: Behav Med* 5:25-31, Oct 1978

Since most insomnia is transitory in nature, initiate therapy at the lowest effective dose for the shortest time advisable to achieve a predetermined therapeutic goal.

References:
1. Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley NJ
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3. Dement WC, *et al: Behav Med* 5: 25-31, Oct 1978
4. Kales A, *et al: Clin Pharmacol Ther 18:* 356-363, Sep 1975

Clinical documentation of efficacy in 4950 patients with insomnia[1]

Dalmane (flurazepam HCl/Roche) efficacy was evaluated in 18 clinical studies. These included paired-night, double-blind crossover, double-blind comparison and patient preference studies. Results of these studies demonstrated and confirmed the effectiveness of Dalmane.

Periodic evaluation of patient's sleep habits may help prevent subsequent insomnia

Should insomnia recur, the patient may need guidance in setting up a regular sleep program. This may include establishment of a regular bedtime hour, taking a warm bath prior to retiring and avoidance of alcohol, stimulants or heavy meals late in the evening.

DALMANE® (IV)

flurazepam HCl/Roche
15-mg and 30-mg capsules

UNMATCHED PROOF, UNMATCHED EFFICACY

Please see summary of product information on following page.

ROCHE®

Protocol for insomnia management

☐ Treat physical and/or psychological factors that may underlie an inability to sleep.

☐ Thoroughly question and evaluate new patients who specifically request sleep medications.

☐ Consider other medications the patient may be taking (including alcoholic beverages) and be aware of possible drug interactions.

☐ Select a sleep medication with objectively *documented* proof of efficacy and a wide margin of safety.

☐ Initiate treatment at the lowest effective dose for the period of time deemed necessary *to achieve a reasonable therapeutic goal.*

☐ Monitor response to therapy and, based upon patient reassessment, determine need for continuing medication on an individual basis.

☐ Advise patients who ingest alcoholic beverages or other CNS depressants against doing so while on sleep medication. CNS stimulants such as caffeine should also be discontinued.

☐ Caution patients against engaging in hazardous occupations requiring complete mental alertness such as operating machinery or driving a motor vehicle shortly after ingesting the drug. In addition, as with all CNS depressants, patients may have reduced motor performance while on hypnotic therapy and should be advised accordingly.

☐ Avoid prescribing for suicidal or dependency-prone individuals whose histories indicate an inability to handle any psychoactive substances including alcohol.

Now available from Roche—important guidance for your patients with insomnia.

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DALMANE® (IV)
flurazepam HCl/Roche

One 15-mg capsule *h.s.*—recommended initial dosage for elderly or debilitated patients.
One 30-mg capsule *h.s.*—usual adult dosage
(15 mg may suffice in some patients).

Before prescribing, please consult complete product information, a summary of which follows:
Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Objective sleep laboratory data have shown effectiveness for at least 28 consecutive nights of administration. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended.
Contraindications: Known hypersensitivity to flurazepam HCl.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

Usage in Pregnancy: **Several studies of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) suggest increased risk of congenital malformations during the first trimester of pregnancy. Dalmane, a benzodiazepine, has not been studied adequately to determine whether it may be associated with such an increased risk. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.
Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies, or with impaired renal or hepatic function. Periodic blood counts and liver and kidney function tests are advised during repeated therapy.
Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase.
Dosage: Individualize for maximum beneficial effect.
Adults: 30 mg usual dosage; 15 mg may suffice in some patients.
Elderly or debilitated patients: 15 mg recommended initially until response is determined.
Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

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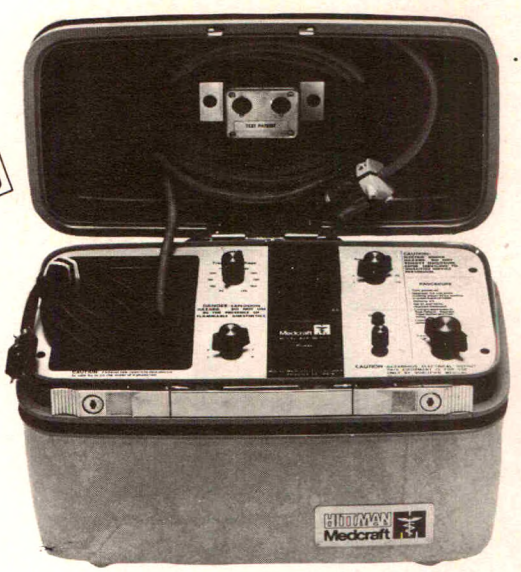
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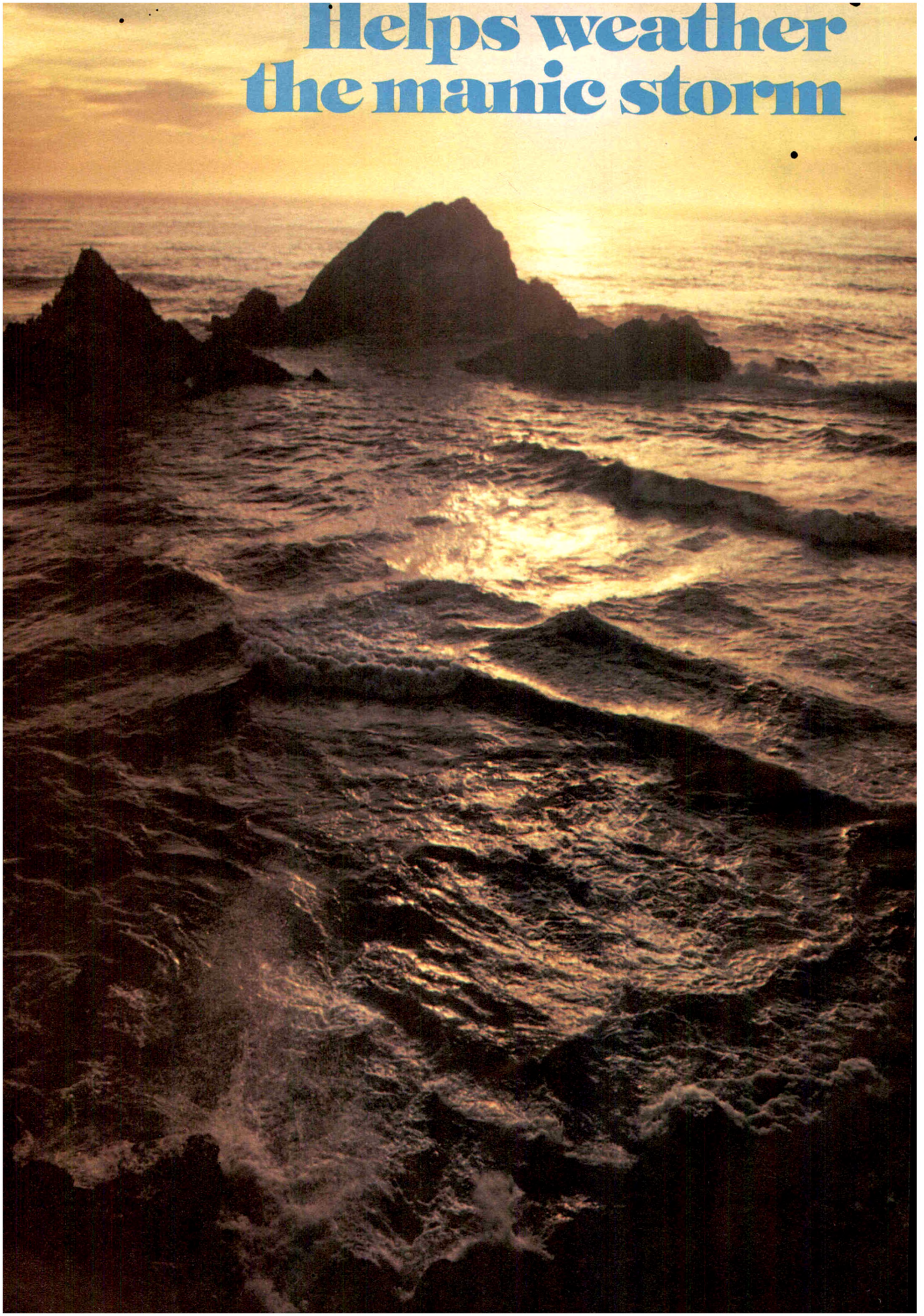
- Line Voltage Compensation — allows compensation for fluctuations in incoming line voltages.
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maintenance therapy with Eskalith®

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In manic-depressive patients with a history of mania, 'Eskalith'

- prevents or diminishes the intensity of future episodes
- can increase the intervals between episodes
- helps patients function within normal limits

Before prescribing, see complete prescribing information in SK&F literature or PDR. The following is a brief summary.

WARNING
Lithium toxicity is closely related to serum lithium levels, and can occur at doses close to therapeutic levels. Facilities for prompt and accurate serum lithium determinations should be available before initiating therapy.

Indications: Treatment of manic episodes of manic-depressive illness. Maintenance therapy prevents or diminishes the intensity of subsequent episodes in manic-depressive patients with a history of mania.

Warnings: Lithium should generally not be given to patients with significant renal or cardiovascular disease, severe debilitation or dehydration, sodium depletion, or to patients receiving diuretics.

Chronic lithium therapy may be associated with diminution of renal concentrating ability; such patients should be carefully managed to avoid dehydration with resulting lithium retention and toxicity. Morphologic changes with glomerular and interstitial fibrosis and nephron atrophy have been reported. When renal function is assessed, progressive or sudden changes in function indicate the need for reevaluation of treatment.

Caution patient and family to watch for diarrhea, vomiting, tremor, mild ataxia, drowsiness, or muscular weakness as signs of lithium toxicity, and to discontinue therapy and contact a physician should they occur. Patients receiving combined therapy with lithium and an antipsychotic should be monitored closely for early evidence of neurologic toxicity and treatment discontinued promptly if such signs appear. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery).

Lithium may prolong the effects of neuromuscular blocking agents. Such agents should be given with caution to patients receiving lithium.

Lithium may cause fetal developmental anomalies, especially involving the cardiovascular system. If lithium is used during pregnancy, or if a patient becomes pregnant while taking it, she should be apprised of the potential hazard to the fetus. Except in rare and unusual circumstances, nursing should not be undertaken while a patient is on lithium therapy, since lithium is excreted in human milk.

Not recommended for children under 12.

Precautions: Lithium tolerance is greater during the acute manic phase and decreases when manic symptoms subside.

Lithium therapy may lead to sodium depletion. Normal diet (including salt) and adequate fluid intake (2500-3000 ml.) must be maintained, at least during initial stabilization period. Protracted sweating or diarrhea can decrease tolerance; in such cases, administer supplemental fluid and salt.

Sweating, diarrhea, and concomitant infection with elevated temperatures may require temporary reduction or cessation of dosage.

Where hypothyroidism exists, thyroid function should be monitored during lithium stabilization and maintenance; where hypothyroidism occurs during stabilization and maintenance, supplemental thyroid treatment may be used.

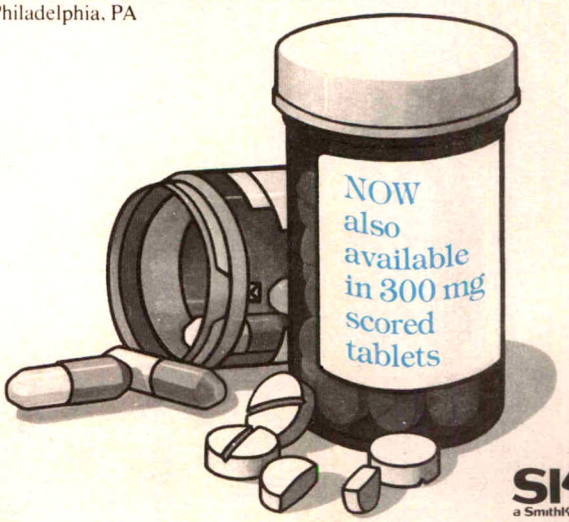
Adverse Reactions: Mild to moderate toxic reactions may occur at serum lithium levels from 1.5 to 2.5 mEq./1., and moderate to severe reactions at levels from 2.0 to 2.5 mEq./1. Fine hand tremor, polyuria, and mild thirst may occur during initial therapy and persist. Transient and mild nausea and general discomfort may also appear during initial therapy. These effects usually subside with continued treatment or temporary reduction or cessation of dosage. If persistent, discontinue dosage. Diarrhea, vomiting, drowsiness, muscular weakness, and lack of coordination may be early signs of toxicity and may occur at levels below 2.0 mEq./1. At higher levels, ataxia, giddiness, tinnitus, blurred vision, and a large output of dilute urine may be seen. Serum levels above 3.0 mEq./1. may produce a complex clinical picture, involving multiple organs and systems. Serum levels should not exceed 2.0 mEq./1. during acute phase.

The following reactions appear to be related to serum lithium levels, including levels within the therapeutic range: **Neuromuscular**—tremor, muscle hyperirritability (fasciculations, twitching, clonic movements of whole limbs), ataxia, choreo-athetotic movements, hyperactive deep tendon reflex; **Central Nervous System**—blackout spells, epileptiform seizures, slurred speech, dizziness, vertigo, incontinence of urine or feces, somnolence, psychomotor retardation, restlessness, confusion, stupor, coma; **Cardiovascular**—cardiac arrhythmia, hypotension, peripheral circulatory collapse; **Gastrointestinal**—anorexia, nausea, vomiting, diarrhea; **Genitourinary**—albuminuria, oliguria, polyuria, glycosuria; **Dermatologic**—drying and thinning of hair, alopecia, anesthesia of skin, chronic folliculitis, exacerbation of psoriasis, xerosis cutis; **Autonomic**—blurred vision, dry mouth; **Thyroid Abnormalities**—euthyroid goiter and/or hypothyroidism (including myxedema) with lower T_3 and T_4. I^{131} uptake may be elevated; rare cases of hyperthyroidism; **EEG Changes**—diffuse slowing, widening of the frequency spectrum, potentiation and disorganization of background rhythm; **EKG Changes**—reversible flattening, isoelectricity or inversion of T-waves; **Miscellaneous**—fatigue, lethargy, transient scotomata, dehydration, weight loss, tendency to sleep.

Reactions unrelated to dosage include: transient EEG and EKG changes, leukocytosis, headache, diffuse nontoxic goiter with or without hypothyroidism, transient hyperglycemia, generalized pruritus with or without rash, cutaneous ulcers, albuminuria, worsening of organic brain syndromes, excessive weight gain, edematous swelling of ankles or wrists, thirst or polyuria, sometimes resembling diabetes insipidus, and metallic taste. A single case of a syndrome resembling Raynaud's has been reported.

How Supplied: 300 mg. capsules in bottles of 100.
300 mg. scored tablets in bottles of 100.

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Indications and Usage: Symptomatic relief of anxiety, tension, agitation, irritability insomnia associated with anxiety neuroses and transient situational disturbances; an associated with depressive symptoms and as a treatment of symptoms of anxiety if such sy toms are a significant feature of functional or organic disorders, particularly gastrointestina cardiovascular.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by syst atic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucom

Warnings: Not recommended in primary depressive disorders or psychoses. As with all C acting drugs, warn patients on lorazepam not to operate machinery or motor vehicles, an diminished tolerance for alcohol and other CNS depressants. ●

Physical and Psychological Dependence: Withdrawal symptoms like those noted with ba turates and alcohol have occurred following abrupt discontinuance of benzodiazep (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Ad tion-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveilla when on benzodiazepines because of their predisposition to habituation and dependei Withdrawal symptoms have also been reported following abrupt discontinuance of benz azepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid o sedation.

Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may resu symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and o sional convulsions.

Observe usual precautions with impaired renal or hepatic function.

Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazep has not been shown of significant benefit in treating gastrointestinal or cardiovascular con nent.

Esophageal dilation occurred in rats treated with lorazepam for more than 1 yea 6mg kg day. No effect dose was 1.25mg/kg/day (approximately 6 times the maximum hu therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn w 2 months of first observation. Clinical significance is unknown; but use of lorazepam for longed periods and in geriatric patients requires caution and frequent monitoring for sympt of upper G.I. disease.

Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depres effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerge rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rab Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastrosch malformed skull and microphthalmia) were seen in drug-treated rabbits without relationsh dosage. Although all these anomalies were not present in the concurrent control group, have been reported to occur randomly in historical controls. At 40mg/kg and higher, there evidence of fetal resorption and increased fetal loss in rabbits which was not seen at l doses. Clinical significance of these findings is not known. However, increased risk of con tal malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam meprobamate) during first trimester of pregnancy has been suggested in several stu Because use of these drugs is rarely a matter of urgency, use of lorazepam during this pe should almost always be avoided. Possibility that a woman of child-bearing potential ma pregnant at institution of therapy should be considered. Advise patients if they become p nant to communicate with their physician about desirability of discontinuing the drug.

In humans, blood levels from umbilical cord blood indicate placental transfer of loraze and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like c benzodiazepines. As a general rule, nursing should not be undertaken while on a drug s many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and gene disappear on continued medication or on decreasing dose. In a sample of about 3,500 anx patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6. weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, sea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, function disturbance, various gastrointestinal symptoms and autonomic manifestations. dence of sedation and unsteadiness increased with age. Small decreases in blood pres have been noted but are not clinically significant, probably being related to relief of anxiety

Overdosage: In management of overdosage with any drug, bear in mind that multiple ag may have been taken. Manifestations of overdosage include somnolence, confusion and c Induce vomiting and/or undertake gastric lavage followed by general supportive care, mor ing of vital signs and close observation. Hypotension, though unlikely, usually may be contr with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined

Ativan® IV for (lorazepam) Anxiety

Dosage: **Individualize for maximum beneficial effects. Increase d gradually when needed, giving higher evening dose before increa daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dos may vary from 1 to 10mg/day in divided doses. For elderly or de tated, initially 1-2mg/day; insomnia due to anxiety or transient s tional stress, 2-4mg h.s.**

How Supplied: **0.5, 1.0 and 2.0mg tablets.**

Wyeth Laboratories
Philadelphia, PA 19101

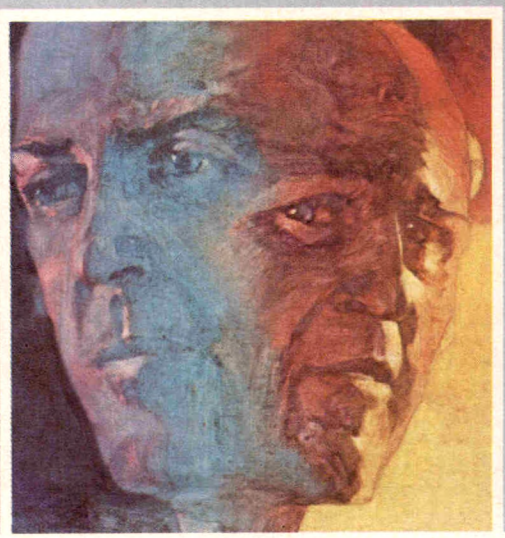
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A mixed syndrome. When drug intervention is indicated in patients whose anxiety is accompanied by depressive symptoms, the choice, essentially, is among three basic strategies: (1) an antidepressant; (2) a tranquilizer; (3) a combination of (1) and (2).

With respect to strategy (2), you should know that Ativan (lorazepam) has been shown to be statistically and clinically significantly effective in alleviating the anxiety component of this mixed syndrome, in common protocol controlled studies of 653 patients (337 on Ativan).

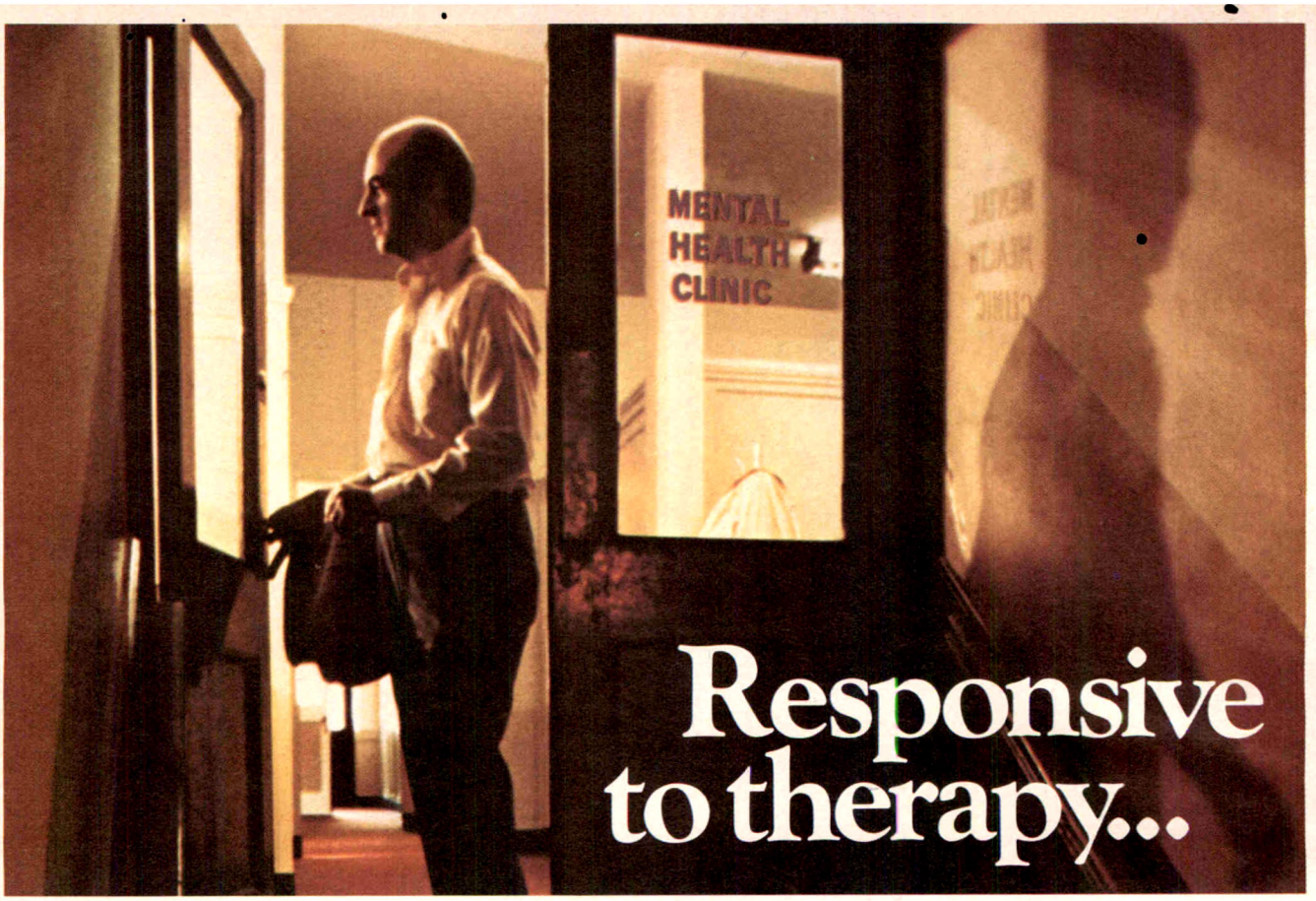
Since Ativan was also found to be compatible with tricyclics, if you opt for strategy (3), your patient has the benefit of rapid anxiolytic action, absence of long-acting metabolites and rapid clearance when you decide to discontinue Ativan. All benzodiazepines, however, including Ativan, produce added CNS depressant effects when administered with alcohol and other CNS depressants.

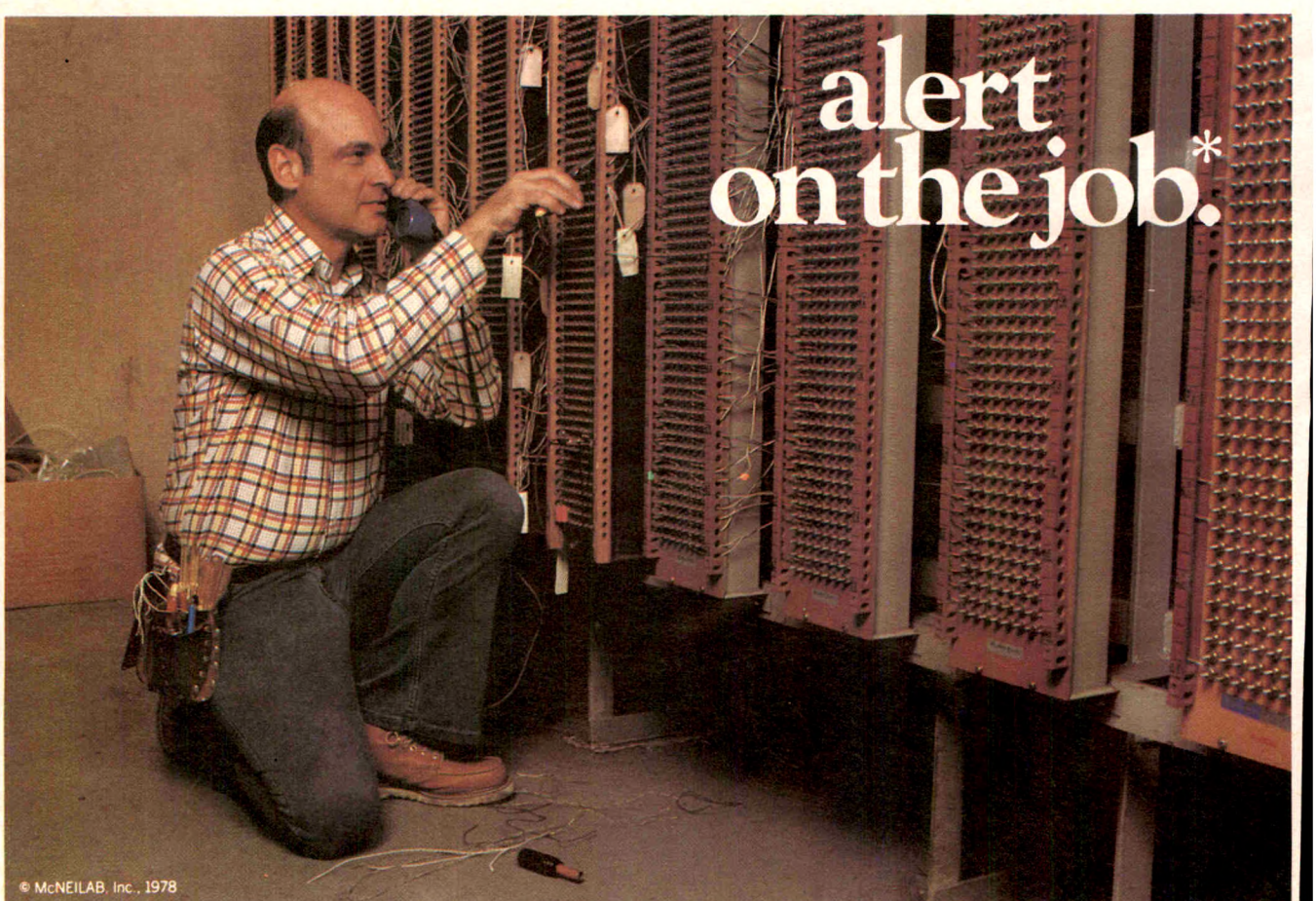
In either case, Ativan offers you the convenience of a b.i.d. schedule and a flexibility not possible with any fixed combination.



See important information on preceding page.

Ativan® (IV)
for (lorazepam)
Anxiety
associated with depressive symptoms


MENTAL
HEALTH
CLINIC
Responsive
to therapy...


alert
on the job.*
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Haldol®
(haloperidol)
tablets/concentrate/injection

For long-term management of psychotic patients... with minimal risk of adverse effects

Highly effective in a wide range of acute and chronic psychotic disorders[1-7]

Permits aggressive titration to effective dosage levels for optimal control[1,4]

Usually leaves patients alert and responsive...
easier to reach with supportive measures[1-3]

Not an actual case history, this situation illustrates the action of HALDOL haloperidol as reported in various clinical studies (available on request).

Minimal risk of hypotension, oversedation, or troublesome anticholinergic effects[3-5,7-9]
Transient hypotension occurs rarely; severe orthostatic hypotension has not been reported. Although some instances of drowsiness have been reported, marked sedation is rare.

Common side effects easily controlled[1,3-7]
Although extrapyramidal symptoms (EPS) have been reported frequently, they are usually dose-related and readily controlled with dose adjustment or antiparkinson drugs. EPS often diminish spontaneously with continued use of HALDOL haloperidol.

References: 1. Man, P.L.: Dis. Nerv. Syst. *34*:113 (Feb.) 1973. 2. Sugerman, A.A., et al.: Am. J. Psychiatry *129*:1190 (June) 1964. 3. Ayd, F.J., Jr.: Med. Sci. *18*:55 (Oct.) 1967. 4. Howard, J.S.: Dis. Nerv. Syst. *35*:458 (Oct.) 1974. 5. Abuzzahab, F.S., Sr.: Psychosomatics *11*:188 (May-June) 1970. 6. Darling, H.F.: Dis. Nerv. Syst. *34*:364 (Oct.-Nov.) 1973. 7. Gerle, B.: Clin. Trials J. *3*:380 (Feb.) 1966. 8. Snyder, S.H., et al: Science *184*:1243 (June 21) 1974. 9. Stimmel, C.L.: Dis. Nerv. Syst. *34*:219 (Apr.) 1976.

Please turn page for summary of prescribing information.

concentrate

A tasteless, odorless, colorless Liquid Concentrate for better patient acceptability: 2 mg per ml haloperidol (as the lactate).

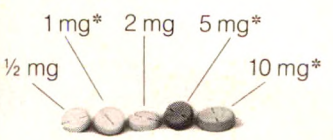
injection

A rapid-acting injection for psychiatric emergencies: 5 mg haloperidol (as the lactate) with 1.8 mg methylparaben and 0.2 mg propylparaben per ml, and lactic acid for pH adjustment to 3.4 ± 0.2.



tablets

5 tablet strengths for convenience in individualizing dosage:



HALDOL® (haloperidol)

*contain FD&C Yellow No. 5 (see Precautions).

tablets/concentrate/injection

A dosage form for every therapeutic need

Summary of Prescribing Information

Contraindications: Severe, toxic CNS depression or comatose states from any cause, hypersensitivity to the drug, Parkinson's disease.

Warnings: ***Usage in Pregnancy:*** Safe use in pregnancy or in women likely to become pregnant has not been established; use only if benefit clearly justifies potential hazards. Infants should not be nursed during drug treatment.

Combined Use With Lithium: Patients receiving lithium plus haloperidol should be monitored closely for early evidence of neurological toxicity.

General: Bronchopneumonia, sometimes fatal, has followed use of major tranquilizers, including haloperidol. Prompt remedial therapy should be instituted if dehydration, hemoconcentration or reduced pulmonary ventilation occurs, especially in the elderly. Decreased serum cholesterol and/or cutaneous and ocular changes have been reported with chemically-related drugs, although not with haloperidol. Mental and/or physical abilities required for hazardous tasks or driving may be impaired. Alcohol should be avoided due to possible additive effects and hypotension.

Precautions: Administer cautiously to patients: (1) with severe cardiovascular disorders, due to the possibility of transient hypotension and/or precipitation of anginal pain (if a vasopressor is required, epinephrine should not be used since HALDOL haloperidol may block its vasopressor activity and paradoxical further lowering of blood pressure may occur); (2) receiving anticonvulsant medication since HALDOL haloperidol may lower the convulsive threshold; (3) with known allergies or a history of allergic reactions to drugs; (4) receiving anticoagulants. Concomitant antiparkinson medication, if required, may have to be continued after HALDOL haloperidol is discontinued because of different excretion rates; if both are discontinued simultaneously, extrapyramidal symptoms may occur. Intraocular pressure may increase when anticholinergic drugs, including antiparkinson drugs, are administered concomitantly with HALDOL haloperidol. When HALDOL haloperidol is used for mania in cyclic disorders, there may be a rapid mood swing to depression. Severe neurotoxicity may occur in patients with thyrotoxicosis receiving antipsychotic medication, including HALDOL haloperidol. FD&C Yellow No. 5 (tartrazine) may cause allergic-type reactions (including bronchial asthma) in certain susceptible individuals, especially in those who have aspirin hypersensitivity.

Adverse Reactions: ***CNS Effects:*** *Extrapyramidal Reactions:* Neuromuscular (extrapyramidal) reactions have been reported frequently, often during the first few days of treatment. Generally they involved Parkinson-like symptoms which were usually mild to moderately severe and usually reversible. Other types of neuromuscular reactions (motor restlessness, dystonia, akathisia, hyperreflexia, opisthotonos, oculogyric crises) have been reported far less frequently, but were often more severe. Severe extrapyramidal reactions have been reported at relatively low doses. Generally, extrapyramidal symptoms are dose-related since they occur at relatively high doses and disappear or become less severe when the dose is reduced. Antiparkinson drugs may be required. Persistent extrapyramidal reactions have been reported and the drug may have to be discontinued in such cases.

Withdrawal Emergent Neurological Signs: Abrupt discontinuation of shor term antipsychotic therapy is generally uneventful. However, some patients o maintenance treatment experience transient dyskinetic signs after abru withdrawal. In certain cases these are indistinguishable from "Persistent Ta dive Dyskinesia" except for duration. It is unknown whether gradual wit drawal will reduce the occurrence of these signs, but until further evidence available haloperidol should be gradually withdrawn.

Persistent Tardive Dyskinesia: Although rarely reported with HALDOL halo peridol, tardive dyskinesia may appear during or after long-term therapy. Th risk appears to be greater in elderly patients on high-dose therapy, especiall females. Symptoms are persistent and sometimes appear irreversible; there i no known effective treatment and all antipsychotic agents should be discor tinued. The syndrome may be masked by reinstitution of drug, increasin dosage, or switching to a different antipsychotic agent.

Other CNS Effects: Insomnia, restlessness, anxiety, euphoria, agitatior drowsiness, depression, lethargy, headache, confusion, vertigo, grand m seizures, and exacerbation of psychotic symptoms.

Cardiovascular Effects: Tachycardia and hypotension. ***Hematologi Effects:*** Reports of mild, usually transient leukopenia and leukocytosis, mir mal decreases in red blood cell counts, anemia, or a tendency toward lyr phomonocytosis; agranulocytosis rarely reported and only in association wi other medication. ***Liver Effects:*** Impaired liver function and/or jaundic reported. ***Dermatologic Reactions:*** Maculopapular and acneiform reaction isolated cases of photosensitivity, loss of hair. ***Endocrine Disorders:*** Lact tion, breast engorgement, mastalgia, menstrual irregularities, gynecomasti impotence, increased libido, hyperglycemia and hypoglycemia. ***Gastrointe tinal Effects:*** Anorexia, constipation, diarrhea, hypersalivation, dyspeps nausea and vomiting. ***Autonomic Reactions:*** Dry mouth, blurred vision, u nary retention and diaphoresis. ***Respiratory Effects:*** Laryngospasm, bro chospasm and increased depth of respiration.

The injectable form is intended only for acutely agitated psychotic patier with moderately severe to very severe symptoms.

Caution: Federal law prohibits dispensing without prescription. 0

IMPORTANT: Full directions for use should be read before HALD haloperidol is administered or prescribed.

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When depressive symptoms come in a cluster of blues...

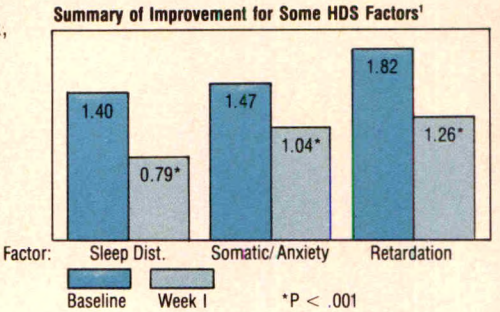


Adapin® (doxepin HCl) helps patients see life in all its colors

Improvements noted at one week

Depressed patients often present with fatigue, anorexia, anxiety, mood swings, loss of interest, *and most frequently, early awakening and insomnia*. In a summation of 9 clinical studies involving 198 patients, significant clinical improvement was noted after the first week of doxepin treatment.[1] However, optimal antidepressant response may not be evident for two to three weeks.

Summary of Improvement for Some HDS Factors[1]



Normalized sleep patterns

In a study of clinically depressed patients with symptoms of insomnia, treatment with doxepin significantly reduced difficulty falling asleep, difficulty staying asleep and frequency of early morning awakening.[2]

Low potential for cardiotoxicity

The incidence of tachycardia and hypotension, which have been observed during treatment with tricyclic antidepressants, has been reported infrequently with doxepin. In 452 patients given doxepin, the incidence of tachycardia was 2.88%; in 495 patients, the incidence of hypotension was 2.62%.[3]

Now available... 75 mg capsules for h.s. dosage

Brief Summary of Prescribing Information ADAPIN® (doxepin HCl) Capsules

Indications–Relief of symptoms of anxiety and depression.

Contraindications–Glaucoma, tendency toward urinary retention or hypersensitivity to doxepin.

Warnings–Adapin has not been evaluated for safety in pregnancy. No evidence of harm to the animal fetus has been shown in reproductive studies. There are no data concerning secretion in human milk, nor on effect in nursing infants.

Usage in children under 12 years of age is not recommended. MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug, as serious side-effects and death have been reported with the concomitant use of certain drugs and MAO inhibitors.

In patients who may use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage.

Precautions–Drowsiness may occur and patients should be cautioned against driving a motor vehicle or operating hazardous machinery.

Since suicide is an inherent risk in depressed patients they should be closely supervised while receiving treatment.

Although Adapin has shown effective tranquilizing activity, the possibility of activating or unmasking latent psychotic symptoms should be kept in mind.

This product contains FD&C Yellow No. 5 (tartrazine) which may cause allergic-type reactions (including bronchial asthma) in certain susceptible individuals. Although the overall incidence of FD&C Yellow No. 5 (tartrazine) sensitivity in the general population is low, it is frequently seen in patients who also have aspirin hypersensitivity.

Adverse Reactions–Dry mouth, blurred vision and constipation have been reported. Drowsiness has also been observed.

Adverse effects occurring infrequently include extrapyramidal symptoms, gastrointestinal reactions, secretory effects such as sweating, tachycardia and hypotension. Weakness, dizziness, fatigue, weight gain, edema, paresthesias, flushing, chills, tinnitus, photophobia, decreased libido, rash and pruritus may also occur.

Dosage and Administration–In mild to moderate anxiety and/or depression: 25 mg t.i.d. Increase or decrease the dosage according to individual response. Daily dosage, up to 150 mg may be taken at bedtime without loss of effectiveness. Usual optimum daily dosage is 75 mg to 150 mg per day not to exceed 300 mg per day.

Antianxiety effect usually precedes the antidepressant effect by two or three weeks.

How Supplied–Each capsule contains doxepin, as the hydrochloride. 10 mg, 25 mg, 50 mg, 75 mg and 100 mg capsules in bottles of 100 and 1000.

For complete prescribing information please see package insert or PDR.

References:

1. Barranco SF, Thrash ML, Hackett E, et al: Early onset of response to doxepin treatment. *J Clin Psychiatry* 40:265-269, 1979 (Sinequan®).
2. Karacan I, Blackburn AB, Thornby JI: The Effect of Doxepin HCl (Sinequan®) on Sleep Patterns and Clinical Symptomatology of Neurotic Depressed Patients with Sleep Disturbance, in Mendels J (ed): *Sinequan®: A monograph of recent clinical studies*, Princeton, NJ, Excerpta Medica, 1977, pp 4-22.
3. Pitts NE: The clinical evaluation of doxepin–A new psychotherapeutic agent. *Psychosomatics* 10:164-171, 1969.

Sinequan® brand of doxepin HCl was the drug used in studies referenced as 1, 2 and 3.

Rx DIVISION
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ROCHESTER, NEW YORK 14623

Adapin® (doxepin HCl) ... effective single-entity drug with antidepressant and antianxiety effects

A-252


THE 48-HOUR
LOXITANE®
Loxapine Succinate
EFFECT...
Prompt
symptom
improvement

...y response—the first step ard early resocialization

...a relatively short time on LOXITANE® ...ipine Succinate, hospitalized ...zophrenic patients usually become ...er to reach, easier to manage, more ...ciable, and cooperative. A series of ...linical studies[1-4] shows an impressive decrease in conceptual disorganization, thinking disorder, hallucinatory behavior and anxiety-depression during the first 48 hours.

The results in one group of 17 young adult schizophrenics treated at a university hospital typify general clinical experience. In the first 48 hours, these patients achieved a mean overall symptomatic improvement of more than 20% in BPRS.[1] (See chart)

Continued overall improvement... helps facilitate a return to more normal activities

Near maximal control was reported by week four; many behavior patterns approached normal. In other studies, a mean overall improvement of almost 80% was seen by 28 days.[2,3] Prompt therapeutic response and steady progress can help strengthen the patient's capacity to cope with daily living and relationships.

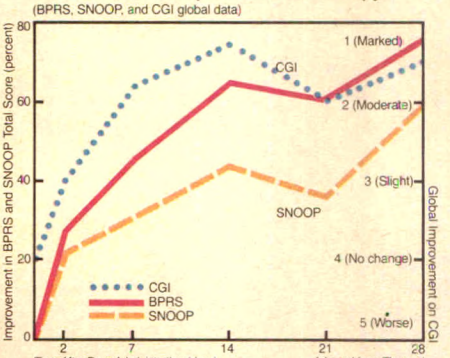
...Minimal risk of certain toxicities... encourages compliance.

On LOXITANE, physical health is rarely compromised by certain toxicities. Renal, hematopoietic, cardiotoxicity, phototoxicity and photosensitivity have not been reported to date.* Therapy in most cases is not interrupted by intolerable side effects. Sexual side effects have not been a significant problem; anticholinergic effects are usually mild, and extrapyramidal reactions are generally easily controlled. Well-tolerated LOXITANE means less frequent need to interrupt therapy—fewer problems with patient compliance.

Note: LOXITANE, as with certain other neuroleptics, lowers the convulsive threshold and should be used with extreme caution in patients with a history of convulsive disorders.

*Although not reported to date, the possibility of phototoxicity, cardiac, renal, or blood toxicity cannot be ruled out at this time. Transient liver enzyme changes not definitely related to LOXITANE have been reported.

Mean improvement in 17 schizophrenic patients on LOXITANE® Loxapine Succinate therapy
(BPRS, SNOOP, and CGI global data)



Adapted from Thomas[1]

CGI (Clinical Global Impressions)
BPRS (Brief Psychiatric Rating Scale)
SNOOP (Systematic Nursing Observation of Psychopathology)

Loxitane®
LOXAPINE SUCCINATE
Lederle

5 mg, 10 mg, 25 mg, 50 mg capsules

See LOXITANE Brief Summary on following page.

For the difference that begins with rapid action...



Loxitane®
LOXAPINE SUCCINATE
Lederle

Recommended Daily Dosage

Dosage	Initially: 20-50 mg During first 24 hours: up to 100 mg Usual therapeutic level: 150 mg/day Maintenance: 60-100 mg/day Maximum: 250 mg/day Dosage may be divided as necessary. Reduced dosage in geriatrics and adolescents should be considered.

Brief Summary
LOXITANE® Loxapine Succinate Capsules
LOXITANE® C Loxapine Hydrochloride Oral Concentrate
LOXITANE® IM Loxapine Hydrochloride

INDICATIONS: Manifestations of schizophrenia.
CONTRAINDICATIONS: Comatose or severely depressed states; hypersensitivity to the drug.
WARNINGS: Safe use during pregnancy or lactation has not been established; weigh potential benefits against possible hazards. Not recommended for use in children under 16. May impair mental and/or physical abilities especially during early therapy; warn ambulatory patients about activities requiring alertness and concomitant use of alcohol or other CNS depressants. Not recommended for management of behavioral complications in mentally retarded patients.
PRECAUTIONS: Use with extreme caution in patients with a history of convulsive disorders; use with caution in patients with cardiovascular disease or in those with glaucoma or a tendency to urinary retention particularly when on concomitant anticholinergic medication. Loxapine has an antiemetic effect in animals which might occur in man masking signs of overdosage of toxic drugs and obscuring intestinal obstruction or brain tumor. Since possible ocular toxicity cannot be excluded, observe carefully for pigmentary retinopathy and lenticular pigmentation. Slightly higher incidence of extrapyramidal effects possible following IM administration.
ADVERSE REACTIONS: *CNS effects,* other than extrapyramidal, infrequent. Mild drowsiness may occur at beginning of therapy or upon dosage increase, usually subsides with continued therapy. Sedation, dizziness, faintness, staggering gait, muscle twitching, weakness and confusional states have been reported. Extrapyramidal reactions often occur early in treatment, manifested by Parkinson-like symptoms (tremor, rigidity, excessive salivation, masked facies, akathisia); controllable by dosage reduction or antiparkinson drugs at usual dosages. Dystonic and dyskinetic reactions, while less frequently occurring, may be more severe, requiring dosage reduction or temporary withdrawal plus appropriate counteractive drugs. *Persistent Tardive Dyskinesia* may appear during prolonged therapy or following discontinuance, the risk greater in the elderly, especially females, on high dosage. Symptoms, persistent and in some patients apparently irreversible, are characterized by rhythmical involuntary movement of the tongue, face, mouth and jaw sometimes accompanied by involuntary movement of extremities. Since there is no known effective treatment, discontinue all antipsychotic drugs if symptoms appear. Reinstitution of treatment, increased dosage or switching to another agent may mask syndrome. The syndrome may not develop if medication is stopped when fine vermicular movements of the tongue first appear. *Cardiovascular Effects:* Tachycardia, hypotension, hypertension, lightheadedness and syncope. ECG changes, not known to be related to loxapine use have been reported. *Skin:* Dermatitis, edema of face, pruritus, seborrhea. Possible photosensitivity and/or phototoxicity; skin rashes of unknown etiology seen in a few patients in hot summer months. *Anticholinergic:* Dry mouth, nasal congestion, constipation, blurred vision (more likely to occur with concomitant use of antiparkinson agents). *Other:* Nausea, vomiting, weight gain or loss, dyspnea, ptosis, hyperpyrexia, flushed facies, headache, paresthesia, polydipsia. Rarely, galactorrhea and menstrual irregularity of unknown etiology.

References: 1. Thomas JL: Loxapine oral liquid concentrate in the treatment of young adult patients with acute schizophrenic symptoms, *Curr Ther Res* 25:371-377, March 1979. **2.** Sharma T: Rapid response to loxapine in acute schizophrenia, *Curr Ther Res* 25:366-370, March 1979. **3.** Serban G: Loxapine in acute schizophrenic disorder, *Curr Ther Res* 25:139-143, January 1979. **4.** Zisook S et al.: Loxapine succinate (LOXITANE®) in the outpatient treatment of acutely ill schizophrenic patients, *Curr Ther Res* 24:415-426, August 1978.

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Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy, observed primarily in patients receiving larger than recommended doses, is characterized by diminution of visual acuity, brownish coloring of vision, and impairment of night vision; the possibility of its occurrence may be reduced by remaining within recommended dosage limits. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving), and increase dosage gradually. Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension since phenothiazines may induce a reversed epinephrine effect on occasion. Daily doses in excess of 300 mg should be used only in severe neuropsychiatric conditions.

Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; rarely, nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—ECG changes (see *Cardiovascular Effects* below). *Other*—Rare cases described as parotid swelling.

It should be noted that efficacy, indications and untoward effects have varied with the different phenothiazines. It has been reported that old age lowers the tolerance for phenothiazines; the most common neurologic side effects are parkinsonism and akathisia, and the risk of agranulocytosis and leukopenia increases. The following reactions have occurred with phenothiazines and should be considered whenever one of these drugs is used. *Autonomic Reactions*—Miosis, obstipation, anorexia, paralytic ileus. *Cutaneous Reactions*—Erythema, exfoliative dermatitis, contact dermatitis. *Blood Dyscrasias*—Agranulocytosis, leukopenia, eosinophilia, thrombocytopenia, anemia, aplastic anemia, pancytopenia. *Allergic Reactions*—Fever, laryngeal edema, angioneurotic edema, asthma. *Hepatotoxicity*—Jaundice, biliary stasis. *Cardiovascular Effects*—Changes in terminal portion of electrocardiogram, including prolongation of Q-T interval, lowering and inversion of T-wave, and appearance of a wave tentatively identified as a bifid T or a U wave have been observed with phenothiazines, including Mellaril (thioridazine); these appear to be reversible and due to altered repolarization, not myocardial damage. While there is no evidence of a causal relationship between these changes and significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients showing characteristic electrocardiographic changes while taking the drug. While proposed, periodic electrocardiograms are not regarded as predictive. Hypotension, rarely resulting in cardiac arrest. *Extrapyramidal Symptoms*—Akathisia, agitation, motor restlessness, dystonic reactions, trismus, torticollis, opisthotonus, oculogyric crises, tremor, muscular rigidity, and akinesia. *Persistent Tardive Dyskinesia*—Persistent and sometimes irreversible tardive dyskinesia, characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and sometimes of extremities may occur on long-term therapy or after discontinuation of therapy, the risk being greater in elderly patients on high-dose therapy, especially females; if symptoms appear, discontinue all antipsychotic agents. Syndrome may be masked if treatment is reinstituted, dosage is increased, or antipsychotic agent is switched. Fine vermicular movements of tongue may be an early sign, and syndrome may not develop if medication is stopped at that time. *Endocrine Disturbances*—Menstrual irregularities, altered libido, gynecomastia, lactation, weight gain, edema, false positive pregnancy tests. *Urinary Disturbances*—Retention, incontinence. *Others*—Hyperpyrexia; behavioral effects suggestive of a paradoxical reaction, including excitement, bizarre dreams, aggravation of psychoses, and toxic confusional states; following long-term treatment, a peculiar skin-eye syndrome marked by progressive pigmentation of skin or conjunctiva and/or accompanied by discoloration of exposed sclera and cornea; stellate or irregular opacities of anterior lens and cornea; systemic lupus erythematosus-like syndrome.

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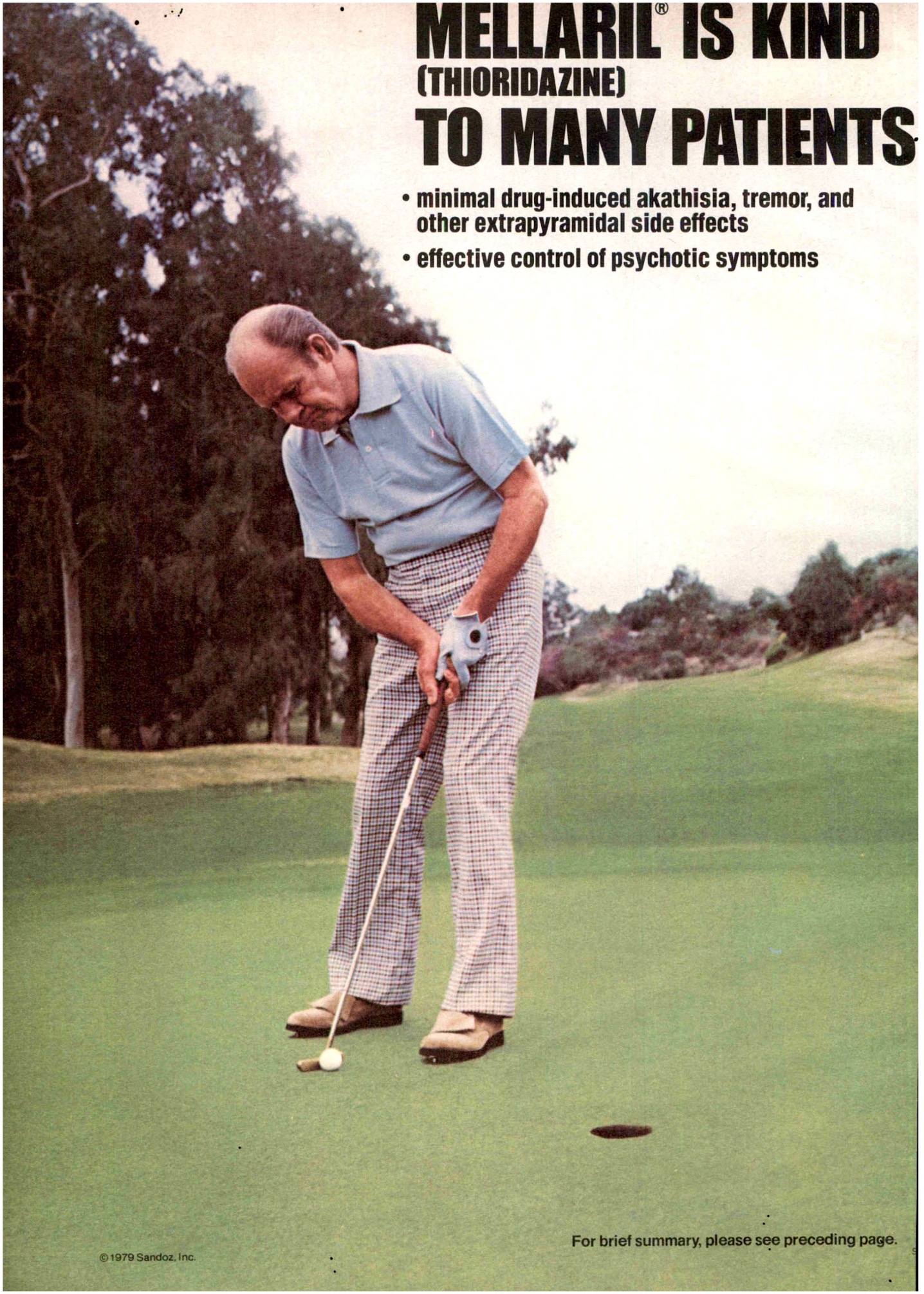
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THE AMERICAN JOURNAL OF PSYCHIATRY

Pathological Grief and the Activation of Latent Self-Images

BY MARDI J. HOROWITZ, M.D., NANCY WILNER, CHARLES MARMAR, M.D., AND JANICE KRUPNICK, M.S.W.

The authors studied the case material for patients treated with either psychoanalysis or brief therapy to examine the basis for the various states of pathological grief after bereavement. They view these states as intensifications or unusual prolongations of states found in normal grief and describe them in terms of the reemergence of self-images and role relationship models that had been held in check by the existence of the deceased person. This conclusion concerning preexisting mental schemata leads to an elaboration and partial revision of theories of regression, ambivalence, and introjection as causes of pathological grief.

For some people, mourning the loss of a loved one involves a process so unbearably painful, protracted, or tenaciously blocked that it can be described as pathological grief. Freud (1) and Abraham (2) attributed such states to preexisting ambivalence toward the deceased, followed after the death by internalization of the other and aggression directed toward the self. They described this process as leading to feelings of self-hatred and deflation of self-esteem. In this paper, we will elaborate on these alterations of the self-image in pathological grief. Our intention is not to present a comprehensive explanation of a multidetermined phenomenon but to focus on one process: the review of self-images and role relationship models initiated by a loss.

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DEFINITIONS

The terms "grief" and "mourning" have been used in many ways. In this discussion, "grief" refers to phenomena of subjective experience and behavior that occur after psychological recognition of a loss. "Mourning" refers to processes in which states of grief are eventually attenuated as the person recognizes and adapts to loss.

"Pathological grief" is the intensification of grief to the level where the person is overwhelmed, resorts to maladaptive behavior, or remains interminably in the state of grief without progression of the mourning process toward completion. "Pathological mourning" involves processes that do not move progressively toward assimilation or accommodation but, instead, lead to stereotyped repetitions or extensive interruptions of healing.

EARLY PSYCHOANALYTIC VIEWS

In a series of papers, Abraham (2, 3) and Freud (1) differentiated normal from pathological grief. Normal grief was characterized by painful dejection, loss of interest, and inhibition of activities. Pathological grief reactions included additional features, such as episodes of panic, hostility toward the self, regression to narcissistic self-preoccupation, and other signs of deflated self-esteem.

The explanation for these supplemental features of pathological grief included several components of pathological mourning. A preexistent ambivalent rela-

tionship with the deceased led to self-hatred after the loss because of internalization of attributes of the lost object into the ego and super ego. Aggressive drives, now directed toward the self, led to self-blame, self-hate, and self-disgust.

This early work indicated the typical signs of pathology, the gradient between mourning and melancholia, the importance of the quality of the previous relationship, and the serious consequences of ambivalence.

Perhaps because of their pioneering nature, the works of Abraham and Freud suggested an excessive cleavage between normal and pathological grief. Subsequent field studies of bereavement experienced by nonpatient as well as patient populations indicate that hostility toward the self, exemplified as pathogenic by Abraham and Freud, is not uncommon in typical grief reactions of relatively random populations (4–8). States of inertia, hypochondriasis, numbness, and apathy have also been noted; these too may not be so unusual, intense, out of control, or prolonged as to be called pathological. Various normal and depressive states of mind were further clarified during the period when the theory of ego psychology was developed.

EGO PSYCHOLOGY OF DEPRESSIVE STATES

A state of sadness accompanied by dejection, loss of interest, and relative reduction of social activities has long been regarded as normal grief and does not merit a diagnosis of pathological grief or any other depressive syndrome. Depressive states take other and varied forms. In one state the person feels frighteningly sad, alone, and needy. In another, instead of feeling bereft and helpless he feels strongly hostile toward himself and others. In still another fairly distinct ego state, the person has feelings that are not so much those of fear or anger as they are feelings of despair, worthlessness, and defectiveness (9, 10). To these states of panic, rage, and deflated self-esteem Jacobson (11) and Schmale and Engel (12) added a fourth, which is much less intense in emotional tone and more hibernative or energy-conserving. This state is characterized by withdrawal and vague sensations of numbness, emptiness, and hypochondriacal concerns.

These depressive states have been explained according to object relations and cognitive-psychodynamic points of view. Internalization of early strained interpersonal relationships may leave a residue of self- and object representations conducive to depression when there is a regressive return to these forms precipitated by the stress of loss (13–15). Incomplete early separation from the mothering figure may render a person especially prone to such regressions in later life (16).

Not in conflict with such formulations but emphasizing an information-processing point of view, Bibring (17) suggested that entry into these different depressive states might be the consequence of particular cognitions, a concept recently elaborated on by Seligman (18) and Beck (19). They observed that the states of agitated sadness and panicky helplessness were initiated by a train of thought that moved from seeing oneself as strong and competent to seeing oneself as weak and helpless. The rage states—anger directed both outward and toward the self—were seen as shifts in conceptualization from the self as good to the self as evil. The states of self-disgust were the result of an alteration in the sense of self from lovable to unworthy or inferior.

Bereavement could initiate these trains of thought about the meaning of the loss of a relationship or the failure to prevent that loss. These implications can shift the sense of self from that of being strong, concerned, and worthwhile to self-images of being weak, incompetent, and uncaring. The various depressive states could be viewed as the results of experiencing such thoughts consciously or unconsciously.

Four depressive states have been described and can be labeled by predominant emotional tone: fear, rage, shame, and hibernation. All of these states occur in normal grief and do not warrant diagnoses. Pathological grief, as already mentioned, is the intensification of such states to a level where the person feels overwhelmed and either resorts to maladaptive behavior or remains interminably in that state. We studied the psychodynamics of people with pathological grief to understand why such intensification or prolongation occurs.

METHOD OF CLINICAL INVESTIGATION

Material for study was available from two contexts. One involved the use of process notes from the psychoanalyses of 3 adults with character neuroses. The patients were 2 women and 1 man who mourned the death of a parent while in treatment, although the loss had occurred years before, during critical developmental periods. The second context was time-limited (12 sessions) brief psychoanalytically oriented therapy of adults with symptoms at the neurotic level after the recent death of someone close to them.

The latter context included systematic evaluations before and after therapy, with audio and video recordings and transcriptions of many of the therapy and evaluation hours. This material was collected after written informed consent was secured from more than 50 subjects participating in a program of clinical research and therapy. Review of the case material for about 25 of these patients led to the present report. Virtually all of the patients were white, middle-class, West Coast Americans with at least a high school education. A little less than three-quarters of the patients were women; the majority of the entire group were between 20 and 40 years of age.

CONFIGURATIONAL ANALYSIS

Detailed case review by configurational analysis evolved from the kind of formulations made during psychodynamically oriented case conferences in major training centers. It consists of 10 systematic steps with related formats, described elsewhere in detail (20). Materials included case summaries, process notes, video and audio recordings when available, and transcripts of selected interviews. The initial descriptions of the problems are amplified first by a focus on observable, reportable experience and behavior as elaborated by analysis of the states the patient is experiencing. Then the self-image and role relationship themes that characterize important recurrent states are modeled through an analysis of the content of each state. These themes, usually expressed as dyadic or triadic structures, are traced through the person's developmental history, the immediate course of entry into problem states, and the period of change that may occur during and after psychotherapy. This is followed by formulation of the person's major active constellations of conflicted aims, ideas, and emotions. The related controls, defenses, and styles of information processing are included.

The problem states of these bereaved patients conformed well to the patterns of pathological grief described above. There were patients whose main complaints could be described as a frighteningly sad response to loss, those who experienced feelings of out-of-control rage after loss, and those who had a deflated or hibernative response to loss. Although each patient might identify one such state as a major problem, several problem states were manifest in all of them. Patients varied in their response to experiencing similar states; in some states they felt more out of control, vulnerable to impulsive action, or distressed than in others. They were able to work their way out of one state and into another with various levels of ease or difficulty. Although all of the patients had the same set of states, they differed in terms of which states were pathological or caused the most problems.

What seemed pathological was not the emotional or ideational content of a state but its intensity or prolongation. An experience that was too intense and therefore pathological was one that crossed the person's usual threshold of tolerance. It was not merely painful but too painful; it was not just threatening but too threatening.

The states themselves were seldom new varieties of experience, although the current episode was often more intense or protracted than previous episodes. Patients could remember times before the loss when they had had shorter and less intense periods of panic, self-hatred, deflation, or hibernation. Infrequently, a new state emerged that the patient did not remember experiencing before the loss. In such instances the state tended to be a repetition of an experience that had emerged for the first time during the loss itself. Examples of such new states might include the recurrence of a depersonalized dazed state that had accompanied initial recognition of the death, a hyperalert excitation after viewing a traumatic sight, or a personal repetition of experiences of the deceased that the patient had observed before the death occurred.

SELF-IMAGES AND ROLE RELATIONSHIP MODELS

Certain self-images and role relationship models predominate in each state; we infer that these are organizing principles which are partly responsible for the repetition of a given state over time. We will describe these models of self and others in sequence. Our observations confirm earlier impressions and add little that is not contained in the clinical literature. What expands the literature is an attempt to clarify the process of reversion to preexistent self-images.

The essential point is that recent internalization of ambivalent attitudes toward attributes of the lost person is not the major reason for states of pathological grief. Although these new identifications and attitude formations are important processes, a major cause of pathological grief appears to be the reemergence of earlier self-images and role relationship models. These were established before the loss but were held in check by a more positive relationship.

News of the loss activates these usually dormant organizers of mental life; it instigates thought processes aimed at examining the implications of the news. Inner models are revised so that they conform to new realities. This involves a completion tendency that leads to repetitive and sometimes intrusive thinking (8). In the course of such ideation, there is review of the current primary model of attachment to the lost person as well as to all other models of attachment (21). Latent self-images and role relationships that are usually unconscious are actively contemplated, whether awake or dreaming, by primary and secondary process modes of review (22).

When there is activation of previously conflicted, troublesome, dissociated role relationship models, the process evokes unusually intense negative effects. Defenses are instigated as a feedback process to reduce pain and threat, with possible interruption of the working-through process. Persistent tendencies to repetition produce intrusive episodes; excessive defenses produce experiences of numbness and avoidance. This combination results in unusually intense and protracted states.

In our sample of patients we found that troublesome preexistent self-images and role relationship models were common precursors to pathological grief. Before reviewing them, we will discuss a model of normal mourning for purposes of comparison.

NORMAL MOURNING

Normal mourning is a theoretical construct, a statement about routes to a hypothetical and ideal acceptance of separation. In this prototype the relationship with the deceased was one in which both self and other were regarded as competent, giving, caring, and responsible. Before the loss this role relationship model fostered states of mutuality and contentment.

In normal mourning the representations remain realistic after the loss. The self is experienced as bereaved but competent to tolerate sadness, and the other is experienced as lost but remembered. The interaction is one of pining, preoccupation, and symbolic search for repair (23). Gradual acceptance of the lost attachment as part of the past, as no longer present, or as part of the future (except in some new form) attenuates the state of sadness, pining, and preoccupation. During the initial period of intense sadness, emptiness, and fear of being alone, the experience is made tolerable by support from others and through intermittent periods of calm achieved by moderate use of denial.

In reality, in addition to the ideal resolution described above, mourning usually includes transition through other states, such as fear, rage, and deflation. The difference between normal and pathological grief is that in normal grief the transition—however painful—is not overwhelming, interminable, or prematurely interrupted. Instead, each model of attachment is reviewed and ties are detached in terms of expectancy for the future. Competent self-images and readiness for a new attachment are stabilized. Personal suffering is accepted, and the lost attachment is retained in the present as a rewarding memory.

PREDISPOSITION TO FRIGHTENINGLY SAD RESPONSE TO LOSS

Reports by clinicians about people who are predisposed to a frighteningly sad response to loss define the preexistent role relationship as one of dependency and orality (24). That is, the person is seen as having relied on the strong, caring other for nurturance. With loss of the other, the person remains dependent and the need becomes frightening. The most frequent self-image in these states is that of a weak, abandoned, and needy waif in desperate hope of rescue, with intense fear that such rescue will not occur. On closer study, however, additional impressions are gained. The preexistent relationship was often one in which both the self and the other were conceptualized as strong; representations of the self as weak were held in check by these people.

The person predisposed to overwhelming, frighteningly sad states had several role relationship models. In two of these role relationship models, the self was weak, either in relation to a strong nurturing other or to an abandoning or lost other. To submerge this traumatic view of the needy self abandoned by a caretaker, the patient developed a role relationship model in which the other served not only as supplier but as stabilizer of the strength of the self. With recognition of the death, the patient was forced to once again contemplate his vulnerability (16, 25).

The patient's self-image shifted back, from that of a strong person well sustained by the relationship with a strong other to the preexistent structure of a weak, helpless waif supplicating in vain for rescue by a lost or abandoning person. The state associated with this role relationship model was more than sad yearning; it was an agitated, desperate, and intolerable sadness that led to insistent demands for help. If help seemed unavailable, there was transition to a state of dullness and defeat (12).

The preexistent self-image as a weak, helpless waif was the structural basis for what Seligman (18) termed "learned helplessness" and Beck (26) referred to as "depressogenic schemata and premises." The developmental precursors in separation and attachment have been well reviewed by Bibring (17), Bowlby (21), and Rubenfine (27).

After the loss of an important relationship, any person will feel frightened and sad as he experiences yearning that cannot be fulfilled. Any person will have a shift to needy, helpless self-images, but these self-images may be less desperate in quality, less discrepant with other self-images, and less compelling as organizers of information than the needy self-images of a person with conflicts or developmental defects in this area. Most people are more readily able to complete a review of the situation, estimate the degree of helplessness, and consequently experience restoration of morale. Competent self-images may become more stable without as much reliance on another person to maintain a view of the self as strong. The predisposed person who has more peremptory self-images as weak and helpless will review the implications of the loss more intensely and will not as readily shift back to a competent self-image.

Of course, other factors, such as fear of sadness and of feeling too weak to tolerate mourning, contribute to the panic of such frighteningly sad states. However, the interrelationship of self-images and role relationships with these other aspects of mental process is beyond the scope of this paper.

RAGE STATES AND DEFLATED STATES

Any attack on the integrity of the self evokes a hostile response. One response to the loss of a relationship is to experience it as an attack on the self with rage at the source of the attack. The attacker can be posited to be the self, the deceased, others involved in the death, or the agents of destiny. People exempted

from the loss may be hated because they do not have to experience the burdens of this fate.

Among the patients whose rage states became a problem in the material we reviewed, the most common images were oscillations between the self as a betrayed needy person and the self as an evil, greedily consuming, destructive person. These self-images were developed long before the loss. They had been held in check by development and stabilization of images of the self as kind and good. This stabilization occurred because the relationship before the loss was construed as between people who were kind and good to each other. The deceased protected the self from evil, in effect, because exchanges of a loving nature maintained a state of mutuality.

In one version of the implications of the separation, the deceased was seen as an evil deserter who willfully betrayed the needy self. This injury led to states of explosive rage, with hostility diffusely expressed toward anyone within range as well as toward the deceased. Role reversal was also very common. With recognition of the rage, the self became the one who was evil, a destroyer of the deceased, who was now cast as victim. This led to states of guilt, remorse, and self-loathing, with a fearful expectancy of accusation or punishment.

Oscillation or reversal of these role models led to shifts between anger and guilt. These shifts in state resulted from an undoing defense aimed at reducing the intensity of either affect. Although this served defensive purposes (28), there were times when transitions to impulsively self-destructive states of mind occurred. Suicidal acts were products not only of self-hatred and self-punishing states but also of despair over weakness, loss of coherence of the self, ideas of merger with the victim, and wishes for retribution from others (by making them mourn the lost self) (29).

The patients who developed excessively intense or prolonged deflated states had preexisting tendencies to self-images as worthless, defective, or disgusting. A relevant relationship model was one between a scornful superior critic and a contemptible, worthless person. As with most role relationship models, the self could be placed in either position.

The relationship before the death had components of admiration and appreciation that stabilized a worthwhile self-image. Loss of the relationship altered this stability and a worthless self-image emerged, organizing experienced thought and emotion. A worthless self-image was also evoked when the self was blamed for being unable to prevent the loss or felt scorned by the other. The idea of having been left because of defects of the self led to feelings of deflation, shame, and dejection accompanied by withdrawal. In an additional defensive shift, there was movement from such relationship views to illusions of self-sufficiency. As is common in such insulation, no joy and little pain was felt; the patient was blank, deadened, and numb (30).

The loss of admiration and appreciation was not limited to loss of a real, present situation. It involved loss of a projected future. For example, in some instances the deceased had been approving in the past but had devalued the patient more recently. The patient then cherished a belief that approval would be restored in the future. Death destroyed that projection, and the patient shifted to worthless self-images.

INTERNALIZATION: SELF WHERE OTHER WAS

If a person fantasizes restoration of the lost other and gradually surrenders these fantasies as unrealistic, a form of learning may take place in which he revises his models. The self may become like the other. At first, whole aspects and traits of the other may be mimicked to gain essential parts. Then, by conscious contemplation, the person may synthesize the important elements of what was lost, so that they are valued and sought in future relationships (31).

For example, the person may no longer expect that the function served by the deceased will automatically be served by new attachments. He learns to fulfill both roles of the lost relationship, to be the strength for others who might otherwise be weak, the good for others who might otherwise be evil, the appreciator for those who might otherwise feel worthless. Such processes lead to the mellowing of interpersonal relationships, which is a positive consequence of grief.

DRIVES AND COGNITIVE PROCESSING

We have paid little attention to drive theories, which are prominent in the pychoanalytic literature on this topic. Briefly, drives can be expressed in derivative forms as responsible for the activation of particular role relationship models and for the intensity of related aims (8, 32, 33). Libidinal or aggressive drive development, as predisposition, can be described in terms of relationship models that have been well established and the priority they have been given. Fixation can be discussed in terms of capacity, readiness, or lack of readiness to give up a specific role relationship model even when retention of the model involves loss of reality-based thinking.

Beck and Kovacs (19, 26, 34–36) have been prominent observers and theorists of cognitive processes in depressed people. For example, premises such as "If I am not loved I cannot go on living" have been seen as ideas that anchor a person to a depressive state. Our experience with the patients whose case material we reviewed supports Beck's assertions of the importance of the cognitive processes of devaluation of the self, the exaggeration of impediments to contented living, and viewing of the future as hopeless. The self-images and role relationship models de-

scribed here are conceptualized as the usually unconscious formats of meaning that influence the organization of such thoughts. Kovacs and Beck (36) have been developing the same view with their concept of depressogenic schemata.

SUMMARY

We have described several states that constitute pathological grief when they are excessively intense or prolonged, together with the prototypical self-images and role relationship models that organize each state of mind. Early traumatic experiences, such as parental loss during childhood, or other developmental strains result in unusual salience of such models during adult life, even when compensatory relationships have developed and more competent self-images have been stabilized.

The compensatory relationship, when present in reality or fantasy, stabilizes the advanced structures and promotes active, harmonious mental states. When such relationships are lost, a person reviews his repertoire of self-images and role relationship models. Many bereaved people experience transient states of frightening sadness caused by weakened views of the self; many experience states of hatred and of deflation arising from evil or worthless self-images. Those predisposed to pathological grief cannot readily complete review of such self-images and relationship models; instead, the state during the review becomes unusually intense and interminable or excessive controls prevent review of activated role relationship models, so that mourning is never completed.

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Tardive Dyskinesia: Summary of a Task Force Report of the American Psychiatric Association

BY THE TASK FORCE ON LATE NEUROLOGICAL EFFECTS OF ANTIPSYCHOTIC DRUGS

Abnormal movements associated with the prolonged use of antipsychotic drugs (tardive dyskinesia) occur in at least 10%–20% of the patients at risk; prevalence is higher among the elderly. The cause is unknown, but increased sensitivity to dopamine in the basal ganglia may contribute to the pathophysiology. Many treatments have been evaluated; none is satisfactory. While the problem is serious, an alarmist view is unwarranted, especially since many cases are detected early and improve spontaneously. The best approach currently is to use antipsychotic drugs thoughtfully for clear indications, the best supported of which, scientifically, is chronic schizophrenia. There is a search for new agents with much less adverse neurologic effect but with adequate antipsychotic efficacy. This article summarizes an APA Task Force Report.

The past two decades have produced revolutionary changes in the practice of psychiatry and in psychiatric research. Particularly striking changes have occurred in the pattern of care of psychotic and other severely disturbed patients. The introduction of effective antipsychotic drugs in the early 1950s contributed to the changes in clinical management of such patients and to diminished emphasis on long-term institutional care. The antipsychotic drugs lead to rapid control of psychotic symptoms and behavior in a wide variety of psychotic illnesses. Furthermore, such characteristics can have useful long-term preventive or ameliorative effects; however, these long-term effects have been scientifically verified only in schizophrenia.

Virtually all of the antipsychotic agents currently used in American medical practice exert undesirable neurological effects. Hence the term "neuroleptic" is often applied to this class of drugs. Most of the early neurological effects (e.g., dystonias, parkinsonism, akathisia, or motor restlessness) are spontaneously reversible or are managed by the temporary addition of an antiparkinsonian drug (1) (see table 1). However, in recent years it has been realized that there may be later development of certain neurological effects, including abnormal movements, usually of a choreoathetotic or dystonic type. These late reactions are referred to as tardive (late and gradually appearing) dyskinesia (2, 3). They can be so mild as to escape notice or can be severely disabling. In an uncertain proportion of cases, these movements persist for many months, sometimes indefinitely, even when the suspected offending agent is removed. Thus, their occurrence in association with long-term use of neuroleptic agents has become a cause of increasing concern to the medical profession and the general public.

CLINICAL DESCRIPTION, DIFFERENTIAL DIAGNOSIS, AND CLINICAL EVALUATION

Tardive dyskinesia is manifested by a wide variety of involuntary movements including orolingual dyskinesia, chorea, athetosis, dystonia, tics, and facial grimacing but excluding rhythmic tremor. The severity and extent of dyskinesias range from isolated orolingual dyskinesia to widespread and sometimes disabling dystonia. Age may influence the topographic distribution of involuntary movements; for example, oral-lingual movements are especially common in the elderly. Tardive dyskinesia may appear after months or years of treatment with neuroleptic drugs and typically worsens during or after drug withdrawal. In some cases, dyskinesias that appear during drug withdrawal subside within several weeks and are referred to as withdrawal dyskinesias (4, 5). It is possible, although unproven, that these cases represent an early and reversible form of tardive dyskinesia.

It is important to emphasize that a wide range of ab-

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Reprints of this article are not available.

This summary highlights some of the clinically pertinent conclusions of the American Psychiatric Association's Task Force Report. Further details on clinical and preclinical aspects of the topic, as well as additional literature references, will be found in the full report.

Copies of the Task Force Report are available from Publications Sales Department, American Psychiatric Association, 1700 18th St., N.W., Washington, D.C. 20009. The cost of the 204-page report is $11.00 per copy. *Orders must be accompanied by payment.*

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TABLE 1
Neurological Side Effects of Neuroleptic-Antipsychotic Drugs

Side Effect	Features	Period of Greatest Risk	Proposed Mechanism	Treatment
Acute dystonia	Spasm of muscles of tongue, face, neck, back; may mimic seizures; *not* hysteria	1–5 days	Unknown	Antiparkinsonian agents are diagnostic and curative (administered intramuscularly or intravenously, then orally)
Parkinsonism	Bradykinesia, rigidity, variable tremor, masked expression, shuffling gait	5–30 days	Dopamine blockade	Antiparkinson agents help (administered orally)
Akathisia	Motor restlessness; *not* anxiety or agitation	5–60 days	Unknown	Reduce dose or change drug; antiparkinsonian agents or benzodiazepines may help
Tardive dyskinesia	Oral-facial dyskinesia; choreoathetosis	Months–years (worse during or after drug withdrawal	May be due to functional excess of dopamine	Prevention is best; treatment is unsatisfactory
"Rabbit" syndrome	Perioral tremor (may be a late variant of parkinsonism)	Months–years	Unknown	Antiparkinsonian agents may help

normal movements that may be called tardive dyskinesia is encountered clinically. This fact complicates the definition and diagnosis of tardive dyskinesia. One of the difficulties arising from currently heightened awareness of tardive dyskinesia is greater sensitivity to minor degrees of abnormal movements, some of which may not be related to neuroleptic drug use and do not represent tardive dyskinesia. These abnormalities include minor movements that may be difficult to distinguish from habit spasms, other tics of unknown cause, psychotic mannerisms, manifestations of senescence, or even normal movements associated with ill-fitting dentures. A major problem in diagnosing tardive dyskinesia is determining the baseline of spontaneous (not neuroleptic-induced) dyskinetic movements in normal populations, the geriatric population, and the schizophrenic population; the latter two groups apparently have a greater incidence of spontaneous movement abnormalities.

Factors to be considered in differential diagnosis include schizophrenic stereotyped movements, reversible neuroleptic syndromes, and dyskinesias induced by other drugs. In addition, one should consider several other neurologic disorders that produce involuntary movements (see appendix 1).

Although it is essential that one be vigilant for early identification of tardive dyskinesia, it is important not to overlook other disorders in which psychiatric symptoms and involuntary movements may coexist and have no relationship to tardive dyskinesia. Once tardive dyskinesia is identified, efforts to rate its severity in a reliable manner are essential for future management. A number of methods exist (6), the most practical of which are multiple-item rating scales (7) topographically arranged by body area. These include the Abnormal Involuntary Movement Scale (AIMS) (8), developed at the National Institute of Mental Health, and a similar but more detailed scale developed by Simpson and colleagues (9).

TABLE 2
Prevalence of Tardive Dyskinesia, in Various Degrees of Severity, Among Chronic Schizophrenic Inpatients and Outpatients Treated with Neuroleptic Drugs[a]

Rating of Severity[b]	Approximate Percent of Patients
2.0	68
2.5	44
3.0	27
3.5	12
4.0	4

[a]Data are based on surveys of over 500 patients diagnosed as schizophrenic and evaluated in 1977 and 1978 at the Harlem Valley Psychiatric Center in Wingdale, N.Y., by Dr. J.M. Smith and his colleagues (15), who provided the data abstracted above.
[b]Severity was evaluated using the NIMH Abnormal Involuntary Movement Scale (AIMS) (8), examples of which appear in the Task Force Report. A rating of 4 in any one body area (rare) represents severe dyskinesia.

RISK FACTORS, PREVALENCE, AND OUTCOME

Epidemiologic studies strongly support an association between the use of neuroleptic drugs and the development of persistent as well as relatively transient forms of tardive dyskinesia (10). While there is some evidence that prolonged exposure or exposure to large total amounts of a drug may be a contributing factor (11, 12), this is not established. There is a very weak correlation between average daily drug dose and tardive dyskinesia (13). Older patients and women may be at higher risk and may have a poorer prognosis for eventual remission (14, 15). The aging brain may have an increased likelihood of neuroleptic-related tardive dyskinesias, especially of the oral region. Other specific predictors of risk (such as type of drug and dose,

TABLE 3
Studies of the Remission of Tardive Dyskinesia After Drug Termination

Study	Patients			Length of Follow-Up (months)
		Improved		
	N	N	%[a]	
Hershon and associates (20), 1972	23	0		4
Hunter and associates (21), 1964	13	0		3–18
Paulson (22), 1968	33	0		3
Edwards (23), 1970	19	1	5	about 12
Crane (24), 1971	39	3	8	6–24
Degwitz (25), 1969	273	52	19	7–10
Uhrbrand and Faurbye (26), 1960	17	6	35	4–22
Turunen and Achte (27), 1967	26	10	38	—
Yagi and associates (28), 1976	19	10	53	12–24
Doller-Wojcek and associates, 1980[b]	13	7	54	6–18
Jeste and associates (29), 1979	21	12	57	3–13[c]
Quitkin and associates (30), 1977	12	11	92	1–24
Total or mean	508	112	22	

[a]The average of remission rates is 22%, although most recent studies cite even higher rates. For the studies reported since 1970, the mean rate is 30%.
[b]J. Doller-Wojcek, A.J. Gelenberg, R.A. LaBrie, and associates: Outcome of tardive dyskinesia: unpublished observations at the Massachusetts General Hospital, Boston, 1980.
[c]Not drug-free for more than 3 months, but followed up to 1 year.

iming of exposure, prior acute reversible extrapyramidal symptoms, use of antiparkinsonian agents, or sex) are *not clinically useful*. Moreover, it is clear hat similar exposure to neuroleptics does not produce ardive dyskinesia in all patients and that there are no groups (as defined, for example, by age or drug history) which are exempt from risk.

It is nearly impossible to specify the precise prevalence rates of tardive dyskinesia since there is no currently accepted standard for diagnosis (14, 15) (see able 2). Certainly, clinically appreciable cases occur n at least 10%–20% of the patients exposed to neuroleptic drugs for more than a year, and the rate is probably higher among the elderly. However, both the medical profession and the public should be aware that profoundly disabling, irreversible dyskinesias are uncommon. Some epidemiological studies suggesting a high prevalence of tardive dyskinesia (16, 17) may reflect an increasing sensitivity to minor degrees of possible movement abnormality. Even in the elderly, studies show that the prevalence of spontaneous buccolinguomasticatory movement abnormalities is close to that found in neuroleptic-medicated geriatric patients (18, 19). At present, it is impossible to say whether patients showing slight hyperactivity of the tongue and slight choreic movements of the fingers have tardive dyskinesia. Some abnormalities of posture and movement were reported in schizophrenic patients before the use of neuropleptics (1). Available studies have applied different criteria and have reported vastly different prevalence rates of tardive dyskinesia, ranging from less than 10% to over 50% (10, 16, 17). Fortunately, an increasing number of patients are being reported who appear to undergo spontaneous remission, usually within a period of months but up to 1–2 years, after discontinuing or decreasing the dose of the suspected offending agent; one-fourth to one-half of the patients may improve markedly within a year (20–30)[1] (see table 3).

Even if the "true" prevalence of tardive dyskinesia is between 10% and 20% of the patients treated with neuroleptic drugs for more than a year, and even if many of these patients eventually improve spontaneously after discontinuing treatment, the problem is clearly a significant one. Moreover, even mild choreoathetosis due to neuroleptic treatment is a valid cause of concern and encourages the development of safer and more selective antipsychotic drugs. On the other hand, considering the present state of knowledge, it would be an exaggeration to conclude that serious and profoundly disabling degrees of irreversible tardive dyskinesia commonly occur in current clinical practice.

ETIOLOGY AND PATHOPHYSIOLOGY

An open-minded attitude concerning the etiology of tardive dyskinesias is called for. Nevertheless, at the present time the conclusion seems inescapable that an important and relatively selective action of the antipsychotic-neuroleptic drugs is to block the actions of dopamine as a neurotransmitter in various regions of the central nervous system (CNS) (31–33). Acute extrapyramidal and sustained neuroendocrine side effects of these agents (such as increased prolactin secretion) are, in part at least, reflections of this action in the basal ganglia and hypothalamus or pituitary (34),

[1]J. Doller-Wojcek, A.J. Gelenberg, R.A. LaBrie, and associates: Outcome of tardive dyskinesia: unpublished observations at the Massachusetts General Hospital, Boston, 1980.

respectively. Antipsychotic effects may reflect antidopamine effects in limbic or cortical portions of the forebrain, although this hypothesis remains tentative (35, 36). There is also excellent evidence that prolonged exposure to antidopamine drugs can induce a variety of secondary and partially compensatory adjustments in the physiology and biochemistry of dopamine neurons and cells with which they interact in the animal or human CNS (33). Among these adjustments, some tend to *increase* the effectiveness of dopamine as a neurotransmitter, particularly in the basal ganglia. These effects may help to explain the clinical observation that the risk of acute extrapyramidal reactions diminishes in time, as the risk of tardive dyskinesia increases (1, 3). These effects are the basis of a "dopamine supersensitivity" hypothesis in tardive dyskinesia (37, 38). Further support for this hypothesis is the clinical pharmacologic evidence suggesting that a functional overactivity of extrapyramidal mechanisms mediated by dopamine is an important aspect of the clinical pathophysiology of tardive dyskinesia (see appendix 2). An explanation of the prolonged and even irreversible course of some cases of tardive dyskinesia awaits further research, especially as it suggests that irreversible neurotoxic or degenerative effects of neuroleptic agents may occur. However, these effects have not been demonstrated in postmortem neuropathological studies of animal or human brain tissue (39, 40).

While the precise cause of tardive dyskinesia remains uncertain, the study of neurological, behavioral, and endocrinological effects of neuroleptic agents on the CNS has contributed to an improved understanding of their actions, at least equal to that of many other drugs used in medicine (34, 41, 42). Moreover, insights arising from studies of the antidopamine effects of antipsychotic drugs in various brain regions promise to lead to the development of less neurotoxic but effective antipsychotic agents (42).

For the future, more work can and should be done to study the possible relationship of neuroleptic drug effects and the tardive dyskinesia syndrome. It would be helpful to have more epidemiological studies in countries where antipsychotic drugs are still not used extensively or are used in consistently different average daily doses than in the United States (43, 44). Dyskinesias follow treatment with neuroleptic-antipsychotic agents after widely differing periods of time, and they manifest various anatomical patterns and degrees of persistence. Thus, they may not necessarily represent a single disease with a unitary pathophysiology. While similar dyskinesias can occur independently in association with senile brain changes and many other overt or presumed states of CNS disease, usually there are no organic, metabolic, or neurological factors found on clinical examination that might contribute to the development of neuroleptic-related tardive dyskinesias. More attempts should be made to reproduce the dyskinetic phenomena by prolonged administration of drugs to intact animals, as well as to those with selective brain lesions, compromised cerebral circulation, or old age, and to make greater use of newer primate models of the disorder (45). There is also a need for further carefully controlled neuropathologic studies in animals and man after prolonged exposure to neuroleptic agents, using classical neurohistology and electron microscopy as well as newer techniques such as chemical assays of transmitters and their metabolites, biochemical and immunohistological assays of neuron-specific enzymes, and labeling assays for neurotransmitter and drug receptors. Such methods are applicable to postmortem human brain tissue for a more direct evaluation of the current pathophysiologic hypotheses concerning the brain's response to prolonged exposure to neuroleptics and development of tardive dyskinesia.

CONCLUSIONS AND RECOMMENDATIONS

Psychiatric chemotherapy must be based on rational guidelines that include objective evaluation of the illness to be treated, consideration of the patient's clinical history and response to treatment, an analysis of the risk-benefit ratio of any prospective agent for that patient, and a plan of action based on whether treatment is for an acute episode, sustained therapy of an active condition, or prevention of possible recrudescence of a successfully treated condition. These criteria are especially pertinent in decisions regarding sustained use (more than 6–12 months) of neuroleptic medications, since such treatment carries a risk of tardive dyskinesia.

It must be emphasized that the rational use of any form of treatment in psychiatry, as in general medicine, is based on careful diagnosis. Until recently, there was a tendency in the United States to use the term "schizophrenia" excessively broadly, almost as a synonym for "psychosis" or "severe psychiatric disorder." Thus, the diagnosis of schizophrenia was applied to many patients who would now be considered to suffer from an affective disorder, acute psychosis of uncertain type, or a personality disorder. Since diagnosis guides therapy, acceptance of a broad definition of schizophrenia probably tends to increase the use of long-term neuroleptic therapy. Prolonged neuroleptic therapy in cases other than chronic psychosis entails an increased risk of tardive dyskinesia when it may well be avoided by the use of alternative treatments. The importance of rigorous diagnosis is becoming increasingly recognized in this country and is a concern of APA, as witnessed by the current revision of its *Diagnostic and Statistical Manual (DSM-III).*

Short-Term Indications for Neuroleptics

In current American medical practice, neuroleptic medications are used for the short-term treatment of

acute psychotic episodes or for sustained maintenance or preventive therapy. Short-term use can reasonably be defined as treatment of less than 6 month's duration. The risk of persistent neurologic sequelae in this period of time appears to be small (except in rare cases of unusually sensitive patients, or perhaps with unusually high dosages). The primary indication for short-term use of a neuroleptic drug is acute psychosis, including 1) first clinical manifestation or exacerbation of schizophrenia, 2) paranoid states and other acute psychotic episodes of uncertain types (idiopathic conditions in which a categorical diagnosis is not possible) (46, 47), 3) mania, since lithium alone may not be adequate for acute reduction of manic excitement (48), 4) certain cases of toxic or organic psychosis (49), 5) childhood psychoses (50), and 6) as an adjunctive therapy in severe depressions with psychotic symptoms (51).

For treatment of manic excitement, when acute control is of primary clinical importance, neuroleptics typically are required in addition to lithium due to the latter's delayed onset of action. Evidence is mixed regarding the effectiveness of tricyclic antidepressants alone in depressions with prominent psychotic symptoms (51, 52); therefore, some patients with these disorders may be found empirically to benefit from the temporary addition of a neuroleptic agent. Nevertheless, antidepressant drugs and ECT remain the basic medical therapies with severe depression (53).

Other indications for the short-term use of neuroleptic drugs include states of acute agitation occurring in acute or chronic brain syndromes, the latter including dementia and mental retardation. Serious consideration should be given to the risks and benefits of neuroleptics compared with other drug treatments or nonpharmacologic management. In these two conditions, as with any psychotic or agitated state associated with cerebral dysfunction, patients are at greater risk for drug-induced delirium, but this risk may be less prominent with neuroleptic drugs than with sedatives (49). There are other occasional nonpsychiatric uses of neuroleptic drugs (54), including preoperative sedation, adjunctive uses in general anesthesia, and control of nausea or vertigo, as well as in the control of neurological or psychiatric manifestations of certain unusual disorders, such as Huntington's disease and Gilles de la Tourette's syndrome (55, 56). Finally, short-term treatment with neuroleptics has been used empirically in clinical psychiatric practice to ameliorate the symptoms of a variety of neurotic and characterologic disorders. The indications for this latter form of pharmacotherapy have not been developed in controlled studies; therefore, this use of neuroleptics requires critical clinical judgment in each case.

Long-Term Indications for Neuroleptics

Prolonged use (i.e., more than 6 months) of a neuroleptic medication requires careful evaluation of indications and risks. Such use is indicated for maintenance treatment of chronic psychotic disorders (mainly schizophrenia) in which the patient, although improved, is not symptom-free and is responsive to continued treatment. Sometimes prolonged neuroleptic treatment is used with Huntington's disease or Gilles de la Tourette's syndrome (55, 56), although the *long-term* efficacy and risk of such therapy require further study. While some controlled studies and substantial clinical experience support the short-term efficacy and safety of neuroleptic agents for conditions other than schizophrenia, as discussed above, there are virtually no controlled studies to verify long-term or maintenance use of these drugs with such conditions, and such use remains largely a matter of clinical judgment in individual cases. For example, it is recognized that in some cases of mania or depression with recurrent psychotic symptoms, neither lithium nor an antidepressant alone is an effective maintenance therapy and a clinical decision to try neuroleptic therapy might be considered

In general, maintenance treatment with neuroleptics is supported by scientifically sound data only for schizophrenia. The efficacy of maintenance neuroleptic medication in preventing psychotic relapse in chronic schizophrenic patients has been well demonstrated in approximately 30 controlled studies, in which an average of 56% (range=17%–100%) of nearly 3,500 patients who received placebo from 3 months to more than a year deteriorated clinically while only 17% (range=0%–49%) of the patients given a neuroleptic agent did so (57, 58). It should be noted that while this is a highly statistically significant mean difference, nearly half of the chronic schizophrenic patients did *not* deteriorate on a placebo. Studies evaluating outcome later than a year are rare. Some evidence suggests that effective doses of neuroleptic drugs during so-called maintenance treatment of schizophrenia may be lower than those in more acute phases of the psychoses (58).

In summary, commitment of a patient to treatment with neuroleptics for more than a few months requires 1) more than one acute psychotic episode without full return to optimal prepsychotic status, 2) objective evidence of continuing psychosis, or 3) good recovery but frequent recurrences that suggest the likelihood of further breakdowns, and 4) evidence of *responsiveness* to treatment. Antipsychotic drugs are to be avoided with patients who do not clearly need them or when they are not effective.

Management of Prolonged Neuroleptic Therapy

Any treatment has four potential outcomes: complete remission, partial relief, no effect, or clinical worsening. Maintenance neuroleptic treatment must be considered in light of these possible outcomes. Documentation of the continued indications and effectiveness of this treatment should be reviewed peri-

odically (at least every 3–6 months) and stated in the patient's records, with regular reconsideration of the benefits and the risk of tardive dyskinesia or other unwanted effects. At those times the patient's neurological and psychiatric status should be recorded. If there is no evidence of tardive dyskinesia, this observation is noted as a significant negative finding.

Even with schizophrenia, for which maintenance treatment has strong research support (57), deliberate and sustained effort must be made to maintain patients on the lowest effective amount of drug and to keep the treatment regimen as simple as possible. After a first acute psychotic episode of any type has clinically remitted, the dose of a neuroleptic drug should gradually be decreased and can usually be safely discontinued within several months. This recommendation is especially pertinent since it is rarely possible to diagnose schizophrenia with confidence based on a single psychotic episode. It is becoming increasingly clear that many acute psychoses eventually prove to be episodes of manic-depressive illness, which has not been demonstrated scientifically to benefit from prolonged maintenance neuroleptic treatment. Increasing the dose of a neuroleptic drug for a schizophrenic patient to unusually high levels can be supported only when smaller doses do not lead to clinical improvement and higher doses appear to be effective. The presumption in such therapy is that the degree of added benefit is thoughtfully balanced against possible additional risk of late complications.

For the special case of chronically hospitalized schizophrenic patients, it is recommended that neuroleptic medications be reevaluated at least yearly and preferably twice yearly. The dose of medication should be reduced by about 10% every 3–7 days until it has been totally discontinued or until clear signs of clinical worsening intervene. The patient should discontinue treatment for at least 2 weeks if his or her clinical status permits and if recent clinical experience does not suggest that such a trial is excessively dangerous. Such periodic evaluations serve two purposes: 1) to detect withdrawal dyskinesia, which may be a prodromal but reversible phase of tardive dyskinesia, and 2) to allow the clinician to evaluate whether continued neuroleptic use is necessary. This practice may also be tried with selected outpatients. However, the ubiquitous and critical shortage of adequate aftercare often does not permit such trials, because without medication and without close follow-up to detect early clinical signs of worsening, there is a high risk of relapse, often with serious medical and social consequences. However, the hospital setting does provide close monitoring and occasionally outpatients or day-hospital patients may be readmitted for periodic reevaluations. Even if such a trial with less medication has been unsuccessful within the past year, it still should be reconsidered at regular intervals and seriously attempted for most patients; the potential benefits outweigh the possible risks and the natural reluctance of those caring for fairly stable chronically ill patients to "rock the boat."

Some practical aspects of the prolonged use of neuroleptics, aimed at reducing the risks of late toxic effects, are summarized in appendix 3.

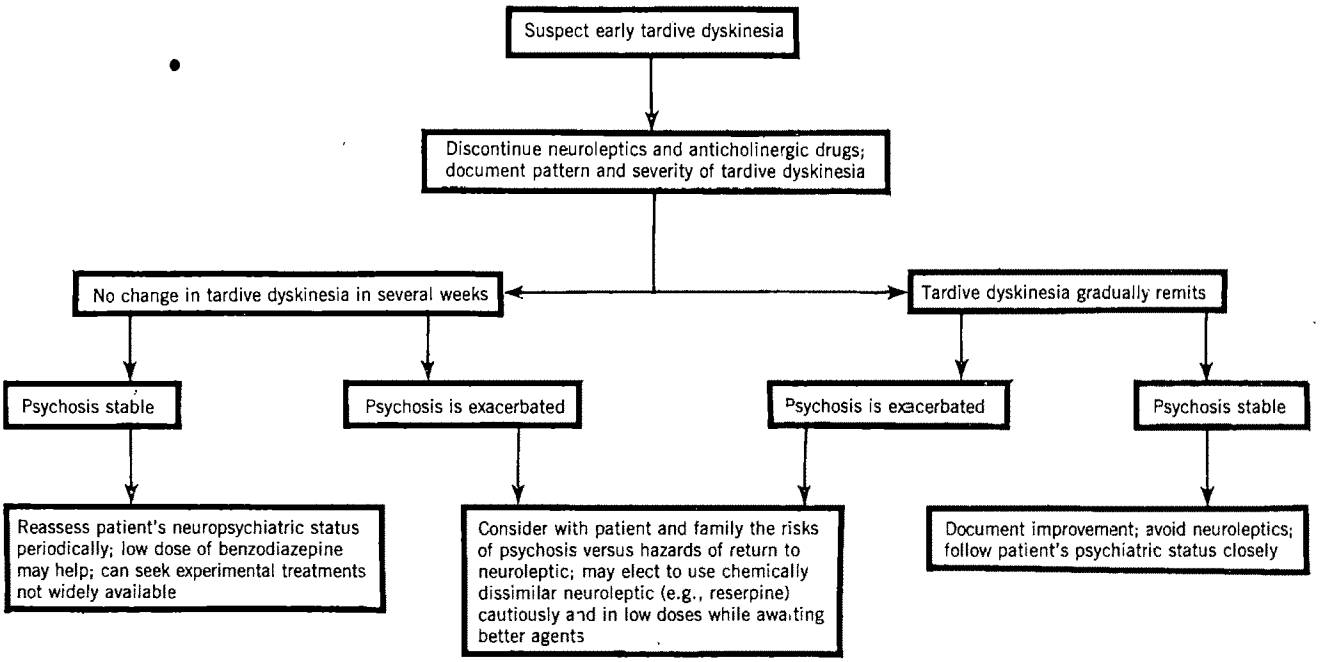
Management and Treatment of Tardive Dyskinesia

If a patient develops dyskinesia during a trial of reduced dosage of a neuroleptic or while fully medicated, the theoretical treatment of choice is discontinuation of neuroleptics (see figure 1). The available data suggest that tardive dyskinesia is often reversible if neuroleptics are withdrawn, especially early in its course, although some patients may require many months to improve. A dilemma is presented when a patient's psychosis is exacerbated after neuroleptic treatment is stopped and tardive dyskinesia is also present. In this situation the clinician, with the patient and family, must weigh the risk of psychiatric worsening, which may be seriously life-disrupting or even life-threatening, against the risk of tardive dyskinesia. Since tardive dyskinesia seems not to be a uniformly and relentlessly progressive neurologic disorder, it may be prudent to continue neuroleptic therapy temporarily if the psychiatric indications are sufficiently compelling. Although there is little scientific evidence to support the practice, some psychiatrists rely on less potent neuroleptic drugs with relatively less tendency to induce acute extrapyramidal effects in such situations. Clozapine had also been used in this way due to its low incidence of acute and late neurologic side effects, but this experimental agent is not available now due to other serious toxic effects associated with its use. Other safer agents are being sought, but none is yet available in American practice.

Although many therapies for tardive dyskinesia have been evaluated (see appendix 1), none has been either thoroughly studied or proven safe and effective. Discontinuation of neuroleptic medication is the theoretically ideal treatment but is not feasible for many patients. Newer investigational therapies are based on partially demonstrated increases in brain cholinergic activity (choline, lecithin, deanol) (59, 60), unproven increases in GABA-ergic activity (benzodiazepines, baclofen, sodium valproate, muscimol) (61, 62), or reduced dopaminergic activity (dopamine-blocking drugs, such as the potent neuroleptics themselves, or dopamine-depleting drugs, such as reserpine) (63, 64). It is possible that tardive dyskinesia is a heterogeneous group of disorders with a variety of anatomical, neuropathological, and pathophysiological bases, corresponding to the varieties of natural history and responses to treatment. No treatment to date uniformly benefits dyskinesias in all patients.

In short, there is no satisfactory or uniformly effective treatment for tardive dyskinesia. Recently proposed experimental treatments, notably attempts to in-

FIGURE 1
Recommended Scheme of Management of Tardive Dyskinesia



crease brain cholinergic function with deanol, choline, or lecithin, are attractive since they do not seem to worsen psychosis (theoretically they might even benefit psychosis as well as dyskinesia) (59, 60). Moreover, attempts to provide such experimental or unproven treatments (it is sometimes difficult to obtain them except in academic centers) offer a hopeful and positive approach to clinical management while the clinician and patient await a spontaneous remission of tardive dyskinesia over a period of many weeks or even months after discontinuation of neuroleptic treatment. Although increasing the dose of a potent neuroleptic may mask or suppress the dyskinetic movements, this tactic carries the theoretical risk of causing further neurologic insult. Nevertheless, this step is sometimes taken when the dyskinetic problem is severely incapacitating (similar to the practice of treating Huntington's disease or Gilles de la Tourette's syndrome).

At the present time, the best available means to deal with the problem of tardive dyskinesia is the use of conservative but effective dosage regimens of neuroleptics for clear-cut indications (continuing objective evidence of psychosis and of responsiveness to treatment).

Physician-Patient Relationship

Good practice necessitates that the psychiatrist sustain rapport with patients at risk of developing tardive dyskinesia. Furthermore, the psychiatrist is obliged to educate the patient as to the nature of his or her illness, the potential benefits of available treatments, and their potential adverse effects. Early in the treatment of a severely disturbed patient, such education may be limited by virtue of the patient's psychosis or confusion. At these times, family members or other persons close to the patient can be involved in such discussions. The need to review the treatment plan and its potential benefits and risks with the patient and his or her family becomes especially significant when prolonged neuroleptic therapy is considered or if tardive dyskinesia has already developed.

Although the routine use of signed and witnessed consent documents to protect the interests of the patient and the physician during prolonged neuroleptic therapy has been proposed, there are many problems with this approach. First, there are marked differences between established pharmacologic treatments and invasive surgical, diagnostic, or experimental procedures for which this type of formal documentation of consent is often (but not always) used. While the latter typically involve brief, acute risks during a patient's care, for which limited procedural consent can meaningfully be given, pharmacologic treatments of chronic disorders are likely to be continued and modified repeatedly over extended periods of time. Written consent would therefore not be feasible unless given in a blanket fashion, making it a potentially meaningless formal exercise. In addition, it is a precedent-setting step to routinely require written consent to institute an accepted, nonexperimental medicinal therapy. Based on these considerations, routine signing and witnessing of written consent for neuroleptic therapy is not recommended. On the other hand, good practice does require that the physician note in the records that the

indications for and risks of prolonged neuroleptic treatment have been carefully considered and reviewed with the patient or family.

Need for Further Research

A review of the present state of the medical treatment of severe chronic psychiatric disorders leaves many unanswered questions and underscores the need for further basic and applied research. There is an urgent need for the development of effective antipsychotic drugs that do not have the long-term risk of inducing tardive dyskinesia. This requires the development by academic and industrial pharmacologists of better drug screening tests (e.g., animal models that are not dependent on drug-induced extrapyramidal effects). Clozapine and sulpiride are examples of experimental drugs that seem to be effective antipsychotic agents with little acute or late extrapyramidal effect. Unfortunately, experimental trials with clozapine have been discontinued due to its association with agranulocytosis, and sulpiride is not available in the United States. Safer analogues of these or similar drugs are now being considered; their rapid development and clinical testing are strongly encouraged.

While we await the availability of more nearly ideal antipsychotic drugs, many questions still need to be answered about the use of currently available neuroleptic drugs. For example, more controlled treatment studies are needed comparing the efficacy of ECT, antidepressants, neuroleptics, and various combinations of these treatments with severely depressed patients. The safety and effectiveness of prolonged neuroleptic treatment of manic-depressive patients or others with episodic psychoses who cannot be managed with lithium alone need to be investigated. Although neuroleptics are sometimes used for patients with neuroses or character disorders and "borderline" patients, such use requires critical evaluation in research studies. Another research area in need of strong support includes various attempts to make the use of available agents for specific patients as effective as possible. Thus, more information on the dose-response relationships in man is required for all antipsychotic agents, especially to support the clinical impression that effective maintenance doses may be lower than those required for acute exacerbations of psychosis. In addition, efforts to apply available and still-evolving methods of analysis of drug metabolism and pharmacokinetics to routine clinical use are needed. While neuroleptic pharmacotherapy has greatly advanced patient care, it must be continually improved through sound clinical research.

Postgraduate Education

Although further research is required to address the many areas of uncertainty already discussed, much is already known that has a bearing on the safe and effective use of antipsychotic drugs in clinical practice. Our survey of North American psychiatric training centers indicates that the majority provide some training in clinical psychopharmacology to residents and practicing physicians. These aspects of postgraduate and continuing medical education are strongly encouraged and should be given even greater support to ensure that the best possible care will continue to be available to the most severely ill psychiatric patients.

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APPENDIX 1
Differential Diagnoses to Consider in Evaluating Tardive Dyskinesia

1. Neuroleptic withdrawal-emergent dyskinesias or other transient acute dyskinesias associated with neuroleptics (see table 1).
2. Late and persistent "classical" tardive dyskinesia itself (both psychiatric disorder and dyskinesia may be present).
3. Stereotyped movements of schizophrenia (both psychiatric disorder and dyskinesia may be present).
4. Spontaneous oral dyskinesias of advanced age or senility (both psychiatric disorder and dyskinesia may be present).
5. Oral dyskinesias related to dental conditions or prostheses.
6. Idiopathic torsion dystonia.
7. Focal dystonias (bromandibular dystonia, blepharospasm, spasmodic torticollis) and habit spasms (tics).
8. Huntington's disease (both psychiatric disorder and dyskinesia may be present).
9. Gilles de la Tourette's syndrome.
10. Wilson's disease (hepato-cerebral-lenticular degeneration due to abnormal copper metabolism), manganism, and other disorders due to heavy metal poisoning (both psychiatric disorder and dyskinesia may be present).

11. Fahr's syndrome and other disorders with calcification of the basal ganglia.
12. Postanoxic, postencephalitic, and encephalitic extrapyramidal syndromes (both psychiatric disorder and dyskinesia may be present).
13. Rheumatic chorea (Sydenham's chorea, St. Vitus' dance).
14. Drug intoxications involving L-dopa and amphetamines and, less commonly, anticholinergics, antidepressants, lithium, and phenytoin (both psychiatric disorder and dyskinesia may be present).
15. CNS complications of systemic metabolic disorders, such as hepatic or renal failure, hyperthyroidism, hypoparathyroidism, hypoglycemia, and vasculitides (both psychiatric disorder and dyskinesia may be present).
16. Brain neoplasm (thalamic, basal ganglia).

APPENDIX 2
The Differential Pharmacology of Tardive Dyskinesia[a]

AGENTS THAT MAY PARTIALLY SUPPRESS TARDIVE DYSKINESIA

Dopamine antagonists
- Apomorphine (in low dose)
- Butyrophenones
- Clozapine
- Papaverine (mechanism uncertain)
- Phenothiazines
- Pimozide

Amine-depleting agents
- Reserpine
- Tetrabenazine

Blockers of catecholamine synthesis
- Alpha-methyldopa
- Alpha-methyltyrosine

Blockers of catecholamine release
- Lithium salts

Cholinergic agents
- Deanol (mechanism uncertain)
- Choline and lecithin (phosphatidylcholine)
- Physostigmine

Miscellaneous agents
- Baclofen (postulated GABA mechanism unproven)
- Valproate (postulated GABA mechanism unproven)

Muscimol

AGENTS WITH VARIABLE, NEGLIGIBLE, OR UNCERTAIN EFFECTS

Alpha-methyldopa
Amantadine
Antihistamines
Barbiturates
Benzodiazepines
Cyproheptadine
Deanol
Isocarboxazid
Methylphenidate
Penicillamine
Physostigmine
Pyridoxine (vitamin B_6)
Tryptophan

AGENTS THAT WORSEN TARDIVE DYSKINESIA

Anticholinergic agents
- Antiparkinsonian agents (e.g., benztropine)

Dopamine agonists
- Amphetamines
- L-Dopa (may have opposite effect in small, repeated doses)

Other agents
- Phenytoin

[a]Some drugs appear in more than one category, reflecting ambiguity in the literature. Note that while apomorphine is usually classified as a dopamine-agonist, it actually has complex mixed actions, may antagonize dopamine at low doses, and has clear antidyskinetic effects.

APPENDIX 3
Suggested Guidelines for the Avoidance and Management of Tardive Dyskinesia

1. Consider carefully the indications for prolonged neuroleptic therapy; the indications should be serious (e.g., chronic psychosis), and there should be objective evidence of benefit.
2. Seek alternative therapies with neuroses and mood and character disorders.
3. Use lower doses with elderly patients and children; strive for minimum effective doses; avoid multiple drugs; discontinue antiparkinsonian agents as soon as possible.
4. Advise patients and families of risks and benefits; arrive at a mutual decision when use of neuroleptic exceeds 1 year. Note discussion and agreement in clinical record.
5. Examine patient regularly for early signs of choreoathetosis and oral-lingual dyskinesia. Consider alternative neurologic diagnoses.
6. Reevaluate patient and document indications and response at least every 3–6 months and attempt to reduce dose.
7. At earliest sign of dyskinesia, lower the dose, change to a less potent agent, or, ideally, stop treatment; await remission as long as psychiatric status permits.
8. Treat dyskinesia with benign agents first (e.g., diazepam, deanol, choline, or lecithin in high doses, possibly lithium); stay alert to new experimental therapies, if only to bide for time and offer hope. Reinstitute neuroleptics only as an extreme measure for disabling dyskinesias, using lowest doses feasible.

The Unstated Problem in a Psychological Testing Referral

BY LESLIE JORDAN COHEN, PH.D.

There is a manifest and a latent content to many psychological testing referrals. Although a diagnostic problem is usually the stated reason for the referral, the unstated and more basic reason frequently has to do with problems the therapist is encountering in attempting to deal with a difficult treatment situation. The testing psychologist who responds at the manifest level alone may not help and at times may even be a hindrance.

Psychiatrists tend to think of psychological testing as a procedure for establishing diagnosis. Their usual assumption is that a case is referred for testing because it poses a diagnostic problem, and psychologists tend to share this view. Psychologists are often trained to think of themselves as possessing in their test instruments the tools to make subtle diagnostic differentiations that might elude psychiatrists with their more "impressionistic" methods of mental status exam and clinical interview. A request for testing phrased in terms of the need for diagnosis fits into everyone's conception of a straightforward testing referral.

A consult sheet directed to the testing psychologist frequently asks for a diagnosis in *DSM-II* terms, with such questions as "Is this patient manic-depressive?" or "Is this a functional or an organic psychosis?" Sometimes the referral question is stated in what might be called diagnostic building block terms, with such questions as "Are there ego deficits?" or "Is there evidence of depression?" Phrased either way, such questions imply that the psychiatrist is experiencing an uncertainty about the case that diagnostic information would help to resolve. The psychologist does his or her best to give thorough answers to these questions.

Even when the referral appears straightforward, however, it is my impression that psychologists and psychiatrists are not infrequently frustrated in their efforts at collaboration. After the work is completed and a testing report entered in the chart, the psychiatrist might be saying to himself, "This report is long and very academic but of not much practical value; it basically does not tell me anything I did not know already." The psychologist might conclude that his or her special skills simply went unrecognized by a practitioner who could not appreciate them. Both sides may be right, but I think in many cases both sides are missing the most important issue. In my own sample of testing referrals, in only a small percentage of cases does the referral question completely state the problem that the therapist needs help with. That is, in only a small portion of cases is it a clear-cut diagnostic problem that has prompted the request for testing.

Testing referrals can be thought of as falling along a hypothetical continuum. At one end of the continuum are the cases where a definitive diagnosis is of crucial importance to the referring physician, and on the other end are the cases in which it is of no practical value at all. On the end where diagnosis is crucial would fall those cases in which proper treatment depends directly on diagnosis, i.e., where treatment carried out under the assumption of an incorrect diagnosis would be not only ineffective but injurious to the patient. An example would be a case of a patient with a brain tumor who is mistakenly being treated for schizophrenia. A little further along the continuum are cases where diagnosis may be more or less important depending on the therapist's treatment orientation. If the therapist is of the opinion that some classes of psychopathology are best treated in certain specific ways—for example, schizophrenia with phenothiazines, psychotic depression with electroshock, certain forms of manic-depressive illness with lithium—then a specific *DSM-II* diagnosis becomes important.

For the therapist whose basic treatment approach is a psychodynamic one, that is, when the main treatment variables to be worked with are unconscious conflict and defense, most cases fall toward the end of the continuum where precise diagnosis is of diminishing importance. For many of the cases a dynamically oriented therapist sets about to treat, there is not a one-to-one relationship between the patient's diagnosis and the thinking involved in mapping out a treatment strategy. In order to work out a modus operandi for treatment, the therapist must solve the problems not so much of diagnosis but of the very individual and specific ways the diagnosis expresses itself in the trea-

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ment situation. The issues that become crucial to understand include how the patient experiences the therapist in the transference, including specific transference fantasies, the basic anxiety that the patient is avoiding and which ultimately must be interpreted, the often times subtle, individualized ways the patient defends against anxiety, and the like.

Although testing referrals tend to be phrased in terms of the need for diagnosis, it is problems in the area of doing this broader and more individualized assessment that at bottom constitute the difficulty of the case and what often cause it to be referred for testing. Thus, one might say that there is a manifest and a latent content to many testing referrals. If the request for testing is responded to in terms of the manifest content alone, i.e., in terms of a diagnosis, it frequently does not go very far in helping the psychiatrist identify the underlying treatment issues that make the case a problematic one.

CLINICAL EXAMPLES

I will give three clinical examples to illustrate this situation. The first is a case where the referral request was "Rule out schizophrenia." The psychiatrist was called at night by the patient's husband with an urgent request for hospitalization. For some weeks this young woman had been accusing her husband of acting improperly toward his niece. The husband explained to the psychiatrist that there was no truth to these accusations, but he had been unable to convince his wife of this. On this particular night she had gone into a rage, shrieked her intention to kill herself, and had apparently overdosed on pills. Once admitted to the hospital, the patient quickly became enraged and demanding and at times exhibited bizarre behavior; the psychiatrist was called repeatedly by the staff for measures to handle her. The psychiatrist, who was making efforts to settle down his new patient first through ordering medication, then restraints, then seclusion, and finally a court order when she announced her intention of leaving, found himself quickly in the same role as her husband had been—the object of her rage and suspicion. He wondered if he had become the focus of a psychotic transference in a paranoid schizophrenic woman. After several days of hospitalization, when she stopped raging at him and treated him with aloofness, he wondered if this was a manifestation of "flattened affect" and further evidence of schizophrenia. It was at this point that he made the testing referral.

The testing psychologist is in a unique position when called into a case like this. He or she has the luxury of being able to do an evaluation at leisure, is spared the responsibilities of handling the case, and is often exempt from the intense transference reactions that are focused on the psychiatrist as primary therapist. In this position, and with the added benefit of standardized or semistandardized instruments to lean on, it is at times easier for the testing psychologist to maintain an objective outlook than it is for the primary therapist, who is in the thick of things therapeutically. In this case, the patient showed no signs of schizophrenic thinking in her test protocol or behavior. What did emerge was a woman with a hysterical character who was prone to dramatic emotional storms, and who had an especially painful vulnerability to narcissistic injury and feelings of humiliation with men. Her psychiatrist's efforts to treat her with restraining measures had intensified her humiliation and made her feel justified in unleasing her fury toward him in whatever way she could think of.

Having arrived at this diagnostic conclusion, the testing psychologist is able to respond to the referral question at the manifest level, but how much it will help the therapist to be told that his patient is not schizophrenic is a moot point. In fact, at the point where he requested testing, he might have been relieved if a diagnosis of schizophrenia had turned up; it at least would have given him a handle for understanding a very difficult case. The latent problems behind the referral, and those that the psychiatrist is still left with, have to do with this patient's need to act out her rage and humiliation and to pressure others to act it out with her. The psychiatrist is pressured to abandon his observing therapeutic stance and to involve himself with her in the role of a humiliating, punishing father figure, the role which she expects of all men. Without realizing it, he has acted out this role. She pays him back by becoming more and more emotional, humiliating herself further, and making him out to be ineffectual by defying his efforts to stop her.

The patient's defensive need to provoke her psychiatrist into this kind of position can be expected to come up repeatedly in any effort to treat her. Therefore, the psychiatrist's basic problem, and the unstated problem behind this testing referral, is finding techniques to help the patient understand this need in herself rather than acting it out with her.

In another case, the psychiatrist telephoned with the following request:

> Please evaluate this adolescent boy for impulsivity and possible ego defects. He has run away from home three times in the past two months. By the way, can you get to the testing by tomorrow? I promised this boy he would only be detained in the hospital three days if he would consent not to run away for that length of time. I think this boy is really upset underneath it all, and I can't afford to lose him.

The referral question is quoted in full because it nicely illustrates how the manifest referral question—the request for an assessment of impulsivity—is embedded in the context of a therapeutic dilemma. The boy, who sounds like he may be a past master at externalizing his unhappiness, has already caught the

psychiatrist up in a transference-countertransference bind in which the therapist feels he has to chase around after the patient and cajole him into doing something for himself. In this position, the boy is basically in control and the therapist fears he may lose the case.

Although the therapist phrases his referral in terms of the need for diagnosis, it is clear in this case that he has already diagnosed the patient's major ego problem. He knows that the boy is impulsive. His implicit question is what to do about it. How does he handle an impulsive boy? How does he build an alliance with someone who makes as a precondition for treatment that he be cajoled and bargained with? Clearly, these are the problems that have him stumped, not the diagnosis.

However, the fact that the referral question is posed in terms of a diagnostic dilemma when the psychiatrist already knows the diagnosis suggests that there is a defense operating. For whatever reasons, it is apparently hard for the therapist to acknowledge to himself that he is having difficulty handling the case. This in turn poses a problem for the psychologist in terms of how to respond to the referral. If he responds in terms of the manifest question and gives diagnostic information, he is at best being academically correct. But to what purpose? The report is of little practical value, since it tells the psychiatrist something he already knows. If the psychologist attempts to give technical advice, he or she is bound to run up against the psychiatrist's defenses and to be experienced as intrusive and presumptuous. This kind of dilemma makes for tension between psychiatrist and psychologist.

In the final clinical example, the psychiatrist asked in his referral for testing whether there was evidence of depression and/or borderline pathology in his female patient. When I arrived for the first testing appointment, I realized that I had walked into a battle between the patient and her therapist and that I was expected to be the arbiter. The patient greeted me by saying, "My psychiatrist says I am depressed, and I say I am not. I think I'm ready to leave the hospital, but he says I can't because I'm mentally ill. I want you to tell him that he's wrong."

I tried to learn what had led up to this battle. The patient had entered treatment some months earlier because of a severe letdown and feelings of anxiety after the youngest of her three children started grade school. While her children were young, she had felt charged with self-confidence, but their reaching school age left her feeling empty and fearing that she would ever after be directionless in life. When she began treatment, which was at first on an outpatient basis, she reported that she quickly got in touch with feelings of rage toward her own hypercritical and controlling mother; she had apparently developed a mother transference toward her therapist around that time, too. With the therapist she remembered longing for unmitigated praise and reassurance, and felt deflated and angry when she did not get this from him. She called him between sessions saying if he did not "give her something" she was afraid of how depressed she might become. He gave her medications, in increasing dosages, as the between-session calls and demands for reassurance escalated. Finally, he told her that outpatient therapy was not sufficient and suggested hospitalization. She panicked; in her mind, her therapist was confirming her worst fear, that she was very sick and on a downhill course from which she would not recover. She declined hospitalization, but by this time he was insisting on it, saying she was too ill to make her own decisions. The battle had begun in earnest. By the time the testing referral was made, the patient had adopted a position that she was, in her words, "just fine, with no mental illness, no defense mechanisms working, and no signs of depression." She felt furious with her psychiatrist who, with threats and angry confrontations, was trying to force her out of her denial.

This is a testing referral which falls on that end of the continuum where diagnosis is of no practical value at all. Both therapist and patient are seeking diagnostic information not as a means for understanding something together, but as a weapon to force the other to capitulate. For the psychologist in this situation, it is hard to see how responding in terms of diagnosis could do anything but intensify the pitch of the battle. Furthermore, this is another case where the psychiatrist seems to have a good enough understanding of the diagnosis before he requests testing. He knows that she has problems with ego functioning, relies on blanket denial, and fears she will be overwhelmed if any feelings seep through. He knows she is dealing with individuation issues in the transference and that she experiences feelings of emptiness when he cannot be with her or do her thinking for her. The problem for him is not in identifying these weak points but in knowing how to deal with them. How can he treat someone who has a phobic attitude toward uncovering her own feelings? How does he help her deal with anxiety over separations? How can he avoid taking the role of an intrusive mother when his patient's behavior seems to be pushing him into it? These are the real problems behind this testing referral.

What can be concluded about the usefulness of psychological testing in cases such as those I have presented? Optimally, simply answering the manifest referral question can serve as an injection of diagnostic reality for the therapist, who can then go on to reassess the underlying treatment issues for himself. This was the situation in the first referral I described, where testing was requested to rule out schizophrenia. Once the hysterical nature of his patient's symptoms was brought back into perspective, the psychiatrist was less alarmed by the patient's outbursts and in a far better position to empathize and understand them with her.

The process does not always work this smoothly, however. Sometimes the countertransference has become too intense or the technical problems too complex to yield without being directly identified and discussed. This was the situation in the last case. Unfortunately it is rare, when the latent referral problems need to be opened up for discussion, to have the kind of collaborative relationship between psychiatrist and psychologist that can make such discussion comfortable and productive. Often the two have not met face to face but communicate by means of consult sheets or telephone calls. The psychiatrist may resist discussion of the therapeutic problems for defensive reasons. The psychologist, too, may have learning problems and resistances that hamper his or her effectiveness. Commonly, the psychologist is so focused on demonstrating diagnostic expertise that he or she fails to appreciate situations where such expertise is irrelevant. If there are resistances on each side, the testing can turn into an exercise for confirming the defensive perceptions of everyone involved, including those of the patient, and thereby actually contribute to the difficulty of the treatment situation. It seems that the more both sides can develop a readiness to listen for the latent reasons behind a referral, the more likely testing can turn out to be a genuinely useful consultation.

Curt P. Richter Prize in Psychoneuroendocrinology

Through the generosity of the Irish Foundation for Human Development, an annual prize has been established for meritorious research in the area of psychoneuroendocrinology. The sum of $1,000 will be awarded annually for the best essay or manuscript (original research or a review article including research) submitted by a scientist or physician under 35 years of age by January 1 of the year of the award. The winning paper must be in a format suitable for publication in *Psychoneuroendocrinology*, the journal in which the winning paper will be published. The aim of the prize is to encourage younger scientists to contribute to this interdisciplinary field.

Manuscripts should be submitted in *quadruplicate* to

Dr. Fleur L. Strand, Secretary
International Society of Psychoneuroendocrinology
Biology Department, New York University
Washington Square, New York, N.Y. 10003

All submissions will be screened by a broad committee of established psychoneuroendocrinologists. The deadline for submission is January 2, 1981.

The Father's Parenting Experience in Divorce

BY HENRY J. FRIEDMAN, M.D.

The author discusses the father-child relationship as it is influenced by divorce. Psychiatrists are often consulted by individuals considering divorce who are concerned about its probable impact on their children. Data gathered from the treatment of fathers during divorce indicate that there can be positive changes in their parental bonds as a result of increased opportunities to relate to children in a conflict-free atmosphere. In such a setting, the father's nurturing experience provides him with a new perspective on parenting. Psychotherapeutic help can lead to a strengthening of parental bonds, with subsequent benefits to both father and child. The author encourages professionals to support men in improving their fathering during the postdivorce period.

Despite the currents of social change, or perhaps because of them, the literature on divorce and family life has tended to favor conservative and even reactionary views about the effects of divorce on child development and parental functioning. Many authorities consider an intact family unquestionably preferable to separation and divorce (1, 2). Authors who conclude their presentations with a cautionary statement that they are not opposed to divorce as a solution to marital conflict still fail to differentiate adequately divorce situations wherein optimum personality structure in the parents leads to a very different outcome than is seen in other divorce situations (3). It is peculiar that despite our experience with the negative effects of persistently conflictual marriages on the development of patients we treat as adults, no attempt has been made to identify the potentially positive effects of divorce on child-parent relationships.

The regrettable absence of such an approach is compounded by a tendency in the psychoanalytic literature to underemphasize the father's role in child development. In a 1973 paper, Burlingham (4) presented material about the role of fathers in the lives of preoedipal children. Although she acknowledged a greater possible role for the father, she continued to maintain that the preferred pattern for development was based on predominantly "feminine" mothers and "masculine" fathers. Burlingham indicated that blurring of feminine and masculine responsibility could lead to a "decrease in variety of experience which might well prove to be a loss to the child and show up later in the development of a flatter, more uniform and less rich affective life." Furthermore, according to Burlingham, the father's interest in nurturing a new offspring seemed to be based on a pathologic feminine identification and competitiveness with the mother.

The purpose of this paper is to discuss the possibilities of positive effects of divorce on the father's experience as a parent. Improvement in the father-child relationship is a possible outcome in many divorces, particularly if the children are young. Divorce increases the possibility for an intensified parental bonding experience through the father's opportunity to do more direct nurturing. I will attempt to present a perspective on the experience of divorce as a possibly positive adaptational experience that can give fathers the opportunity for increased nurturing experience and hence greater intuitive appreciation of their child's growth and development. The importance of a nurturing relationship for the development of an adequate bond between father and child has rarely been acknowledged, while the emphasis on good mothering as a necessity for future health has been a constant theme. There is an element of the nurturing experience that goes beyond providing the basic requirements for the child's development. The importance to the child of being nurtured by the father resides in the increased attachment of the father to the child and the resultant increase in paternal intuition. That later paternal attachment might be enhanced by fathers having intense bodily responsibility for their children in infancy seems axiomatic but has not been widely acknowledged.

The divorced father faces a crucial choice about his involvement with his children. Unfortunately, until recently custody and visitation arrangements tended toward minimal paternal involvement. Now that this has changed, more fathers are having to face the intricacies of a parenting role. In this paper I will emphasize the role of psychotherapists in helping fathers en-

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hance their participation with children. I will also discuss the sexual confusion, fear, and withdrawal from the parental role that often interfere with a father's positive adaptational response but can be ameliorated by proper clarifications in psychotherapy.

Psychiatrists are in the unique position of seeing parents in various stages of marriage, separation, and divorce. Although we see a wide range of disturbed reactions, we also observe situations in which there can be a strengthening and even improvement of a father's roles in his children's lives. When parents seen in individual psychotherapy assert that they are staying in a marriage for the children's sake, one often sees a pattern in the fathers of overwork, absence from the home, and a tendency to give over control of the children to the wife. It is unlikely that high-level intensive fathering is occurring in such situations, even though there is no overt split between the parents.

Adult patients who have grown up in homes with unhappily married parents almost always report fantasies and wishes that their parents had separated in order to reduce tension and possibly permit a more harmonious relationship with each parent. Often this picture seems quite compatible with the therapist's speculations about what might have happened had a divorce actually occurred.

THE FATHER IN A CONFLICTUAL MARRIAGE

Burlingham (4) noted that "during the last and beginning of this century it was considered unsuitable for a father to be openly involved with the infant's care, as if taking the maternal role in this respect was undignified for a man and betrayed a lack of masculinity." While Burlingham acknowledged some shift from this extreme position in the English fathers she observed, she was skeptical about the changes apparently occurring in the liberated upper-middle-class families of the United States. In fact, the fathers she described would be considered unacceptable as parenting partners in marriages between compatible, loving adults in this country. Improved family planning and greater equality in decision making and in sharing economic burdens have meant that fathers are no longer remote "instrumental" figures (5). Fathers in the modern intact, educated family have a far greater share in the actual handling, feeding, bathing, and clothing of the newborn infant. Of course, when both parents adhere to a strict division of function, the importance of the father's role is delayed and often diminished. However, with more women returning to work soon after childbirth, sharing (not blurring) of child care responsibilities has become more the rule than the exception.

In families where incompatibility of emotional needs is reflected in overt or covert conflict, optimum sharing rarely occurs. Instead one sees some estrangement of the father from the newborn child. When marital tensions are chronic, the development of the father-child relationship is disturbed. Although few patients are in treatment for sufficient time to observe this deterioration at each stage of the father-child relationship, it is possible to reconstruct from male patients at various stages the deviations from optimum parenting style. If the conflicts between the parents remain unresolved and the marriage continues, there is a tendency for the father to become a shadowy, less significant parental figure.

CASE REPORTS

Case examples illustrating the nature of the father-child relationship in various stages of divorce are presented to show that the effects on the child-parent interaction are not always negative. When the father is in analytic psychotherapy or analysis, as were the two men described herein, it is possible both to study the nature of the father-child interaction and to influence it through therapeutic interaction. Of course, the results obtained in the cases I will describe are unusual in that patients capable of being in intensive therapy often have better developed ego capacities for experiencing emotion and modifying their interactions with others.

Case 1. Mr. A, a 35-year-old professional man, entered treatment because of marital discord. His wife was spending increasing amounts of time away from the home and becoming involved with younger colleagues from her work. Mr. A had responded to his wife's absences from the home, most of which were for social activities, with increasing exasperation, temper tantrums, and depressive affect. He had, however, remained active in his profession and was quite successful despite his personal unhappiness.

On closer examination, the marriage appeared to have been unsatisfactory in many ways before Mrs. A began to move away from her husband. She did not admire his considerable accomplishments and in fact resented the demands of his professional life. The couple had two children, ages 8 and 5 when Mr. A's treatment began.

As treatment proceeded it became clear that the patient was seen by his children as an intrusive, frightening, and unpleasant individual, a perception that had much basis in their interactions with him. Mr. A worked long hours and was seldom at home. He often returned to be faced with the children's fighting or uncooperative behavior. He tended to blow up at the children and yell at them for their inadequacies. Mr. A seldom took the time to understand the complexity of his interactions and often displaced his anger from his wife to the children. As a result, he seldom had a pleasant interaction with the children. Although he was sentimental about them, it seemed unlikely that there could be much basis for actual positive feeling on their part. The children gave many indications that they were content with their father's absences from the home.

In treatment Mr. A's attention was drawn to the basic nature of his interaction with the children, which focused on irritability and harassment rather than understanding and caring. Even when the marital discord decreased, Mr. A's

relationship with the children remained vaguely troubled. He could not understand their "childish" responses to each other, their "dawdling," and their frequent tearfulness.

Eventually Mr. A decided to end his marriage. At the time of separation he did not have much feeling about leaving his children; he seemed more like a single man looking forward to a life of freedom. However, on the therapist's urging, Mr. A declined job offers in other cites and took up residence within walking distance of the family home. He spent time investigating private schools for one of his daughters and eventually arranged for her to attend a school much better suited to her developmental needs.

The children began to be more real for Mr. A as he faced taking care of them on his own without the interference of his conflict with his wife. He realized that in the past his attention to the children's problems had often led to arguments with his wife, so he never saw his interventions and interactions with them through to a successful conclusion. He could now experience his emotional needs for closeness with his children. The therapy has continued, with evidence that a real parent-child relationship is developing.

Case 2. Mr. B's history is presented to show the fallacy of assuming that a father's continued presence in the home, despite severe marital conflict, is helpful in developing an adequate father-child relationship. As is often true in divorce, one spouse (the wife in this case) had a subtle but severe pathology.

Mr. B was an administrator in his late 40s who sought treatment for chronic depression, work inhibition and ineffectiveness, and distress related to his recent loss of another important position. The patient felt that he often failed to accomplish important work tasks despite direct pressures, and he had in fact been fired for this reason. He expressed his feelings about his failure primarily in terms of disappointing his wife. Although Mr. B described her as an excellent wife, she did not meet what would ordinarily be considered the standards of reasonable companionship. Mrs. B was chronically angry and depressed and blamed the patient for her distressed state.

The couple had two children who were adolescents at the time of the treatment. Mr. B had no relationship at all with the children, who had formed an almost symbiotic bond with their mother. Although Mr. B said he got along better with his older daughter, she seemed extremely narcissistic in her relationship to him, not caring much about his wants or needs. The younger daughter was heavily allied with her mother; Mr. B said she shared her mother's every whim and was concerned exclusively with her comfort and needs rather than those of her father. As the patient began to reflect on his relationship with his children, his sadness was intense. He had always been aware of some marital unhappiness, but he had felt certain that he must remain in the marriage for the children's sake. When he had worked through his feelings about his relationship with his children sufficiently, Mr. B felt able to leave his wife and to give up the illusion of a satisfactory relationship with his children.

The effects of the separation seemed largely positive, within the confines imposed by the established preference of the children for their mother. However, for the first time Mr. B can see some positive aspects in his relationship with both children as they are forced to develop some relationship as individuals.

EARLY DIVORCE AS A MODE OF CONFLICT RESOLUTION

If in a disturbed couple the father tends to withdraw or is excluded by the mother, one can hypothesize that the normal attachment process of father to child or child to father will be hampered. Divorced fathers who take complete care of their infant offspring, at least on a part-time basis, have a special opportunity to experience the child in an intimate setting. Men in conflictual marriages, and possibly in most marital relationships, may view their relative passivity in regard to using their full capacity as nurturing, caring figures as a reasonable response to their children. Although some attention has been paid to the need for men's liberation from rigid role models, this has not been done with the same intensity or ferment that has attended the reexamination of the woman's role. However, there is some indication from social observation that many fathers are becoming more aware of their children emotionally, particularly of the importance to their narcissism of their children's caring responses. The father involved in separation and divorce often reports a new awareness of the joys of child care. Fathers who might have bathed, fed, and comforted their children only on occasion within the marriage may find themselves doing such things regularly, with very positive results. Orthner and associates (6), in their study of fathers with exclusive custody, reported that "if there is one most impressive conclusion we can make from our interviews with these single parent fathers it is this: These fathers feel quite capable and successful in their ability to be the primary parent of their children." These researchers were clearly surprised by their finding that fathers did quite well tending to their children on their own.

FACTORS IN A FATHER'S POSITIVE ADJUSTMENT TO DIVORCE

Although fathers in the United States may hold, handle, bathe, and feed their children during the first year of life—serving as perfectly acceptable alternative caretakers—this is often done only on rare occasions and as a "favor" to the mother. There is often no necessity for the father to exercise his potential for caretaking in the intact family. When the mother works, caretaking may be relegated to another family member or to a paid caretaker.

In severely conflicted marriages, pregnancies and births still occur. The birth of a child may increase the marital estrangement, but it is fairly unusual for separations and divorce to occur when there are children under 1 year of age. There are several probable causes for this fact. First, even in difficult marriages, a pregnancy may be an attempt to forestall an actual break. After the birth of the infant, the mother's physical

needs and vulnerability may delay a separation through a combination of both neurotic and reality factors. Possibilities of separation increase during the child's second year of life. If it is hypothesized that direct care of the infant increases the father's attachment, then this may be an optimal time for the change to occur. When children are older, the opportunities for increased intensity of attachment are limited by family alliances (usually stronger to the mother) that have formed during the earlier years. Further, the older child is at a developmental stage in which his or her energies are directed away from the primary object in general. The newly divorced or separated father with a child under 2 years of age or several children under 5 years of age faces his own developmental challenge as well as an opportunity to expand his experience of his children. In cases of later separation the father may be able to separate himself from a morass of strife that has prevented him from conveying to his child a clear image of who he is and to receive from the child a relatively uncontaminated response that depends on adequate separation from the mother.

The challenge of single fathers revolves around an issue of sexual role identity. Lerner (7) has noted that sexual role stereotypes are more adaptive for individuals whose sexual identity is weak or brittle. The man who can do nothing that is identified culturally as feminine and the woman who cannot engage in any activities commonly identified as masculine represent, according to Lerner (7), the less healthy end of the sexual identity spectrum. Men who adhere rigidly to narrow, demanding views of masculinity are unable to take advantage of the opportunity of being a "separate" parent. These men tend to withdraw from their children or see them very little for a period after the separation. This withdrawal may persist until the children have reached an age where they no longer require "maternal" care. Unfortunately, such men often view psychotherapy as threatening to their masculinity and avoid treatment as something unacceptable to their view of themselves as men. Men burdened with this machismo syndrome, which consists of rigid adherence to a narrow set of male characteristics and an insistence that certain activities are exclusively masculine or feminine, are often forced into quickly finding a new woman to depend on. The dependency is expressed in terms of activities that "must" be done by women (cooking, cleaning, arranging a social calendar, doing the laundry), not in terms of closeness to another person. Since there are so many single and divorced women in our culture, men with such needs often find a female replacement for the lost, discarded, or rejecting wife quickly. This woman is then used as the new caretaker for any small children during periods of visitation.

The centrality of a flexible sexual identity for single fathers cannot be overestimated. Only with such flexibility is a new form of male parenting related to the divorce experience possible (8, 9). It is curious that our profession has shown so little concern about this concept. In their book on part-time fathers, Atkin and Rubin (10) described their method of helping fathers adjust to the arduous task of being with their children. They try to teach fathers how to be comfortable with children for whom they must care during visitation periods. It is clear from the authors' examples that they deal largely with men who have had little experience with the requirements and joys of maintaining a house and feeding and clothing small children. In my clinical experience men are not always so rigid and often have started on their own to achieve the special relationship that can occur when a father cares directly and exclusively for his child. Current custody practices are such that it is still rare for a father to obtain exclusive custody of small children unless the mother refuses custody or is declared unfit. On the other hand, men are increasingly asking for and obtaining joint custody or a much greater level of visitation. A concerned father may see his child for two or three days a week, often on weekends, during which time he is responsible for the physical care, entertainment, and understanding of the child. Contrary to what might be expected, if separation experiences are early and consistent, the young child usually makes an intense and satisfactory attachment to the father. The father who is open to and unthreatened by the nurturing role begins to experience what have been described as maternal feelings. The "in-tuneness" of the good mother becomes a part of his ego capacities. It is difficult to describe a man's discovery that his nurturing capacities are greater than he had anticipated. Furthermore, this intricate and intense understanding of the child is something that "normal" fathers may persistently lack.

Divorced fathers who have an opportunity to live with their children as caretaking parents rather than Sunday afternoon baby-sitters also report that their sense of loss and alienation as a result of the divorce has been lessened. They describe a different and far less intense or absent experience of loss resulting from this experience with their children. Many of these men report great pleasure with the parenting experience and voice regret that they did not have this experience sooner.

The importance of this new form of attachment for men lies in sharing an intensity of experience that was largely the domain of mothers in the past. This has to do not only with socially conditioned patterns of parenting but with the fact that the early symbiotic union is a basis for empathic understanding of subsequent childhood development. The father who knows his young child is more inclined to be aware of the phase-specific problems and conflicts of each developmental stage and, later, to recognize the child's transition in latency to a more adult sensibility. Awareness of the child's personhood is intensified, and the special nature of the father-child unit is increased. This special

quality deserves further consideration. The divorced father who has had considerable contact with his child thinks and feels more like a traditional mother, who is never far from including the child in some way in her emotional life. Fathers—and mothers—fantasize about the child's inclusion at a social event, on a trip, or on summer vacation. Furthermore, there is beginning to be support in the social milieu for the importance of the father-child relationship. Friends and relatives ask about the child by name, and schools have started to include the father in a much more direct and meaningful way.

Of course, parenting is never free from ambivalence, and one experience of the father as exclusive parent is an awareness of the extremes of love and frustration with the child. The "maternalization" of the father creates a new psychological experience for both parent and child. The father also achieves a kind of indestructibility as an object. There is less risk of postdivorce withdrawal by the father and of his gradual exclusion from the child's life. Even when the mother is hostile to the father, his loving, caring, and limit-setting involvement with the child prevents his becoming a shadowy figure who may be fantasized about but not experienced fully. Children who have lived part-time with their fathers and have been cared for by them directly cannot fail to identify with the father's real characteristics and understand them better.

IMPLICATIONS FOR CLINICAL PRACTICE

It is important that psychiatrists recognize the potential of early separation and divorce in facilitating father-child attachments. People who otherwise might not consult psychiatrists and psychiatric social workers do so in the face of deteriorating and possibly lost marriages. Psychiatrists, like anyone else, are subject to time-honored prejudices that are viewed as facts. Many do not believe in divorce and assert that it is always detrimental to the children. Of course, many situations deviate from the ideal described in this paper, but it is useful for psychiatrists to have a guideline for these positive effects of separation and divorce. It is obvious that if separation and divorce lead to the father's abandoning the family—literally or figuratively—detrimental effects are far more likely. By stressing the positive effect of attachment-bonding for the father, one can emphasize the inadequacy of stereotypic thinking about human experience.

It is undeniable that divorce can cause hardship for children and parents, but this is not inevitable. In clinical practice it will increasingly become psychiatrists' responsibility to help divorced (or separated) men and women to cope with their new status. Recognition of the father's potential as an adequate caretaker is essential for the helping person. In addition, it is often necessary that the mother be given the opportunity to recognize, over time, the father's importance to the children. Even reasonably healthy women tend to underestimate the importance of fathers as nurturing figures. Fathers are often viewed as providers, as performers of chores, or as representatives of authority and the "real world." In the optimum postdivorce situation the mother has increasing and regular opportunities to see the child's importance to the father and the father's importance to the child. All but the most retaliatory and sadistic women will learn to view the father as a positive factor in the child's life.

Professionals who treat separated or divorced fathers need to recognize the importance of supporting the father's continued interest in and interaction with his children. In particular, men should be helped to recognize and feel comfortable with their so-called maternal or feminine side. To achieve this, it is essential that periods of visitation or shared custody be adequate for the child and the father to achieve a true attachment to each other. This assertion is contrary to the position of Goldstein and associates (11), which advocates the need for an intense attachment to only one parent. Those authors believe that the minimization of conflict between parents takes precedence over all else.

A shared basic attachment enables a child to identify with and introject parental qualities from the father as well as the mother. The loss to children whose fathers are excluded from the developmental matrix cannot be assessed only in terms of a narcissistic injury leading to depression in the child. The loss of introjected standards, qualities, and characteristics is of equal importance. Psychoanalysts have been slow in recognizing the significance of social change and social role modification as positive factors. There has been a marked tendency to suspect the worst in terms of outcome and in terms of the unconscious motivations of the male parent who wants to truly care for his offspring.

We must ask ourselves whether the concept of role confusion is not based on sexist assumptions. The differences between mothers and fathers that are significant to children are not related to the kind of work, taste, sensibilities, or dress that either possesses: they are genital differences, which a child must come to appreciate and integrate. An in-depth approach to development should lead to the conclusion that superficial interests or activities do not determine gender identity.

This paper has stressed the potential positive effect of separate caring experiences for fathers. It should be added that truly shared caretaking in intact homes also is likely to strengthen the father-child bond and aid the development of a separate relationship, thus improving adjustment for all of those involved in the process.

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Urinary MHPG and Clinical Response to Amitriptyline in Depressed Patients

BY DUANE G. SPIKER, M.D., DAVID EDWARDS, PH.D., ISRAEL HANIN, PH.D., JOHN F. NEIL, M.D., AND DAVID J. KUPFER, M.D.

The authors treated 18 rigorously diagnosed depressed patients with amitriptyline after baseline urine samples were collected for the measurement of 3-methoxy-4-hydroxyphenylglycol (MHPG). Neither age nor severity of depression before treatment correlated with MHPG excretion. There was also no significant correlation between baseline MHPG excretion and clinical response at the end of at least 25 days' treatment with amitriptyline. The authors discuss the relevance of amitriptyline and nortriptyline plasma levels to MHPG and the noradrenergic/serotonergic theories of depression.

In recent years several different attempts have been made to develop laboratory tests that might predict the response of depressed patients to various tricyclic antidepressants. One approach has been to measure urinary excretion of 3-methoxy-4-hydroxyphenylglycol (MHPG). MHPG is believed to be the principal metabolite of central nervous system norepinephrine (1, 2). It was originally studied to test the hypothesis that depression in humans is associated with a deficiency of catecholamines in the CNS (3, 4). Subsequent investigators used MHPG levels to differentiate subtypes of depression (2, 5, 6). Further investigation suggested that the excretion of MHPG might predict the response of depressed patients to different antidepressants (7–12).

In 1972 Maas and associates (7) reported on the relationship between urinary MHPG and response to two different antidepressants. In the first group of 12 patients treated with either imipramine or desmethylimipramine, the group of responders excreted smaller amounts of MHPG than did the group of nonresponders. These findings were replicated in a second series of 16 patients who were all treated with imipramine. In 1974 Maas and associates (8) summarized these data and noted that 8 of 8 depressed female patients with MHPG excretion of less than 1000 μg/24 hours responded to imipramine or desmethylimipramine. In 1975 Beckmann and Goodwin (9) replicated this work using 23 unipolar patients treated with imipramine. They reported that 9 "unequivocal responders" to imipramine had significantly lower MHPG excretion than 7 "unequivocal nonresponders" and that there was no overlap between the two groups. Although these data are impressive, there is one small study with contradictory results. Shopsin and associates (10) reported that 3 depressed women who had urinary MHPG values within the normal range nevertheless responded to imipramine. The reason for this discrepancy is unclear.

The relationship between MHPG excretion and response to amitriptyline has not been as extensively studied. In 1973 Schildkraut (11) reported that of 6 patients treated with amitriptyline, the 3 with the lowest MHPG values failed to respond to the drug, while the 3 patients with the highest values did respond. However, the 2 patients with the lowest values were diagnosed as bipolar. This confounds the results because bipolar depressed patients have consistently been found to have low MHPG excretion (2, 5, 6). In 1975 Beckmann and Goodwin (9) avoided this criticism by reporting results from a group of 16 unipolar patients treated with amitriptyline. They described 4 subjects who were "unequivocal responders" to amitriptyline and 4 who were "unequivocal nonresponders." The responders excreted significantly higher amounts of MHPG than did the nonresponders, and there was no overlap between the two groups. Sacchetti and associates (12) reported that 4 of 5 unipolar men, including 2 with very low MHPG values, responded to amitriptyline. Thus, the relationship between MHPG excretion and response to amitriptyline is less clear than that between MHPG excretion and imipramine.

Based on the above findings and other work, it has been proposed that there may be two biochemically definable types of depression, which have been called Group A and Group B (13, 14). Group A patients are characterized by low pretreatment MHPG excretion, a

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favorable response to imipramine or desmethylimipramine, an elevation in mood after a trial with dextroamphetamine, and a failure to respond to amitriptyline. Based on these observations, Maas has suggested that Group A patients have a depression associated with an alteration in the noradrenergic system. Group B patients are characterized by normal to high pretreatment urinary excretion of MHPG, a favorable response to amitriptyline, a lack of mood change during a trial with dextroamphetamine, and a failure to respond to imipramine. Maas has proposed that Group B patients have an abnormality associated with the serotonergic system (14). However, as he noted, there is at least one potential flaw in this method of classification. Although amitriptyline is probably not an effective blocker of norepinephrine uptake, its metabolite nortriptyline is (14). Consequently, patients who have low pretreatment MHPG excretion and thus presumably have a depression associated with a deficiency of the noradrenergic system should be nonresponders to amitriptyline unless their plasma nortriptyline level is in the therapeutic range. If their nortriptyline level falls within the therapeutic range, they should be responders. To the best of our knowledge, no one has attempted to test the noradrenergic/serotonergic theories of depression in this manner.

The first purpose of this study, therefore, was to attempt to replicate earlier studies which suggested that high baseline urinary excretion of MHPG predicted a favorable response to amitriptyline. The second purpose was to test the noradrenergic/serotonergic theories of depression by measuring plasma nortriptyline levels in patients who had low MHPG excretion and had received an adequate trial of nortriptyline.

METHOD

All subjects were inpatients on the clinical research unit at Western Psychiatric Institute and Clinic. At the time of admission patients were given a traditional psychiatric interview and a complete physical examination. During the subsequent 2-week drug-free period, they underwent a variety of routine laboratory tests including thyroid function tests, an EEG, and any other tests indicated based on their history and physical examination. Any patient who was found to have a medical illness affecting the CNS was excluded from the study. All patients gave informed consent.

At the end of the 2-week drug-free period, the Schedule for Affective Disorders and Schizophrenia (SADS) (15) was completed using information from the initial interview, case records, collateral information from relatives, observation on the University of Pittsburgh and Western Psychiatric Institute and Clinic Clinical Research Unit, and a second interview with each patient. The SADS is a structured research interview that collects information necessary to make diagnoses using the Research Diagnostic Criteria (RDC) (16). For inclusion in the study the patients had to meet the RDC for major depressive disorder, primary, unipolar, and endogenous subtypes. In addition, they could not meet the criteria for the psychotic subtype. The severity of depression was measured by the Hamilton Rating Scale (HRS), using the sum of two raters on the 17-item HRS (17). Patients entered the study if the combined HRS score for two raters was 28 or more. They were subsequently rated twice weekly using the HRS. The patients were then given placebo tablets four times a day for 5–7 days. Amitriptyline was gradually substituted for the placebo tablets until the daily dose reached 200 mg per day. All patients received amitriptyline for a minimum of 25 days of treatment, and for at least the last 14 days they received 200 mg per day.

During the placebo period, at least two 24-hour urine samples were collected and aliquots were frozen at −20° C. MHPG was subsequently measured using the methods of Dekirmenjian and Maas (18). Creatinine was measured using an automated method based on the Folin and Wu procedure (19). Urine collections were considered complete only if the 24-hour creatinine excretion exceeded 1400 mg or 20 mg/kg for men and 1000 mg or 15 mg/kg for women. These values were chosen because they were approximately one standard deviation below the mean values reported in several studies (20–22). Theoretically, these values would exclude only approximately 16% of the urine samples which were in fact complete. This was considered a reasonable rate to accept to ensure that the MHPG values were not skewed downward by the inclusion of incomplete collections. Blood collection and preparation for measurement of plasma levels of amitriptyline and nortriptyline were done as previously described (23). Levels of significance were determined using Pearson's product-moment correlation (24).

RESULTS

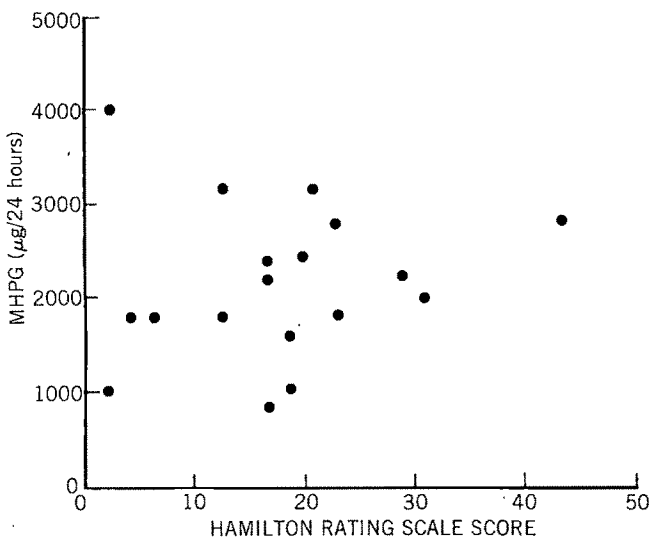
Eighteen patients (5 men and 13 women) had at least two complete 24-hour urine collections during the drug-free period and received an adequate trial of amitriptyline. Their data are shown in table 1. The MHPG values ranged from 725 to 3906 μg/day (mean ±SD=2148±822 μg/day). There was no significant correlation between age and MHPG excretion (r=.09) or between Hamilton scores before treatment with amitriptyline levels and MHPG excretion (r=.10). The baseline MHPG values were plotted against the final Hamilton scores in figure 1. No significant correlation was observed between MHPG and final Hamilton scores (r=.10). There was also no significant correlation between MHPG excretion/creatinine excretion and final Hamilton scores (r=.01). There were 6 clear-cut amitriptyline responders (final Hamilton score of

TABLE 1
Urinary MHPG, Plasma Amitriptyline and Nortriptyline Levels, and Hamilton Rating Scale Scores for 18 Depressed Patients

Patient	Age (years)	MHPG (μg/24 hours)	MHPG/Creatinine (ratio)	Hamilton Scale Score		Amitriptyline (ng/ml)[a]	Nortriptyline (ng/ml)[a]
				Initial	Final[a]		
1	18	725	.66	28	16	150	202
2	36	990	.89	52	2	66	69
3	36	1005	.65	38	18	93	68
4	23	1649	1.78	43	18	98	51
5	34	1793	1.59	30	6	105	126
6	37	1817	1.79	38	12	136	135
7	53	1834	1.85	31	22	115	174
8	36	1868	1.77	38	4	79	17
9	30	1957	1.26	30	30	119	115
10	58	2205	1.15	32	28	218	190
11	27	2295	1.96	40	16	311	221
12	39	2352	1.64	48	16	79	48
13	48	2367	3.19	40	19	97	72
14	28	2775	1.64	48	42	89	48
15	30	2791	1.16	30	22	121	158
16	35	3144	2.62	34	12	—	—
17	27	3198	1.65	46	20	229	152
18	36	3906	1.46	38	2	72	49

[a]Values were obtained at the end of 25 days of treatment with amitriptyline.

FIGURE 1
Baseline MHPG Values and the Final Hamilton Rating Scale Score for 18 Depressed Patients



12 or less), and the MHPG excretion (mean±SD) in these patients was 2253±1065 μg/day. There were also 6 clear-cut nonresponders (final Hamilton score of 20 or more), and the mean MHPG excretion in this group was 2460±541 μg/day.

Table 1 shows the plasma amitriptyline levels at the end of 25 days of treatment with amitriptyline. The plasma level of amitriptyline that is effective to block the serotonergic system is unknown. However, if patients with a normal or high MHPG excretion have a depression associated with the serotonergic system, one would expect some correlation between plasma amitriptyline level and final Hamilton score. Normal MHPG excretion is approximately 1400–1600 μg/day (25–29). In the present study 14 patients had MHPG excretion greater than 1600 μg/day; their plasma amitriptyline levels were determined. There was no significant correlation between final Hamilton scores and plasma amitriptyline levels (r=.15). This correlation would not be significant even if only the 8 patients with MHPG excretion greater than 2000 μg/day (and with plasma amitriptyline levels determined) were evaluated (r=−.45).

Table 1 also shows the plasma nortriptyline levels after 25 days of treatment with amitriptyline. Of the 3 patients with low MHPG values, 2 had plasma nortriptyline levels between 50 and 150 ng/ml. One patient was a clear-cut responder and one was a partial responder (final Hamilton score of less than 20 but more than 12). One patient had a nortriptyline plasma level of 202 ng/ml and was a partial responder.

DISCUSSION

The major purpose of this study was to test the hypothesis that high baseline urinary excretion of MHPG correlates with a favorable response to amitriptyline. Our data do not support this hypothesis. Of the 6 patients in our sample with the highest MHPG values, only 2 responded to amitriptyline. In the whole sample of 18 patients, there was no correlation between baseline MHPG excretion and final Hamilton score. Correcting the data for the daily creatinine excretion did not improve the correlation. A comparison of the unequivocal responders (final Hamilton score of 12 or less) and the unequivocal nonresponders (final Hamilton score of 20 or more) also was not helpful. In fact,

the responders had a lower mean excretion of MHPG than did the nonresponders, although the difference was not statistically significant. The data were also examined using several other statistical approaches, with nonsignificant results.

The reasons for our not finding a correlation between MHPG excretion and response to amitriptyline are not clear. Diagnostically, our patients were well defined and relatively homogeneous. Our criteria for excluding urine samples because they may have been incomplete were more stringent than those of most other studies. This probably explains why the patients' mean MHPG excretion was comparatively high. One possible explanation is that in spite of adequate oral doses, some of the patients did not receive enough amitriptyline. However, the measurement of amitriptyline plasma levels allowed us to control for this variable, and that did not explain the lack of correlation. In addition, the majority of our subjects were routine admissions from the emergency room, and only 4 had had a reasonable prior trial of amitriptyline (patients 12, 13, 14, and 16). Consequently, most of our patients represented fairly typical cases of depression. At any rate, on the basis of our data, one must conclude that there are patients with normal or high baseline MHPG excretion who fail to respond to amitriptyline in spite of an adequate trial.

The interpretation of our data regarding plasma nortriptyline levels, baseline MHPG excretion, and response to amitriptyline in the context of the proposed noradrenergic/serotonergic theories of depression is less conclusive because only 3 of our patients had low MHPG excretion. One hypothesis relevant to this study is that depressed patients who excrete low baseline MHPG may have a depression associated with a deficiency of CNS norepinephrine. Although amitriptyline is not an effective blocker of the reuptake of norepinephrine, nortriptyline is. Consequently, patients who excrete low baseline MHPG should not respond to amitriptyline unless their plasma nortriptyline level is within the therapeutic range (14). Although there is some question about the upper limits, a reasonable estimate of this range is 50–150 ng/ml (30–33). Of the 3 patients in our study with low MHPG values, 2 had nortriptyline values within this range (see table 1). As noted previously, one of those was a responder and the other a partial responder. Sacchetti and associates (12) have also reported on patients with low MHPG excretion who responded to amitriptyline. In a sample of 5 subjects, 3 had low MHPG excretion and 2 of these 3 responded to amitriptyline. Sacchetti and associates' data could be used as an argument against the noradrenergic theory of depression. However, our data suggest that this apparent discrepancy may be explained by the noradrenergic blocking effect of nortriptyline. Further study in this area is indicated, and obviously plasma tricyclic antidepressant levels should be measured.

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The Physical Examination in Office Practice

BY WILLIAM H. ANDERSON, M.D.

Physical illness may first declare itself as a disturbance in thinking, mood, or behavior. Recognition of physical disorders is enhanced by informed use of the relevant procedures of physical examination. Objections to physical examination are based on the assumptions that it is overly time-consuming, is psychotherapeutically contraindicated, or requires unusual skill, but observation and minimally intrusive procedures can provide much information that may exclude emergent organic conditions. Different patients, illnesses, and settings require different levels of examination; the author describes the appropriate procedure for psychotic patients, substance abusers, patients taking psychotropic medications, and other patients.

Convincing evidence now exists that physical illness may first assert itself as a disturbance in thinking, mood, or behavior (1–3). Early recognition of such conditions is imperative in order that effective treatment begin before complications supervene. As hyperspecialty medicine continues to be a predominant mode of care delivery, the psychiatrist is called on to recognize and sometimes to treat these conditions. It is possible that the internist may not have special skill in this area and may ascribe such disturbances to psychological causes.

The physical examination supplies data for the logical process of differential diagnosis. It is a procedure that must be congruent with the clinical situation. The diagnostic process generates hypotheses from the patient's history (4), attempts to support these hypotheses by observation, examination, and laboratory findings, and orders the winnowed hypotheses according to the extent of critical need for action (5). Of the plausible diagnostic hypotheses, the one that leads to a specific effective treatment should be entertained first.

When we replace the concept of physical examination as ritual with the concept of hypothesis confirmation, the usual objections to such procedures can be seen in perspective. All patients thus require a "physical examination," but the specific content must be a variable set of procedures that depend on the clinical context. If we are told by another physician that "the physical examination is negative" for a psychotic patient, this is not highly informative, as we are unaware of the physician's opinion as to which observations are relevant to the differential diagnostic questions involved. If we are told that the temperature is 37° C, the pulse is 84, the pupils are equal and reactive at 3 mm and there is no diaphoresis, we have some useful information which helps us to rank our diagnostic hypotheses in a rational order of plausibility.

The general problem, therefore, is to erect a hierarchy of examination techniques that identify serious conditions when present and yet do not subject the patient to an involved ritual of low-yield procedures. This may be accomplished by having one physical examination procedure for psychotic patients, one for possible substance abusers, one for those taking psychotropic medications, and one for all others. I will consider each of these groups separately.

Five reasons have been advanced for psychiatrists' avoidance of physical examinations (6–7). Most of these have grown out of the implicit assumption that the physical examination is a single standard procedure. To the contrary, since its purpose is to confirm or refute specific diagnostic hypotheses generated by the patient's history, the proper physical examination ought to have different areas of emphasis depending on the illness and situation. It will surprise no one that a cardiologist and an ophthalmologist will have greatly different skills, yet each is capable of performing the relevant areas of a physical evaluation. In the case of the psychiatrist, the relevant skills include the mental status examination and all other procedures and observations that discriminate between organic brain syndromes and functional conditions.

OBJECTIONS TO PHYSICAL EXAMINATIONS

1. Other physicians have greater skill in performing physical examinations. Surely the cardiologist is more astute at detecting heart murmurs, but this skill is not pertinent to the psychiatrist's task. No physician will have more skill than the psychiatrist at taking the

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blood pressure, pulse, or temperature, noting movement disorders, or observing signs of autonomic dysfunction. It is likely that an internist will have less skill in certain of these critical areas than the psychiatrist, who sees many more patients who have subtle extrapyramidal and autonomic disturbances secondary to treatment with psychotropic drugs.

2. It is impractical in office practice to have the patient disrobe. This is true, yet for the relevant observations and procedures disrobing is most often unnecessary.

3. The physical examination gives a low yield of positive findings. The truth or falsity of this statement depends on the patient and circumstances. For the healthy outpatient it is usually true, but for an elderly psychotic patient in the emergency room it is usually false.

4. The performance of a physical examination poses unmanageable transference and countertransference problems. If this formulation is ever true, it applies only to those patients and psychiatrists who are currently involved together in psychoanalytic psychotherapy. This is a subset of psychiatric intervention for which diagnosis is not pertinent.

5. Performing a physical examination would expose the psychiatrist to risk of malpractice litigation. Surely the reverse is true. To look for a significant abnormality and not find it is clearly more defensible than to avoid looking altogether. As physicians responsible for the patient's care, we are obligated to have direct knowledge of those physical findings which are related to the disturbance at hand.

PHYSICAL EXAMINATION FOR COMMON TYPES OF PATIENTS

Psychotic Patients

Patients with psychosis, especially in its acute forms, require that careful attention be paid to certain physical findings in order to exclude the variety of medical conditions which may resemble manic-depressive disease or schizophrenia. Careful history and laboratory examination are also invaluable and are discussed elsewhere (8).

The relevant aspects of physical examination for psychotic patients are neither intrusive nor time-consuming. Most often they can be accomplished by the clinician's deft, firm, and gentle manner. Examination can usually take place without resorting to antipsychotic drugs before diagnosis; for very agitated patients this ideal is not always practical. In such cases a small dose of high-potency antipsychotic drug (e.g., 5 mg of haloperidol) may be injected to facilitate the examination.

There are many possible medical causes of a psychotic syndrome (9). In office practice it is generally not practical to exclude them all. It is therefore necessary to order the index of suspicion such that the most serious possible causes are considered first. Fortunately these are few in number and most often discernible by brief examination:

1. Infectious diseases. In cases of meningitis and encephalitis fever, headache, and pain on neck flexion are important to identify. Systemic infections such as subacute bacterial endocarditis cause fever and tachycardia.

2. Toxic conditions. Poisoning by tricyclic antidepressants or other anticholinergic agents may be recognized by tachycardia, fixed dilated pupils, flushed dry skin, and diminished bowel sounds. Delirium tremens (sedative withdrawal syndrome) is characterized by tremor, hyperreflexia, diaphoresis, fever, mydriasis, and tachycardia.

3. Metabolic and endocrine disorders. Hypoglycemia is suggested by diaphoresis and tachycardia, especially in an insulin-dependent diabetic. Cerebral hypoxia is suggested by history of cardiopulmonary disease with changes in vital signs. Wernicke's encephalopathy may be recognized by confusion, nystagmus, ataxia, and ophthalmoplegia. Thyrotoxicosis is suggested by enlarged thyroid, tachycardia exophthalmus, and diaphoresis.

4. Vascular conditions. Intracranial hemorrhage is suggested by sudden onset of headache with nuchal rigidity. Hypertensive encephalopathy is excluded by a relatively normal blood pressure.

While this is not an exhaustive list of the organic brain syndromes that may mimic functional psychosis, it contains most of those conditions which may cause death or serious impairment in a very short time. The principal physical findings do not require excessive manipulation or more than usual experience and dexterity.

In summary, the initial physical examination of the psychotic patient should include at least the following: 1) vital signs, especially temperature; 2) general appearance—whether the patient is neatly dressed, which would suggest very acute onset; 3) head—any signs of injury; 4) eyes—pupil size and dynamics, presence of nystagmus or exophthalmus; 5) neck—rigidity or thyroid enlargement; 6) skin color and perspiration level; 7) hands—presence of tremor, asterixis, or chorea; 8) reflexes—whether they are hyperactive or asymmetrical and whether the suck, snout, and grasp reflexes are present.

Possible Substance Abusers

Patients who may have high risk of substance abuse because of history or demographic factors require a special set of observations. Findings may differ depending on the drugs in question and whether the current state is one of intoxication or withdrawal.

Visible signs of opiate use include miosis and a smiling, sleepy appearance. Needle marks are generally concealed by clothing. Signs of withdrawal include

mydriasis, diaphoresis, gooseflesh, yawning, lacrimation, and rhinorrhoea.

Amphetamine, cocaine, and methylphenidate intoxication may cause diaphoresis, tachycardia, and elevated blood pressure. The patient is alert or even hypervigilant. The pupils are dilated and reactive to light. After abrupt withdrawal, lethargy and depression may occur. Drugs of the cannabis type produce tachycardia and reddened conjunctivae. Phencyclidine intoxication produces ataxia and nystagmus (10).

Acute intoxication from alcohol, barbiturates, or related sedative-hypnotics causes ataxia, slurred speech, and nystagmus. Chronic use of alcohol may sometimes produce facial erythema and edema or hoarseness. Early or mild withdrawal signs include tremor and diaphoresis. Later and more severe signs of withdrawal are tachycardia, mydriasis, hyperreflexia, and fever.

Atropine, scopolamine, and related drugs are occasionally abused. More frequently, patients develop anticholinergic toxicity from prescribed antipsychotics, antidepressants, and antiparkinson drugs. Recognition of these signs is described below.

Thus, the initial examination for the substance abuser should include at least the following: 1) vital signs; 2) general appearance—ataxic gait, slurred speech, or hoarseness; 3) skin—degree of moistness, presence of flushing, gooseflesh, or needlemarks; 4) eyes—pupil size and reactivity and presence of nystagmus or red or jaundiced sclera.

Patients Taking Psychotropic Medications

Adverse effects of psychotropic drugs depend on the pharmacologic class of drug and the clinical time frame. Some of those which arise in the initial weeks of treatment are recognized by history rather than physical findings. Examples are blood dyscrasias, liver disease, and allergic phenomena, which are serious but will not be considered here. I will examine only those conditions which are best identified by observation.

Antipsychotic drugs. Observable adverse effects are best considered as early (occurring in the first 10 weeks of treatment) and late (occurring months or years after treatment begins). Early observable effects consist of the Parkinson spectrum of movement disorders and anticholinergic effects.

The Parkinson syndrome spectrum consists of akinesia (which may be mistaken for depression or the flat affect of schizophrenia [11]), dystonia (sometimes mistaken for hysteria), akathisia (sometimes thought to be anxiety or psychotic agitation), and pill-rolling tremor. During the early weeks of antipsychotic treatment these effects should be anticipated and, if necessary, treated by dosage reduction or antiparkinson medication. Cogwheel rigidity is frequently discernible in the wrist joints even when other manifestations of the syndrome are minimal.

Anticholinergic action is characteristic of all antipsychotic drugs but is most pronounced in the lower potency ones such as chlorpromazine and thioridazine. Observable anticholinergic effects include tachycardia, mydriasis, fever, and diminished bowel sounds. Mental status changes, including psychosis, may occur as well. It is essential to recognize this possibility lest the anticholinergic psychosis be treated with a higher dose of antipsychotic drug, which would exacerbate the condition.

Later observable effects of antipsychotic drugs include tardive dyskinesias and skin and eye changes. The first signs of tardive dyskinesia are usually seen in the tongue, lips, and fingers. The essential feature of these abnormal movements is randomness. The tongue cannot be protruded steadily for a period of more than a few seconds. Fine vermicular movements may be seen within it. The lips may smack and pucker at random. The fourth and fifth fingers may flicker involuntarily.

Peculiarities of skin pigmentation may occasionally be seen. These are usually gray to violet, more marked in areas exposed to the sun, and frequently reversible.

Deposits of particulate matter may occasionally be seen in the cornea and lens of the eye. These are frequently reversible and may not substantially interfere with visual acuity. Thioridazine in doses above 800 mg per day has been reported to cause nonreversible and visually damaging pigmentary retinopathy.

Tricyclic antidepressants. The chief observable adverse effects of these drugs are anticholinergic, as described above, and orthostatic hypotension.

Antianxiety agents. Signs of intoxication include sedation, slurred speech, and ataxia. Signs of withdrawal may be tremor, diaphoresis, and tachycardia.

Lithium carbonate. Patients receiving this drug may be seen to have neuromuscular disturbances even at normally therapeutic levels, although generally they are seen when the lithium concentration in the blood is above usual. The major finding is essential tremor, which may be seen in the outstretched fingers and heard as a quavering of the voice. As lithium is an antithyroid drug, occasionally a euthyroid or hypothyroid goiter develops. Periodic palpation of the gland is therefore indicated.

The physical examination for patients taking psychotropic medication should thus include the following: 1) vital signs; 2) skin—moist versus dry, unusual pigment; 3) eyes—papillary dynamics, visual acuity, color of sclera, opacities of cornea and lens; 4) neck—thyroid enlargement; 5) neurological functioning: Parkinson's syndrome—akinesia, akathisia, tremor, cogwheel rigidity, and gait; tardive dyskinesia—tongue, lips, fingers; and essential tremor.

All Other Patients

In the usual office practice the typical patient on his or her first visit will not fall into the above three cate-

gories. The history will exclude many diagnostic entities that may coexist with psychiatric complaints. If there are subjective physical complaints the patient may reasonably be instructed to have these investigated by a general internist. Here the physical examination should consist mainly of a mental status examination and observation and should involve minimal procedures.

The general observations should include checking skin color for suggestions of pallor or jaundice. (For patients with marked pigmentation, their palpebral conjunctivae may be checked.) The quality of the voice may raise the possibility of hypothyroidism (a deep croak) or chronic alcoholism (hoarseness). The patient's gait and hand movements may suggest the presence of tremor or chorea. The hands may show clubbing of the fingers (suggestive of cardiopulmonary or gastrointestinal disease) or edema. The proper procedures include a handshake to judge the amount of sweating; diaphoresis might imply anxiety, hyperthyroidism, or sedative withdrawal. Taking the pulse is hardly an intrusive procedure and may yield information concerning arrhythmia or autonomic function. Blood pressure might also be checked, not because hypertension is typically associated with psychiatric manifestations, but rather as a simple check for a very prevalent and damaging treatable disease. If any of these observations suggests abnormality the psychiatrist might order laboratory tests or make a referral as appropriate.

The patient who is not psychotic, a substance abuser, or a taker of psychotropic medications may nevertheless benefit from any of the above observations but especially from having his or her pulse taken and from observation of his or her general appearance, e.g., grooming, cleanliness, appropriate dress, unusual odors, voice, skin, hair, hands (clubbing, edema, sweating).

The Neurological Examination

Whenever a psychiatric patient gives a history suggestive of neurological impairment, a detailed examination is warranted. The complexity and subtlety of such an examination may tend to discourage the psychiatrist. Nevertheless, it is important to be aware that some neurological examination procedures are especially useful. While not a substitute for a full examination by a neurologist under ideal conditions, the following procedures are included here because of their relative simplicity and high yield in terms of likelihood of discovery of pertinent findings:

Mental status. Defects of recent memory, orientation, and fund of information suggest organicity. Tactile hallucinations suggest toxic or metabolic encephalopathy, delirium tremens, or temporal lobe epilepsy.

Cranial nerves. Tests of the olfactory and optic nerves are the most useful.

Motor system. Weakness may be roughly assessed by looking for drift of outstretched arms with the eyes closed.

Sensory examination. The most useful procedures are double simultaneous stimulation, stereognosis, and stereographia.

Cerebellum. Nystagmus and past pointing tests may be useful.

Reflexes. Increased deep tendon reflexes suggest upper motor lesions and alcohol and sedative withdrawal. Useful pathologic reflexes are grasp, palmomental, suck, and glabellar tap.

COMMENT

Generally it is the patient with acute psychotic illness who most requires evaluation by the set of procedures described in this article. Even in the case of psychosis the relevant data may be obtained with observations and procedures that are only minimally intrusive. Of course, all psychiatric patients deserve and require careful medical screening. Those who are psychotic, those who may be substance abusers, and those taking psychotropic medication may especially benefit from early recognition of medical conditions. All patients should have whatever screening is clinically indicated in the opinion of the psychiatrist or the patient's general physician. The procedures described here are not intended to replace the general physical and neurological examination properly used on admission to an inpatient service. Instead, this method is useful for early identification of serious medical illness that may initially declare itself as behavioral change. Such patients often enter the health care system through a psychiatrist's office or by psychiatric consultation in the emergency department. Psychiatrists must be justly confident of their capacity to recognize medical illness that causes or coexists with the disturbances of thinking, feeling, and acting, which are our professional concern. The relevant physical examination is an essential tool for the accomplishment of this purpose.

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The Manfred S. Guttmacher Award

The American Psychiatric Association invites applications for its biennial Manfred S. Guttmacher Award for 1981. The award is now given in odd-numbered years for outstanding contribution to the literature of forensic psychiatry in the form of a book, monograph, paper, or other work, including audio-visual presentations (films, etc.), submitted to or presented at any professional meeting or published during the period from December 1, 1978, to December 1, 1980. A plaque and an honorarium of $750 will be presented to the award recipient at the Convocation of Fellows at the 1981 APA annual meeting. Please note that the award recipient is now required to give a lecture at the annual meeting.

Anyone who wishes may apply by submitting five copies of the work as well as five abstracts to Robert L. Sadoff, M.D., Chairperson, Guttmacher Award Board, 1700 Eighteenth Street, N.W., Washington, D.C. 20009. Entries will be acknowledged but will not be returned.

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The Phenomenology of Near-Death Experiences

BY BRUCE GREYSON, M.D., AND IAN STEVENSON, M.D.

The authors studied retrospectively 78 reports of "near-death experiences" using subjects' narratives and questionnaires, interviews, and medical records. Prior experiences suggestive of transcendence of death were more common among these subjects than among control populations, but prior experiences suggestive of extrasensory phenomena were less common. Subsequent changes in attitudes were more common than among subjects in other studies who had had psychic experiences. The influence of cultural and psychological factors, sensory deprivation, and reflex adaptive responses to stress explain some but not all of the features of near-death experiences. Their potential value to our conceptualization of dying and to suicide prevention and the care of the terminally ill justifies further investigation.

Profound subjective events experienced on the threshold of death, so-called "near-death experiences," have been reported by people who have been seriously injured or ill but unexpectedly recovered and by people who had anticipated imminent death in potentially fatal situations but escaped uninjured (1). The investigation of near-death experiences may contribute not only to our understanding of the dying process but to our care of terminally ill patients, our ability to help grieving families, and our approach to suicidal patients. In this paper we describe the characteristic features of a large self-selected sample of people who had had near-death experiences and discuss the implications of these data for the interpretation of such phenomena.

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METHOD

We retrospectively collected 78 reports of near-death experiences from three self-selected sources: letters to a national magazine from correspondents who had read an article on near-death experiences (18 cases), responses to announcements of our interest in studying such cases in professional newsletters and in magazines that specialize in popular articles about parapsychological phenomena (22 cases), and communications from informants who knew of the long-standing interest of one of us (I.S.) in the nature of death (38 cases).

For each case we obtained a firsthand written or tape-recorded narrative account from the person reporting the experience and supplemented these accounts whenever possible by a detailed questionnaire, personal interview, and examination of medical records relevant to the close brush with death.

We transferred characteristics of each respondent's reported near-death experience to a checklist, either directly from questionnaire responses (38 cases) or by extracting such data as were available from the narrative report, interview, and medical records of those respondents who did not complete the questionnaire (40 cases). Frequencies of individual items were then computed, and certain cross-tabulations, selected for their relevance to explanatory hypotheses were evaluated by chi-square tests.

RESULTS

Predisposing and Precipitating Factors

At the time of the near-death experience 27% of our respondents were 18 years old or younger, 32% were 19 to 35 years old, and 40% were older than 35 years. All of our subjects were Caucasian; 63% were women. At the time of the near-death experience 47% were married, 40% were single, 6% were separated or divorced, and 6% were widowed.

At the time of the study 76% had completed high school, and 53% had attended college. Our respondents included executives and professionals (25%), service workers and laborers (29%), and students and housewives (31%). Fifty-eight percent of our sample were Protestant, 18% Catholic, 4% adherents to Eastern faiths, 8% agnostics; none were Jewish or Muslim.

The incidences of apparent mystical or paranormal

TABLE 1
Mystical or Paranormal Experiences in Subjects Who Later Had Near-Death Experiences, the General Population (2), and Psychiatric Inpatients (3)

Experience	Percent of Group: Subjects with Near-Death Experiences (N=78)	General Population (N=354)	Psychiatric Inpatients (N=92)
More frequent among people with near-death experiences			
Sense of unity with nature	50	28	—
Sense of God within oneself	37		
Apparent perception of persons not present	48	17	30
Apparent memory of a previous life	43	8	—
Auras/halos sensed around others	29	5	—
"Out-of-body experiences"	27	14	19
Apparent communication with the dead	24	8	15
Less frequent among people with near-death experiences			
Apparent clairvoyance or precognition while awake	31	38	47
Apparent clairvoyant or precognitive dreams	28	36	40
Figuring in another person's apparent psychic experience	13	18	21
Seeing objects move without apparent cause	5	8	—

experiences that occurred before the near-death experience are summarized in table 1, where they are compared with data from previous surveys of the general population (2) and of psychiatric inpatients (3). In addition, 28% of our respondents reported having had a close brush with death prior to the one being investigated, and 25% acknowledged having read something about near-death experiences before having this close brush with death.

Before the near-death experience 50% of our respondents had expected dying to be painful; only 3% had expected it to be pleasant. Seventy-seven percent had expected to pass into some unearthly realm of existence after death, 63% had expected to be reunited with deceased acquaintances, 29% had expected to be reborn on earth, and 17% had some other expectation of postmortem survival; 35% had expected to cease existing at or shortly after death. Thirty-two percent stated that they either wanted to die or were ambivalent about dying at the time of the near-death experience.

Medical personnel were present during or immediately after the near-death experience in 80% of our cases. The experience was precipitated by medical illness in 40% of our cases, by traumatic injury in 37% (of which 70% were accidental), by surgical operation in 13%, by childbirth in 7%, and by drug ingestion in 4%. Forty-seven percent of the subjects reported having taken some type of drug or alcohol on the day of the experience.

Forty-one percent of our subjects reported that they had been considered dead during the near-death experience; an additional 41% reported some degree of physical injury, and 18% reported escaping the close brush with death without any injury. Thirty percent of our respondents reported that the duration of the experience was more than an hour; 67% reported that it seemed to last more than an hour.

Features of Near-Death Experiences

The impression of feeling or seeing oneself to be outside the physical body, an "out-of-the-body experience," was reported by 75% of our respondents. The apparent exit from the body was most commonly described as easy (by 96% of those reporting out-of-the-body experiences) and instantaneous (68%). In 66% of our cases the initial impression that one was out of the body was reported as due to seeing one's body as if from another point in space.

Ninety-five percent of those reporting out-of-the-body experiences stated that they seemed to move about while seemingly out of the body, most commonly within a limited area a few yards from the body (45%); 71% claimed that they could see and hear persons physically present. The apparent re-entry into the physical body was most commonly described as easy (79%) and instantaneous (71%).

The impression of having some sort of nonphysical body separate from the physical body was reported by 58% of our respondents (77% of those reporting out-of-the-body experiences). The nonphysical body was most commonly described as lighter in weight than the respondent's physical body (74%) but the same size (68%) and the same age (84%). The nonphysical body was described as showing some indication of "life" (e.g., pulse, breath) by 67% of those reporting a nonphysical body and as "linked" to the physical body in some way by 28%. Twenty percent of those reporting a nonphysical body claimed that sensorimotor or structural defects present in their physical body (e.g., partial deafness, missing limbs) were absent in the nonphysical body; 3% reported such defects to be present in the nonphysical body.

The impression of passing through a tunnel or similar structure was reported by 31% of our respondents, more frequently by executives and professionals than by laborers and service workers ($\chi^2=14.75$, df=3, $p<.01$). Seeming to enter some unearthly realm of existence was reported by 72% of our subjects, more frequently by women than by men ($\chi^2=7.58$, df=1, $p<.05$); such reports were not correlated with prior beliefs or expectations. Seeming to reach a border or "point of no return" was reported by 57% of our respondents.

Forty-nine percent of our subjects reported seeming to meet some person(s) not physically present; these

persons included a "being of light" (not otherwise identifiable) (27%), religious figures (25%), deceased acquaintances (16%), living acquaintances (14%), and unidentified strangers (26%). These apparent encounters with persons not physically present were more likely to be reported as part of a near-death experience occurring at home or outdoors than as part of one that occurred in a hospital or other public place ($\chi^2=14.95$, df=4, $p<.01$). There was no correlation between such apparent meetings and prior beliefs or expectations. Thirteen percent of our respondents reported a feeling of being judged or held accountable for their lives.

Unusual visual phenomena (e.g., lights, auras) were reported by 48% of our respondents and occurred predominantly during nighttime near-death experiences ($\chi^2=13.42$, df=2, $p<.01$). Unusual somatic sensations (e.g., warmth, analgesia) were reported by 71% of our respondents, unusual auditory phenomena (e.g., music, noises) by 57%, unusual olfactory phenomena by 25%, and unusual gustatory phenomena by 10%. There were no significant diurnal variations in reported sensations other than visual phenomena.

Distortions of the sense of time were reported by 79% of our respondents; 54% reported that time seemed to pass more slowly than usual during the near-death experience. This subjective slowing of time was inversely correlated with the use of drugs or alcohol on the day of the experience ($\chi^2=5.88$, df=2, $p<.05$) and with prior religiosity ($\chi^2=7.44$, df=2, $p<.05$).

Apparent extrasensory experiences during the near-death experiences were reported by 39% of our respondents; 21% claimed that during the experience he or she figured in another person's psychic experience. Reports of extrasensory events during a near-death experience were not correlated with the respondent's prior knowledge about parapsychological phenomena, nor with reports of prior experiences suggestive of parapsychological phenomena.

Fifty-two percent of our subjects reported having believed during the near-death experience that they were dying. This belief was inversely correlated with the subjective duration of the experience ($\chi^2=7.38$, df=2, $p<.05$); that is, the longer the experience seemed to last, the less likely the subject was to believe that he or she was indeed dying.

Review of past events, or "panoramic memory," was reported by 27% of our respondents; memories were most commonly described as appearing all at once (38%) rather than in some particular sequence.

The near-death experience was reported to have been a very positive affective experience by 15% of our respondents, mildly positive by 40%, neutral or mildly negative by 45%, and very negative by none. Positive affect was significantly correlated with distortions in one's sense of time ($\chi^2=6.89$, df=2, $p<.05$).

No single feature of the near-death experience was significantly correlated with the respondent's having had one previously or with the extent of the respondent's prior knowledge about near-death experiences.

TABLE 2
Percent of People Who Reported Changes in Attitudes After a Near-Death or Psychic Experience (2)

Subject of Attitude Change	Near-Death Experience (N=78)	Psychic Experience (N=354)
God or religion	75	23
Self	74	27
Own death	73	—
Death in general	72	13
Life and its meaning, health	64	25
Humanity	55	17
Suicide	50	—
Family, friends	50	17
Nature	45	12
Material possessions	45	9
Sex, marriage	42	16
Personal fame or power	42	5
War, murder	37	5
Education	37	6
Science	34	5
Society	29	6
Business	29	6

Sequelae

The near-death experiences reported in this study occurred 1-67 years before our investigation (median=30 years). At the time of the investigation, 60% of our respondents claimed to have detailed recollections of the experience, and 83% claimed that the memory of the near-death experience was clearer than his or her memories of other events from the same time period.

Respondents' changes in attitudes after their near-death experiences are presented in table 2, where they are compared with data on attitude changes following psychic experiences among the general population (2). Subsequent changes in attitude were more frequent among those of our respondents who believed during the experience that they were indeed dying ($\chi^2=5.17$, df=1, $p<.05$) and among those who reported panoramic memory experiences ($\chi^2=4.02$, df=1, $p<.05$) but were not correlated with any factor prior to or precipitating the near-death experience.

DISCUSSION

Because our cases were not randomly selected, the preponderance of whites, women, and single persons among our respondents may reflect sampling biases. Our subjects were characteristic of white Americans in terms of education and occupation but included a disproportionate majority of Protestants.

Our respondents as a group shared a distinct pattern of reported mystical and paranormal experiences prior to the near-death experience: they acknowledged a

higher incidence than either the general population or identified psychiatric patients of experiences that might suggest transcendence of or survival after death but a lower incidence of prior apparent extrasensory phenomena than either the general population or the sample of psychiatric patients.

The correlations in our data between occupation and seeming to enter a tunnel and between sex and seeming to enter another realm of existence suggest cultural or psychological factors influencing the perception or report of a near-death experience. Despite the absence of correlation between prior beliefs and specific features of the experience, the finding that our respondents also reported prior experiences suggesting transcendence of death more often than comparison groups further supports an influence of prior experiences on reports of near-death experiences.

Comer and associates (4) and Noyes (5) suggested that sensory deprivation may produce phenomena similar to those reported in near-death experiences. In our data, the incidence of unusual visual phenomena in near-death experiences occurring at night was higher than that during daylight hours, which may suggest the influence of decreased sensory input; this diurnal variation in unusual sensory phenomena was not reported for other sensory modalities that would not be affected by darkness. Our finding that apparent meetings with persons not physically present were reported more frequently in near-death experiences occurring in isolation (outdoors, at home) than in those occurring in public buildings may be related to the relative lack of sensory stimulation in the former settings.

Noyes and Kletti (6) suggested that the apparent separation from the physical body, the impression of time moving more slowly, and the hyperalert but narrowly focused cognitive and sensory functioning seen in near-death experiences constitute a reflex adaptive response to a life-threatening danger: potentially fatal crises may induce a focusing on accelerated internal processes, producing a comparative subjective slowing of external time and a concomitant feeling of depersonalization. The correlation in our series between positive affect during the near-death experience and distortion of one's sense of time is compatible with the hypothesis that temporal slowing acts as a defense mechanism. The inverse correlation between the impression of temporal slowing and religiosity also supports Noyes and Kletti's interpretation of near-death experiences if it is assumed that death may be perceived as more threatening by the less religious. Our finding that the subjective slowing of time was reported significantly less often by those who had taken drugs or alcohol on the day of the experience suggests that certain drug effects may impede the reflex response of internal focusing; consequently a near-death experience occurring in a drug-free individual may paradoxically diverge more from the normal state of consciousness than would a drug-inhibited near-death experience. Osis and Haraldsson (7) reported that drug intoxication during a close brush with death diminished the likelihood of a near-death experience.

The interpretation of near-death experiences as adaptive responses does not adequately explain the mystical components of these experiences (1, 6). Melges and associates (8) suggested that temporal disorganization and depersonalization, both seen in near-death experiences, may produce the mistaken impression of paranormal connections between unrelated events. Our finding that apparent extrasensory experiences during the near-death experience were not correlated either with subjective slowing of time or with reported out-of-the-body experiences does not support this hypothesis.

The reported effects of the near-death experience on the subsequent attitudes of our respondents are consistent with those reported previously (5, 6) and are far more pervasive than reported effects of other mystical or paranormal experiences in the general population. If near-death experiences are suggestive of postmortem survival, as those who report such experiences commonly claim (5–7), then their potential contribution to our understanding of death, and of the process of dying, alone justifies further investigation. On the other hand, even if near-death experiences are the epiphenomena of adaptive defense mechanisms or transient delirious states, their profound impact on subsequent attitudes justifies their further study. We suggest that further investigations of near-death experiences be conducted prospectively on patients who are at high risk for such experiences, such as a cohort of coronary care unit patients, in order to eliminate the sampling biases that have occurred in retrospective studies and the possible errors of interpretation attendant on such biases.

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Nightmares: Clinical Characteristics and Personality Patterns

BY ANTHONY KALES, M.D., CONSTANTIN R. SOLDATOS, M.D., ALEX B. CALDWELL, PH.D., DENNIS S. CHARNEY, M.D., JOYCE D. KALES, M.D., DAVID MARKEL, M.D., AND ROGER CADIEUX, M.D.

The authors evaluated the development and clinical course of nightmares and the personality patterns of people with this disorder by studying 30 adults who had a current complaint of nightmares. Nightmares usually began in childhood or adolescence and were chronic. Psychological factors played a major role in the development and persistence of nightmares, major life events were often associated with the onset of the disorder, mental stress usually increased the frequency of events, and psychological testing indicated relatively high levels of psychopathology. Nightmare sufferers in general were distrustful, alienated, and emotionally estranged, and many showed a chronic schizoid pattern of adjustment, but they were not overtly psychotic.

Nightmares are nocturnal episodes of intense anxiety and fear associated with a vivid and emotionally charged dream experience. Nightmares are common in children but are not infrequent in adults; in a survey of a large metropolitan area (1), 5.3% of the adults reported a current complaint of nightmares.

The word "nightmares" traditionally has been used to refer to both actual nightmares, which are the common frightening or bad dreams, and the much less common night terrors (pavor nocturnus in children and incubus attack in adults). Nightmares occur during REM sleep (2, 3), whereas night terrors occur in slow wave sleep (stages 3 and 4) (3–5). In most cases, however, nightmares can be easily differentiated from night terrors on the basis of their general clinical characteristics. The night terror is accompanied by much more anxiety, vocalization, motility, and autonomic discharge. Also, whereas there is amnesia or very little recall of the night terror event, there typically is detailed recall of the nightmare (2–6).

Most clinical research on nightmares has focused on the content of terrifying dreams, particularly in childhood, and their psychological meaning (7–10). Thus there is little general clinical information available on this disorder in adults. The primary goals of this study were to assess the development and course of nightmares, clinical characteristics, and psychological correlates in adults, including personality patterns.

METHOD

We evaluated 30 adult subjects with a current complaint of nightmares. Their mean age was 39.1 years (range, 19–77 years); 27 were women and 3 were men. All of the subjects responded to an advertisement by our Sleep Research and Treatment Center for individuals with a current or past problem of nightmares. To qualify for the study, each subject had to have a frequency of at least 12 nightmares per year.

Each subject participated in an interview that was structured to obtain information on the clinical characteristics of his or her nightmares, and each completed the Minnesota Multiphasic Personality Inventory (MMPI) as well as a comprehensive sleep questionnaire. A subsample of 20 subjects with a current complaint of nightmares whose mean age was 39.7 years (range, 19–77) also completed a special questionnaire that provided additional dream history data, the Symptom Check List (SCL-90), and information on personal and family mental health.

The general sleep history data as well as the MMPI scores of the group of subjects with nightmares were compared with those of a group of 30 age- and sex-matched control subjects. The control subjects, who were required to have negative histories for past and current sleep disorders, were also recruited through a newspaper advertisement.

The dream data were analyzed by chi-square tests, except for the number of nights per year when dreams were recalled, which were analyzed using Student's t test. Data from the mental health history were ana-

A preliminary report of some of these data was presented at the annual meeting of the Association of the Psychophysiological Study of Sleep, Houston, Tex., April 1977. Received Aug. 13, 1979; revised Jan. 28, 1980; accepted March 11, 1980.

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lyzed by chi-square tests. Individual MMPIs were considered invalid if more than 20 of the 550 questions were left unanswered, if the raw score on the F scale was higher than 22, or if both the raw score on the F scale was between 17 through 22 inclusive and the difference between the raw F minus raw K scores was more than 11. The MMPI data were analyzed in terms of absolute elevations of the individual scales, by mean elevations within groups, and by code or profile pattern types. The between-group differences in the mean scores for each of the eight major clinical MMPI scales and the averages of the number of pathologically elevated scales per individual (T score of 70 or above) were compared using Student's t test. Chi-square comparisons were made between groups as to the number of individuals with at least one pathologically elevated MMPI scale.

Finally, the raw scores for the nine symptom dimensions and the three global indices of the SCL-90 were scaled on the basis of normative data derived from a nonpatient sample of men and women (11). Comparisons between the 20 subjects with nightmares and 20 matched control subjects for the mean values of the SCL-90 were made using Student's t test. The mental health data for these groups were compared using chi-square tests. The minimum confidence level for all statistical analyses was $p<.05$.

RESULTS

The results are reported in relation to three major areas: 1) the development and clinical course of nightmares, 2) characteristics of dreams and nightmare events, and 3) personality patterns and mental health correlates of nightmare sufferers.

Development and Clinical Course of Nightmares

Half of the group of subjects with nightmares had an age of onset before age 10; two-thirds had experienced nightmares by age 20. The distribution of the age of onset by 10-year epochs was as follows: 1–10 years, 15 subjects; 11–20 years, 5; 21–30 years, 4; 31–40 years, 3; 41–50 years, 1; and 51–60 years, 2. The mean duration of this sleep disorder was 21.6 years (range, 2–50 years). The occurrence of a major life event preceding the onset of the disorder was reported by 60% of the subjects.

The peak frequency for nightmares was about 3 episodes per week; the current frequency was about 2½ episodes per week. A very high percentage of the subjects (90%) reported that mental stress increased the frequency of their episodes. One-third (33%) thought that fatigue increased the frequency of their episodes, and only a few (13%) reported that a change in sleep environment increased the frequency of their episodes.

Positive histories for other current or past sleep disorders were reported by nightmare subjects as follows: 43%, insomnia; 33%, sleeptalking; 17%, sleepwalking; 13%, enuresis; and 7%, night terrors.

Characteristics of Dreams and Nightmare Events

When comparing the general dream data of the subjects with nightmares (independent of their nightmare dreams) with the dream data of the normal control subjects, we found that the subjects with nightmares reported recall for their dreams more nights per year (181.6 versus 90.9, $p<.05$). In addition, these subjects more frequently reported that their dream recall was detailed ($p<.01$) and that their mood was affected the next day by the previous night's dreams ($p<.05$).

Nightmares did not typically occur during any one period of the night. The average time of occurrence of nightmares was 212.3 minutes after sleep onset. Five of the subjects with nightmares were unable to specify a given time of night, although they indicated that their episodes were less likely to occur early in the night. Nineteen of the remaining 25 subjects with nightmares reported a time of occurrence of their episodes that corresponded to the second and the last thirds of the night. The mean reported duration of the nightmare episodes was about 12 minutes (range, 1–25).

Typically, the subjects reported that they spontaneously awakened from their nightmares; awake time following nightmares averaged 23.2 minutes. Usually the content of the nightmare was associated with fear. Specific fears were reported by the following percentages: fear of attack, 73%; fear of falling, 73%; and fear of death, 60%. Thirty percent of the subjects with nightmares reported a feeling of choking or suffocating. Most of them (83%) reported that they often experienced recurring nightmares.

Personality Characteristics and Mental Health Correlates

Table 1 lists the mean values of the clinical MMPI scales for the subjects with nightmares and for their control group. One nightmare subject (a 35-year-old man) had an invalid MMPI and was excluded from the analysis. When compared with their controls, the subjects with nightmares had higher mean values on all eight clinical scales ($p<.01$ by Sign test) (see table 1). The group of subjects with nightmares also had at least one pathologically elevated clinical scale two and a half times as often as the normal control group (68% versus 26%, $p<.01$). The mean number of elevated scales per individual for the nightmare subjects was 2.6, versus 0.3 for the control group ($p<.01$). The subjects who had an age of onset of the nightmare disorder after age 18 had relatively higher scores on every clinical MMPI scale except for mania in comparison with the subjects who had an earlier age of onset.

The three highest mean scores for the subjects with nightmares were, from highest to lowest, for the scales of psychopathic deviate, schizophrenia, and hysteria.

TABLE 1
MMPI Findings in Subjects with Nightmares and Normal Control Subjects

MMPI Scale	Subjects with Nightmares		Normal Control Subjects	
	Mean	SE	Mean	SE
Hypochondriasis (Hs)[a]	63.0	2.1	48.5	1.4
Depression (D)[a]	63.8	2.2	49.2	1.9
Hysteria (Hy)[a]	66.2	1.8	54.0	1.7
Psychopathic deviate (Pd)[a]	69.1	2.4	55.2	1.8
Paranoia (Pa)[a]	63.4	1.8	55.2	1.2
Psychasthenia (Pt)[a]	65.3	2.4	50.3	1.4
Schizophrenia (Sc)[a]	68.8	2.7	52.8	1.7
Mania (Ma)	59.0	2.0	55.6	1.7

[a]The difference between the groups was significant by Student's t test ($p<.01$).

The complete mean profile for the subjects with nightmares coded *'48 37 261*-9. The highest percentages of subjects with nightmares having pathologically elevated scores on a given scale were 48% for psychopathic deviate, 41% for schizophrenia, and 38% for hysteria. Also, the density values for these scales were similar in terms of their being one of the three highest scales for each subject's MMPI pattern: 56.3% for psychopathic deviate, 51.1% for schizophrenia, and 46.6% for hysteria.

The psychopathic deviate and schizophrenia elements were predominant. Not only were these among the three highest scales in 56% and 51% of our profiles, respectively, but 5 of the valid 29 profiles coded 4-8 or 8-4. Subjects whose MMPIs code as 4-8/8-4 are typically described as distrustful, alienated, and estranged (12). They are quick to withdraw emotionally and to overreact to mistreatment or to what they perceive as mistreatment. They lack persistence in the pursuit of goals and repeatedly have difficulties in making the sacrifices that are necessary to maintain meaningful long-term relationships. These characteristics lead to serious difficulties, such as underachievement in school and work, repetitive sexual problems, and repeated involvement in broadly sadomasochistic and mutually destructive relationships. They often seek clinical help at times of situational stress and acute distress, especially when they have alienated the other person in their current primary relationship. When more disturbed and depressed, they may force care and intervention (and thus reassurance against their alienation) by attempting suicide.

The consistency of elevations on the schizophrenia scale in our sample suggests long-term schizoidness and perhaps what Meehl termed "schizotypy" (13). However, there was no indication of any overt psychoticism. Finally, the values for the psychopathic deviate and hysteria scales were among the three highest mean scores for the group as well as among the three highest scales in individual profiles. This indicates a potential for dissociative episodes in our sample.

The SCL-90 scores for the subjects with nightmares were significantly higher on each of the nine symptom dimensions compared with the scores for the control subjects. In addition, the scores for each of the three global indexes were significantly higher ($p<.01$) among the subjects with nightmares; general symptom index, 68.9 versus 54.1; positive symptom total, 61.4 versus 51.0; and positive symptom distress, 70.0 versus 53.4.

Analysis of the questionnaire items pertaining to personal and family mental health showed that more subjects with nightmares than control subjects reported having had emotional problems ($p<.05$) and having considered committing suicide ($p<.01$). Also, more nightmare subjects than control subjects reported emotional problems in their families ($p<.05$) and that family members had committed suicide ($p<.05$).

DISCUSSION

Clinical Course and Characteristics

Among our subjects with nightmares, the nightmares usually began in childhood or adolescence and were a chronic condition. In some cases, however, the onset was in middle life or even later. The high frequency of nightmares at the time of their peak occurrence and at the time when the subjects were evaluated suggests that the clinical course of the disorder is relatively consistent. It is possible, however, that individuals with a low nightmare frequency were less concerned and did not contact us.

Nearly all of the subjects with nightmares were women. It is unlikely that the sex ratio in our sample approximates the sex ratio of people who have nightmares. Nightmares have been reported to be slightly more frequent among women, and even this small difference has been considered a function of societal expectations: to preserve their masculine image, men would be less likely to admit to having nightmares (14). If this assumption is correct, even fewer men with nightmares would be expected to respond to an advertisement such as ours.

In relation to general dream characteristics, our nightmare sufferers in comparison with their controls reported more frequent and intense dreams; they also reported that these dreams had a greater impact on their mood the next day. Our finding of a high prevalence of other sleep disorders in our nightmare patients compared with the prevalence of these disorders in the general population (1) confirms previous observations (15-17). The relatively high prevalence of insomnia in the subjects with nightmares may reflect the sleep disruption directly caused by the nightmares and/or the psychopathology that commonly underlies both insomnia and nightmares. As for the high prevalence of sleeptalking in these subjects, this may be accounted for by the sleeptalking that often occurs during the nightmare episodes themselves.

Etiological Considerations

From a developmental standpoint, childhood nightmares are most often related to specific phases of growth: they are most common during the preschool and early school years. Mack (7) has reviewed and discussed the relationship of nightmares to ego development and to conflict. One could also postulate that neurophysiological changes related to the maturing brain contribute to the occurrence of nightmares during these stages of development.

The persistence of nightmares into adulthood in our subjects appeared to be correlated with the presence of serious psychopathology. Major life events preceded the onset of the disorder in 60% of the subjects, and mental stress increased the frequency of episodes in 90% of the subjects. In addition, the SCL-90 data show high levels of psychopathology for every symptom dimension as well as for the three global indexes. Furthermore, the MMPI data, in addition to showing that these patients had considerable psychopathology, also indicate a common personality pattern for nightmare sufferers (4-8/8-4 MMPI code types). The nightmare sufferers in general were distrustful, alienated, estranged, oversensitive-overreacting, and egocentric. Their distrustfulness, however, was diffuse and generalized, and in this context was not genuinely paranoid in terms of fixed persecutory beliefs that would be directed toward a specific object. None of the subjects was overtly psychotic, although many had an underlying chronic schizoid pattern of adjustment.

We consider morbid nightmares to be episodic releases of intense emotions in individuals with long-term schizoid adjustments and chronic difficulties in dealing with interpersonal resentments and fears of hostility from others. When the individual cannot adapt to the excessive fear and hostility resulting from conflictual and antagonistic object relations by sufficiently discharging them during the daytime, the fear and hostility may be expressed during sleep in the form of the nightmare. Thus the nightmare would serve as a vehicle not only for discharging such unreleased fears of hostility but also for extinguishing unfinished anger and generally negative emotionality. When this distressing emotionality is finally expressed in the nightmare, it becomes anxiety-provoking and produces more fear because of its punitive consequences.

The dissociative elements that were apparent in our subjects, as suggested by the elevations on the hysteria scale in combination with the psychopathic deviate scale of the MMPI, appear to have an ego-protective function by enhancing the degree to which the nightmares are experienced as ego-alien, as if the nightmares somehow came from "outside" the person. The dissociativeness of the 3-4/4-3 profile is also directed toward avoiding frightening, hostile interactions. From this perspective, the fears and hostility in the nightmares, originating from unresolved anger in everyday life, would be more acceptable if viewed as not "self-owned."

Our findings extend the results of several other investigations, including our preliminary report on nightmare sufferers (18), a subsequent brief report on adult nightmare patients (19), and a study of insomniac patients who also had nightmares (20). The correlation of nightmares and psychopathology is also in agreement with a study of psychotic patients showing that frequent nightmare sufferers demonstrated greater manifest anxiety and lower ego strength (17). Further, a significant positive correlation was found between scores on the Manifest Anxiety Scale and nightmare frequency (16). Our findings in nightmare sufferers are similar to those of Cartwright (21) in individuals with repetitive dreams, many of which were unpleasant. Specifically, she related repetitive dreams to periods of stress associated with strong affect during the day.

The subjects with nightmares were found to have an underlying "schizotypy" (13) (a chronically schizoid pattern of adjustment), but none was overtly psychotic. The high value for the MMPI schizophrenia scale in our sample is in general agreement with the finding by Hartmann and Russ (19), although their subjects scored even higher on this scale. Nevertheless, their sample had a mean age of 22 years, and it is well documented that disturbed younger people often obtain elevated scores on this scale. If, according to their contention, nightmares were a "marker for schizophrenia," one would expect the score for the schizophrenia scale in a considerably older sample of people with nightmares such as ours to be higher and to include distinctly psychotic profiles, but this was not the case. In general, their results were difficult to interpret because they did not present data on group mean profiles, percentage of primed profiles, or code type frequency. In our subjects the MMPI depression scale was not prominent, as it was in their study (19), but our subjects reported a significantly greater frequency of past suicidal ideation than did controls. The lack of elevation of the depression scale may have been due to the limited number of men in our sample because it was prominent only in the men studied by Hartmann and Russ. This is of potential clinical importance because when the depression scale is elevated in subjects showing the 4-8/8-4 code type, the risk of suicide attempt increases.

Clinical Applications

The evaluation of the patient complaining of nightmares begins with taking a thorough history, including age at onset, frequency of episodes, time of occurrence, behavior associated with the episode, length of the episode, degree of recall of mental activity, general daytime behavior and adjustment, and relation of the onset or recurrence of nightmares to stressful major life events. Since many drugs, including alcohol, may induce marked changes in both the frequency and in-

tensity of dreaming (22, 23), a complete drug history is very important.

A thorough history is almost always sufficient in differentiating between nightmares and night terrors. The night terror typically occurs early in the night and is identified by excessive vocalization and motility, a marked increase in autonomic response, impaired arousal following the event, and total amnesia or limited recall of the episode (2–6, 24). The nightmare, in contrast, may occur at any time during the night and is characterized by limited vocalization and motility, minimal autonomic response, unimpaired arousal, prolonged wakefulness after the event, and vivid, detailed recall of the episode.

In the assessment of the adult patient with nightmares, a careful psychiatric evaluation should follow the taking of the history. If there is an indication of a considerable degree of psychopathology, as was often the case with our nightmare sufferers, the nature of the intrapsychic conflicts should be explored. For example, the physician may determine that early life experiences cause greater concerns and preoccupation with death in patients with nightmares (25). Although monosymptomatic psychiatric conditions, such as chronic fears or phobias, may be responsible for nightmares, it is usually a more global psychological disturbance that causes nightmares in adulthood, as demonstrated in the present study.

Since the nightmare sufferer's emotional condition is often complicated rather than monosymptomatic, we would expect behavioral treatment (26–28) to be effective only in selected cases but insight-oriented psychotherapies more likely to be effective in the vast majority of cases. The psychotherapist should be aware of the distrustfulness and alienation often present in the nightmare sufferer and the consequent likelihood of his or her suddenly terminating treatment. Thus a major consideration at the beginning of therapy is to deal with issues of trust and develop a sound therapeutic relationship and then to help the patient achieve a better understanding of his or her emotions, particularly anger. This would enable the patient to express his or her emotions more efficiently, rather than becoming excessively frustrated and, as a consequence, discharging resentments in nightmares.

The psychotherapist should be alert to the schizoid adjustment in the nightmare sufferer's lifestyle, but he or she should understand that the patient's coping mechanisms generally are not psychotic in nature. When overtly psychotic behavior is associated with the presence of nightmares, however, the psychiatrist should proceed with the indicated antipsychotic treatment. He or she should also be alert for the presence of depression, especially in the male nightmare sufferer, since he tends to avoid professional help and thus a suicidal potential may go unnoticed.

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Identification of a Subgroup of Tardive Dyskinesia Patients by Pharmacologic Probes

BY DANIEL C. MOORE, M.D., AND MALCOLM B. BOWERS, JR., M.D.

Some patients with tardive dyskinesia fit the cholinergic-dopaminergic imbalance theory, but some do not. In an attempt to study this heterogeneity further, the authors measured the responses of 10 patients with tardive dyskinesia to intravenous challenge doses of drugs that facilitate or inhibit acetylcholine transmission (physostigmine or benztropine, respectively). They then measured the response of these patients to an open outpatient deanol trial. They found that the responses of half of the patients followed the classic theory, 2 responded paradoxically, and 3 responded inconsistently. They suggest that there is a subgroup of tardive dyskinesia patients who fit the theory but that more research is needed to identify the subgroups who do not.

Over the past 5 years there has been increased attention to prevention and treatment of tardive dyskinesia because the frequency of this disorder seems to be increasing. In particular, the strong relationship between long-term treatment with neuroleptics and the development of the involuntary movements that characterize tardive dyskinesia has threatened therapeutic strides made in the treatment of schizophrenia and other psychotic disorders in the last 20 years.

Recent theories of tardive dyskinesia have focused on the neuropharmacologic interactions of the dopamine and acetylcholine systems in the corpus striatum of the basal ganglia. Chronic administration of neuroleptic medications is thought to cause denervation hypersensitivity of the dopamine receptors, resulting in relative dopaminergic predominance and a cholinergic-dopaminergic imbalance (1, 2). Similarly, it has been observed clinically that anticholinergic agents make tardive dyskinesia worse and that in some cases the disorder has disappeared after the anticholinergic agent has been discontinued (3–5). Klawans and Rubovits (6) showed that tardive dyskinesia could be temporarily improved with the rapid-acting anticholinesterase inhibitor physostigmine, which presumably restores the cholinergic-dopaminergic balance by increasing acetylcholine activity. Results of studies attempting to find cholinergic agents that would be effective on a long-term basis, such as deanol or choline chloride, have been mixed: some patients have improved remarkably, some have become significantly worse, and still others have responded little or not at all (7–9). These findings, plus case reports of paradoxical positive responses to the anticholinergic agent benztropine (10, 11), suggest that only a subgroup of patients with tardive dyskinesia fit the current cholinergic-dopaminergic imbalance theory.

In an attempt to study this apparent heterogeneity further, we first measured responses of patients with tardive dyskinesia to brief i.v. challenge doses of drugs that facilitate or inhibit acetylcholine transmission (physostigmine or benztropine, respectively). We then measured the subsequent response of these patients to an open outpatient deanol trial.

METHOD

Subjects

Our subjects were 10 chronic psychiatric patients, 2 men and 8 women, who were admitted to the Connecticut Mental Health Center research ward after giving informed consent. They received physical, neurologic, psychiatric, and routine laboratory examinations. Criteria for inclusion in the study included 1) visible dyskinetic movements following prolonged exposure to neuroleptics, 2) no known family history of the heredodegenerative diseases that involve dyskinetic movements, 3) no exposure to medications that may predispose to dyskinetic movements (e.g., amphetamines, L-dopa) other than neuroleptics, anticholinergics, or antidepressants, 4) duration of tardive dyskinesia of at least 6 months, and 5) no medical contraindication. All patients were kept on their routine medications, which were unchanged for at least a

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month before the study. No attempt was made to tabulate the many different neuroleptic and anticholinergic medications patients had been given in the past because this information was not available in reliable form.

Drug Schedule

Once admitted, patients were allowed to become accustomed to the ward for 2 days. A videotape record was then made of their movement disorder; this served as a baseline for comparison following administration of deanol. For the next 3 days each patient spent 2 hours a day in the research room in a hospital bed with his or her head elevated. An i.v. line was placed in one arm, and a blood pressure cuff and stethoscope were attached to the other so that they could be manipulated by a nurse behind a screen. The patients could see the video camera, which was placed at the end of the bed. On the first of 3 test days the subjects received an injection of methscopolamine, .50 mg i.m., 30 minutes before the procedure began. After two 1-minute baseline videotapings 10 minutes apart, 2 ml of saline was infused over 5 minutes, followed by five 1-minute videotapings 10 minutes apart over the next hour.

The same overall procedure was followed for the next 2 days, alternating randomly between 1) methscopolamine, .50 mg i.m., 30 minutes before the procedure, and physostigmine, 2.0 mg i.v., infused over 5 minutes, and 2) saline, .5 ml i.m., 30 minutes before the procedure, and benztropine, 2.0 mg i.v., infused over 5 minutes. Patients were pretreated with methscopolamine because it blocks the unpleasant peripheral side effects of physostigmine without affecting the central actions. The effects of intravenous test doses of physostigmine or benztropine lasted less than 2 hours so that there was virtually no likelihood of drug carry-over from day 2 to day 3.

After challenge trials were completed, the patients were discharged and treated with deanol, 900 mg/day, in three divided doses. If this dose did not completely suppress the tardive dyskinesia, the dose was raised by 300 mg/day every 7 days up to a total dose of 1800 mg/day. If side effects occurred, the dose was lowered to a tolerable level and held at that level. Subjects were videotaped while receiving 900 mg, 1200 mg, 1800 mg, or the highest tolerable dose—in one case only 600 mg—for a total of 7 days.

Evaluation of Dyskinesias

Changes in dyskinetic movements were rated by choosing the most significant and disfiguring movement for each subject and counting its frequency over a 1-minute period. The correlation coefficient for interrater reliability between the two raters, one blind (M.B.B.) and one nonblind (D.C.M.), varied from .82 to .98. The average for all 10 patients was .92. Rating changes in amplitude were found to be unreliable and so were omitted.

DATA ANALYSES

We analyzed the results from the i.v. challenges and the deanol trial in a number of ways to find consistent patterns of response to the different drugs administered. We examined the data from the i.v. challenges with saline, physostigmine, and benztropine by averaging the two baseline measures and using a paired t test to compare averaged baseline scores with scores of 10, 20, 30, 40, and 50 minutes after administration of each drug. This approach produced no statistically significant results even though the observed changes seemed to show a theoretically coherent pattern in a subgroup of patients. Consequently, results in individual patients were evaluated by averaging the three scores for the 30–50-minute time period and expressing them as percent change from baseline (see table 1).

Changes with deanol were evaluated by taking the average of the four baseline scores and comparing it with the average of the four deanol scores using paired t test and percent change from baseline. Finally, patients were divided into responders and nonresponders to physostigmine and deanol to see if one could predict response to the other.

RESULTS

Challenge Drugs

The scores of 6 subjects decreased (i.e., they had fewer dyskinetic movements) with physostigmine by 14.7%–55.1% (average, 33.3%), while 4 increased their movements. Given benztropine, 7 patients increased their score by 2.3%–94.7% (average, 41.8%), while 3 had reduced movements. Since the changes occurred in both directions, the group findings did not reach significance with either drug. As can be seen in table 1, certain patients, such as patients 3, 4, 5, and 8, showed clear decreases when given physostigmine, and others (patients 6 and 7) showed dramatic increases. Still others (patients 1 through 5) showed marked increases in movements when given benztropine. Unfortunately, the striking variability of the baseline patterns made changes in either direction difficult to document statistically.

Seven patients improved when given deanol at dosages of 900–1800 mg/day. Generally, the patients given the 900–1200-mg range showed the greatest improvement. However, the improvement was not significant for the group as a whole because patients 6 and 8 had such significant increases in dyskinetic movements on deanol (225.6% and 146.7%). When these 2 patients were removed from the analysis, the deanol improvement became significant at the $p<.001$ level.

Taking the 7 patients whose movements decreased in response to deanol as a group, we examined their response to the experimental drugs by comparing each

TABLE 1
Drug Effect on Tardive Dyskinesia in 10 Patients: Percent Change from Baseline[a]

Patient Number	Saline	Physostigmine	Benztropine	Deanol	Deanol Dose (mg/day)
1	−21.9	−14.7	+49.3	−31.0	900
2	+8.0	−23.3	+43.1	−39.6	1800
3	+4.7	−37.1	+94.7	−78.3	1200
4	+6.8	−33.6	+44.5	−62.9	1200
5	−0.03	−36.2	+41.7	−100.0	1800
6	−48.2	+180.8	+2.3	+225.6	600
7	−17.8	+39.1	−5.4	0	900
				+10.5	1800
8	+25.4	−55.1	−7.8	+146.7	1800
9	+15.4	+21.4	+17.1	−33.7	900
10	−64.7	+11.8	−5.4	−24.7	900

[a]A positive percent change indicates an increase in the number of dyskinetic movements observed.

time period with the baseline to see if there was a significant trend. With benztropine, the dyskinetic movements of these patients were significantly increased ($p<.05$) at 20, 40, and 50 minutes after drug administration. There were no statistically significant changes with physostigmine or saline.

The pattern of the responses of patients 1 through 5 fits the current cholinergic-dopaminergic imbalance theory (see table 1): cholinergic agonists like physostigmine and deanol improved tardive dyskinesia and cholinergic antagonists like benztropine made it worse. Patients 6 and 7 showed a paradoxical pattern, with increased movements in response to physostigmine and deanol and no change in response to benztropine. The final 3 patients did not fit any consistent theoretical pattern.

DISCUSSION

Until recently tardive dyskinesia has been viewed as a single disease with a single etiology. Our findings show that 50% of the patients with tardive dyskinesia whom we tested followed the classic cholinergic-dopaminergic imbalance theory. They formed a major subgroup within the larger group of tardive dyskinesia patients and could be distinguished by pharmacologic challenges with cholinergic and anticholinergic agents. Casey and Denney (12) had similar results in an elegant study of 6 patients. They challenged each patient with L-dopa (a dopamine agonist), droperidol (a dopamine antagonist), physostigmine, and benztropine. They then placed patients on a double-blind deanol and placebo trial and found that 3 patients improved significantly on physostigmine, droperidol, and deanol in accordance with current theory, while 3 other patients became significantly worse on the same three drugs and better on benztropine. In other words, approximately 50% of their patients fit the current pharmacologic theory that dopamine antagonists and cholinergic agonists will decrease tardive dyskinesia, a finding that agrees with our own results, i.e., 5 out of 10 patients improved on physostigmine and deanol and became worse on benztropine.

A survey of the literature on the use of cholinergic agents in tardive dyskinesia reveals a consistent recurrence of this 50% figure, which may represent a subgroup. For instance, eight different investigators, who have administered physostigmine in doses of 1–3 mg i.v. to a total of 53 tardive dyskinesia patients, found that 28 (52.8%) showed decreased movements, 10 (18.9%) showed increased movements, and 15 (28.3%) showed no change (6, 8, 12–17). Similar figures emerge from i.v. trials of benztropine, 2.0 mg, or scopolamine, 1 mg. Of 24 cases studied by four investigators, 12 (50%) followed the theoretical pattern by getting worse, while 5 (20.8%) had decreased symptoms and 7 (29.2%) showed no change (6, 11, 12, 17).

Casey (18) reviewed the therapeutic use of deanol in tardive dyskinesia. Using the rigorous criteria for improvement of 50% change in symptoms, he noted that of 75 cases reported in the literature, 30 patients improved (40%), 44 had a slight or no response (58.7%), and 1 became worse (1.3%). Looking at double-blind studies only, he found that of 29 patients, only 7 (24.1%) significantly improved. He noted no patient characteristics or clinical aspects of tardive dyskinesia that would predict response to deanol.

By way of comparison, a double-blind crossover study with choline chloride, a more likely precursor of acetylcholine (19), found that 5 of 20 patients improved greatly and 4 patients improved moderately. Movements were unchanged in 10 patients and worse in 1. This 45% improvement rate is the best reported to date with longer-acting cholinergic agents.

All four drugs seem to show a consistent subgroup of approximately 40%–50% of patients who respond according to current pharmacologic theory. However, at this stage of research in tardive dyskinesia, we cannot rule out a number of possible sources of experimental variability that might explain the lack of uniformity of response on a technical basis. Many authors have discussed, and Simpson and associates (20) have documented, the day-to-day changes in tardive dyski-

nesia. Combined with the tendency of patients either to suppress or release movements when anxious or observed, this variability makes changes in movements extremely hard to score reliably in both acute and chronic drug trials. Another source of confusion is the failure of most investigators to control for a number of factors that are generally acknowledged as influencing the severity of tardive dyskinesia. These include maintaining patients on anticholinergics during drug trials (6, 9, 12–14), discontinuing psychotropic medication immediately before administering experimental drugs (8, 17, 20, 21), or disregarding the duration of tardive dyskinesia (7, 8, 11, 20).

Further studies will be necessary to control for these sources of variability more rigorously, and the result of this more careful attention to experimental method may be the development of subgroups of patients with tardive dyskinesia who respond more uniformly to specific drugs. These subgroups might be based on age, sex, duration of tardive dyskinesia, total duration of neuroleptic treatment, diagnosis, or some other as yet unknown factor. We have recently shown that a group of patients with tardive dyskinesia was heterogeneous with respect to CSF measures of central dopamine activity (22). Our present study suggests that approximately 50% of tardive dyskinesia patients can be identified as responders to cholinergic treatment consistent with the cholinergic-dopaminergic imbalance theory. Just as intriguing is the etiology of tardive dyskinesia in the 50% who do not respond in the expected fashion to cholinergic agents. Advanced age and diagnosis of organic brain syndrome are important risk factors for senile (spontaneous) dyskinesia as well as for tardive dyskinesia, and it may be that older or demented patients have a more permanent lesion that is less responsive to pharmacologic intervention. Still another subgroup of patients seems to react paradoxically, such as our patients 6 and 7, who had increased movements when given physostigmine and deanol. Their response is similar to that of 3 out of 6 of Casey and Denney's patients, who became worse on physostigmine, droperidol, and deanol (12). They may have changes in different areas of the corpus striatum, resulting in a "hypercholinergic" form responsive to anticholinergics as theorized by Casey and Denney (12) and Gerlach and associates (23).

Studies that treat all tardive dyskinesias as a uniform clinical entity for statistical purposes may obscure significant clinical responses of subgroups of patients who, as shown above, may respond in diametrically opposite ways to the same drug. To understand and treat tardive dyskinesia more successfully we will need to try multiple drugs on the same population of patients and then examine individual responses to identify specific subtypes.

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The Psychiatry Internship and the Development of Professional Identity

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Advocates of the psychiatry internship believe the experience fosters professional maturity. The authors examined the maturing process of 10 psychiatry residents during their internship year. Changes in the interns' professional identity including expectations of self and others are described and seen as a goal of the experience. The authors outline danger signals of poor professional development and suggest interventions to help interns resolve conflicts.

The purpose of an internship for psychiatrists has been debated for years. In 1970 the American Board of Psychiatry and Neurology eliminated the internship requirement; in 1975 the Board reinstituted it in modified form. Controversy ensued as educators struggled to find—and to fund—a meaningful experience (1-3). Many arguments did not distinguish between what the internship offered in the acquisition of medical skills and what it offered in helping the young physician to mature professionally. The acquisition of medical knowledge does not justify the internship year. Medical facts alone can be taught more concisely, and certainly more comfortably, to a psychiatry resident than to an exhausted intern. The requirement may be justified if the internship catalyzes a maturational process that is essential to becoming a psychiatrist. In this paper we examine how the internship fosters that process.

Many observers have suggested that the internship promotes professional maturity, but this notion has not been documented (4). Sociological studies have described the broad attitudinal changes that accrue in different hospital environments and historical periods (5, 6). These reports have not been supplemented by an examination of the psychological changes. Karasu and associates (7) compared psychiatry residents who had completed an internship with those who had not and found that the internship residents were considered more proficient in relating to staff and patients, more capable in their handling of medical problems, and more responsive in emergencies. In a follow-up study (8) Karasu and Stein found that internship residents were rated by supervisors as having better clinical judgment and that these residents carried larger caseloads, had fewer patients drop out of treatment, and prescribed medication more often. They concluded that "the internship does have a maturing influence." Unfortunately, the studies did not define professional maturity. In addition, because these studies were retrospective and uncontrolled, they could not determine whether the internship residents were more mature to begin with—and perhaps chose to take an internship for that reason.

THE STUDY GROUP

We decided to conduct a prospective study to examine what constitutes professional maturity and how the internship year helps it develop. Throughout one year, we observed the professional development of 10 psychiatric residents during their internship year at New York Hospital. Each resident spent approximately 6 months on general medical services, including 2 months in private medicine, 1-2 months on a specialized cancer treatment service, 1 month on neurology, 1 month on pediatrics, and 5 months on psychiatric services, including 2 months in a walk-in clinic and 1 month each on three inpatient units specializing in the treatment of geriatric, alcoholic, and research patients. Our observations took place in a discussion group that we established in order to provide a "home base" for the interns who were rotating singly from service to service.

Once a week the 12 of us gathered to review case vignettes, reactions to patients, interactions with staff, ideas, and feelings—whatever seemed related to the experience of being an intern and becoming a physician. Initially, attendance at the group was excellent, but it tapered in the last two months. We stated from the start that the group would not focus on individual personality styles or intimate problems. The primary objective of the group would be to examine the psychological issues relevant to the internship experience.

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Only after several weeks did we appreciate a second objective of the group: the process of examining the conflicts of internship was helpful in resolving them.

GROUP DATA

The group study gradually revealed the psychological conflicts that the internship evokes. We learned that the training experience presents opposing views of one's professional self-worth. The interns expressed conflicts between feeling knowledgeable or ignorant, certain or uncertain, helpful or helpless, authoritative or deferential, and responsible or subordinate.

These conflicts were particularly acute for the residents during their medical and pediatric rotations, where they were required to make rapid diagnostic decisions, work endless hours and perform invasive procedures such as venipunctures, lumbar punctures, and liver biopsies, and institute complicated, unproven, and sometimes life-threatening treatment protocols for cancer patients. They could not avoid an emotional involvement with frightened patients and their disrupted families. As the residents recounted case vignettes they recognized the complex contributions of family involvement, medical knowledge, and socioeconomic variables that contribute to decisions to initiate or terminate intensive medical treatment. While in pediatrics, the residents were forced to confront their own painful emotions about youthful patients and to recognize the effect these feelings had on their treatment decisions. Simultaneously, the interns were expected to be receptive, inexperienced students, to follow the advice of their attending physicians, and to follow residents' directions about chart notes, daily routines, filling out forms, and getting X rays.

The conflict between feeling omnipotent and impotent is not exclusive to this year of training, but the candid group discussions repeatedly emphasized how the internship uniquely polarizes these extremes. The unshakable presence of physical illness forced the interns actively to confront this conflict. The interns—unlike psychiatry residents—could not retreat into a position of passive neutrality. The option of psychiatrists to defer responsibility and deflect the problem back onto the patient in the privacy of an office is not available to the intern on a medical, surgical, or pediatric service. During a patient's cardiac arrest, not to decide is to decide! The experience thereby not only intensified conflicts between omnipotence and impotence, but through its requirement for action pressed for an adaptive resolution.

The early group discussions exposed only one pole of this conflict: the impotence. The interns complained of feeling overwhelmed, inexperienced, and inadequate. They felt like "imposters" when writing orders or doing procedures. One intern thought the nurses must be joking when they addressed her as "doctor"—after all, the nurses knew far more than she did, and they certainly knew she was not *really* a doctor. Conversations centered on the most ill patients. The interns, an insightful group, realized that because they felt vulnerable and helpless themselves, they sympathetically identified with the most dependent patients. The interns were less quick to realize that—also like these neediest patients—they had developed unrealistic expectations of their own "caretakers" by overidealizing attending physicians and by viewing residents as incredibly knowledgeable. Having a sharp resident provided a sense of security—and a sense of panic when that maven left for the day and the interns were confronted with handling patients on their own.

This idealization of the attending staff and residents had a short life. Although the interns, in response to their own feelings of inadequacy and frustration, continued to believe that they were merely "puppets on a string," they stopped regarding their superiors as so grand. The interns began to express contempt for residents, attending physicians, and hospital administrators who had "let them down" by not fulfilling the great expectations of a top training program. Elder physicians were "incompetent and outdated," younger attending physicians were "ambitious and insensitive," and residents were "competitive and unavailable." A litany of complaints permeated the group; the interns sharply criticized a cancer research protocol, picked apart a treatment plan by a prestigious attending physician, and derided a resident for failing to order further tests for a dying patient. This disappointment reached a crescendo in the group when a patient committed suicide in the psychiatric hospital. The interns greeted the news with angry incredulity. How could it happen? Why couldn't the staff stop him? Why didn't the psychiatrist predict the suicide and take proper precautions?

During this phase the interns regarded their superiors not only as incompetent but malevolent as well. They felt they were handmaidens designated to do the undesirable work so that private practice physicians could live comfortably. One group member suggested that the internship is the result of a necessary financial collusion between hospital administrators and exploitive attending physicians. Other members believed the only rationale for the experience was that it was a type of hazing ritual for the rite of passage into the medical guild. They talked about the need of insecure, older physicians to inflate themselves by speaking about "days of the giants, when ships were made of wood and men were made of steel."

The interns' failure to find any value in the internship experience reached its highest pitch when one of the group members decided to take a leave of absence. The interns' rotation schedules had to be revised. They were irate. They felt maligned, "political

pawns" whose lives were being manipulated for the sake of the department. The prearranged schedule was the one thing they had relied on. Now that, too, was being taken away.

Their general disappointment in their residents and attending physicians and their specific disappointment in having to readjust their schedules catalyzed a dramatic change in the interns' view of themselves. Feelings of being helpless victims and incompetent neophytes were replaced with feelings of being powerful, prestigious, and influential. "We really keep the hospital going." "We're the ones on the inside track." "The attendings come and go but we're where it's at—on top of things." Esoteric medical jargon then infiltrated the group discussions as a symbol of the interns' acquired status. An intern mentioned that she had walked out of the psychiatry conference on her medical service because "all they do is talk," and she felt that was a meaningless exercise when one was treating people who were "really sick." Another intern boasted that he had made the important management decisions regarding a patient while the rest of the staff vacillated. Despite the complexity of the issues involved, the intern refuted any possibility that his judgment might not be absolutely right. The implication was that if the interns could not rely on attending physicians and residents for foolproof answers, at least they could rely on themselves. They bragged to each other about their fast workups and diagnostic acumen. Their complaints of overwork decreased. One intern even complained of having an insufficient work load!

At approximately midpoint in the year the group gradually entered its third phase. Whereas during the first phase the interns saw themselves as incompetent, with power delegated to others, and whereas in the second phase the interns devalued their superiors and assumed the power themselves, the third phase was characterized by their realization that neither they nor others were omnipotent. As one intern stated succinctly, "Medicine isn't magic—not the pills, not the people."

With this realization came a heaviness in the group. The interns' despair over recognizing limitations made difficult problems seem utterly hopeless. "What can doctors really do? Patients never really get well. They just die or stay sick a long time." Group members compared notes about patients whom one intern had admitted, treated, and discharged, only to have another intern readmit the patient for the same medical problems a few weeks later. The despair over therapeutics was compounded by the interns' disappointment about what gratification they could expect from their work. Patients would not worship the doctor who did not cure them, and they might even resent the doctor who inflicted pain. One intern poignantly described her conflict over drawing blood from an adolescent who accused her of torturing him. When their discouragement became most intense, the interns discussed the "worthless" and "meaningless" nature of the internship in particular and of medicine in general. One intern considered quitting, and others discussed returning home "tearful." The group agreed the year was not necessary for those planning careers in psychiatry.

During the last three months the group discussions shifted into a fourth and final phase. The interns' mood improved, and they began to express pride in what they had accomplished—or at least survived. Although still disappointed in the limitations of medicine and of physicians, the interns expressed a more realistic appraisal. They regarded physicians as human beings who get tired, make mistakes, but take reasonably good care of patients. The interns' descriptions of patients gained new dimensions as well; they regarded them as both helpless and helpful, cooperative and uncooperative. Maintaining an all-lovable, infallible image with patients became less of a goal. Members reported vignettes about setting limits with patients even when doing so caused resentment, such as refusing to sign a patient's disability form. The interns were more comfortable in experiencing anger with patients who tried to frustrate or humiliate them or make excessive demands, and they distinguished this anger from the unreasonable anger at patients who did not respond to treatment.

USE OF GROUP PROCESS

The discussion group provided both a way of determining whether an intern was inflexibly stuck in one of the phases we have described (feeling helpless, omnipotent, depressed, or cynical) and an environment to help the interns find more adaptive solutions. Through trial and error, we found that several technical maneuvers were useful in that process.

During the first phase, when the interns revealed an exaggerated sense of their own inadequacy, our tactic was to refuse to accept these notions. We asked the interns what was being accomplished by their feeling so helpless. This question forced the interns to recognize that their sense of incompetency was a distortion. Furthermore, as the interns used their insight and intellect to analyze the question, the process of using these more mature ego functions short-circuited the regressive wish to be taken care of.

Another maneuver during this first phase was to reject the interns' helplessness within the discussion group. The interns expected us to structure the group, relieve the silences, and come up with the final answers. We refused this overidealized role. Instead, we allowed silences and discussed our own mistakes and doubts about patient care. Similarly, during the crisis when one intern took a leave of absence, we refused to accept their notion that we should be able to prevent or correct such situations. We pointed out that such

things are unavoidable. As the interns assumed more responsibility for making the best of this bad situation, they recognized that they had previously viewed themselves in the subordinate position of being interns in order to feel entitled to be taken care of. By the end of this first phase the interns understood that making oneself dependent does not ensure that an all-providing, dependable parent will appear.

During the second phase the group process challenged the interns' fragile grandiosity while at the same time offering collegial support. We helped the interns see that their need to feel admired—to be the giants who rescued mankind from the grip of disease—was an attempt to master feelings of uncertainty, futility, and aloneness. When a member of the group described how brilliantly he or she had handled a case or managed an inhuman work load, the other members would respectfully permit the intern to boast, but in time they would gently question the idealized account. The mutual support of the group members and the sharing of uncertainties became a more adaptive way of facing their limitations as physicians.

The devaluation of attending physicians in the second phase posed a special challenge to the group coleaders. There was a great temptation for us to identify with the attending physicians who were being berated and to counterattack defensively. We tried to resist this temptation. We reminded ourselves that to counterattack would only increase the interns' insecurity and thus increase their need to feel omnipotent. However, we did not want to appear helpless. We found that an effective way to interact with the interns was to agree to disagree by stating our opinions firmly so that the interns knew what we did and did not believe. We also tried to depersonalize the discussions and get the residents to face their own conflicts rather than attack authority. We asked more general questions. Where can a physician reasonably expect to get gratification? From work? From the admiration of patients? From monetary reward? From the improvement of patients? From a final job well done?

The group appeared to have its greatest value during the third phase, when it definitely helped the interns cope with their grief over lost self-idealizations. By this time we had sufficient data to interpret the interns' despair. We pointed out that the demands and limitations of the internship were forcing the group members to come to terms with who they were and who they were not. Although these interpretations were useful, the group process was even more helpful. It provided a supportive place where the interns could withdraw for a moment to contemplate the impact of the internship experience. During this phase we also had to confront our own limitations and resist the temptation to try "to make it come out all right" for the interns. They needed to understand that physicians cannot cure the unavoidable disappointments, losses, and frustrations of life.

During the final phase, as the interns established more realistic professional expectations for themselves and for others, the group reinforced this process by inviting senior faculty members to attend the group and discuss conflicts of their own professional lives. The chairman, vice-chairman, director of training, and the coordinator for the next year all came and informally revealed their professional goals, frustrations, successes, and failures.

DISCUSSION AND CONCLUSIONS

The group data supported the view of internship as a crisis in professional identity. The pattern in which all of the interns passed through similar phases as they attempted to cope with the stress of developing a more consolidated professional identity conforms to the theory of phase resolution of crisis (9, 10). Psychosocial and psychological studies (11–15) have emphasized that establishing a realistic appraisal of oneself is not just the struggle of the young child, but a lifelong process with definable critical periods and developmental tasks. Internship can be seen as just such a critical period—a physician's "professional adolescence" when issues about professional self-worth are heightened. Interns are forced to close the gap between their own ideal self-expectations and exaggerated sense of inadequacy.

The study group revealed the four phases of conflict resolution and some techniques for fostering intern growth: 1) a phase of helplessness when the interns overidentify with the patients and view themselves as totally incompetent and delegate exaggerated powers to others; 2) a phase of aggrandizement when the interns inflate themselves with magical expectations for cure and admiration; 3) a phase of despair when the interns grieve over the lost omnipotence in themselves and others; and 4) a phase of transformation when the interns set more reasonable expectations for themselves and others, accept their limitations, and continue to grow by finding positive aspects in their work and colleagues with which to identify.

Our study group also helped us to recognize when interns are in trouble with their professional maturity. If interns remain stuck in phase 1, they will be paralyzed by uncertainty, will be unwilling to assume responsibility in difficult situations, and will rely too heavily on colleagues, consultants, or the institution of medicine rather than assuming an autonomous position. Similarly, if interns remain stuck in phase 2, they will avoid the complex, chronically ill, or dying patients who challenge their magical view of themselves. They will also resent patients who do not love and admire them and will be demeaning and contemptuous toward colleagues who are less than perfect. Interns' clinical judgment will be impaired by their wish to please patients at all costs, which will lead them to avoid per-

forming painful procedures or to prescribe unnecessary medications. Finally, if interns remain stuck in phase 3, they will be forever disenchanted with medicine and with themselves, constantly frustrated about how little they and the profession have to offer.

The group study was a first step in defining professional maturity. Mature physicians can simultaneously accept their limitations and maintain their therapeutic optimism and concern for patients. Their clinical judgment will be impaired by neither a need to be omnipotent nor a therapeutic nihilism. Mature physicians will be able to tolerate their emotional involvement with chronically ill and dying patients without seeing them as totally helpless victims or biological specimens. They will recognize the complexities of family ties, socioeconomic variables, and medical knowledge, which operate in determining patient care.

We recognize that this study supplies neither a complete definition of professional maturity nor a definitive answer about the questionable value of medical rotations for the psychiatric resident. However, it is a beginning.

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Race Bias in the Diagnosis and Disposition of Violent Adolescents

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The authors compared psychiatric symptoms, violent behaviors, and medical histories of an entire one-year sample of adolescents from the same community who were sent either to the correctional school or the only state hospital adolescent psychiatric unit serving the area. The initial hypothesis that the incarcerated group would be equally disturbed but more violent than the hospitalized group was contradicted. Violence as well as severe psychiatric symptomatology was equally prominent in the two groups. The most powerful variables distinguishing the groups were race, accidents, and injuries (especially head injury), and sex. The medical and social implications of the findings are discussed.

In this paper we report clinical and epidemiological evidence of race bias in the ultimate disposition of aggressive adolescents. We shall report similarities and differences between adolescents from the same community sent either to a correctional institution or to a psychiatric hospital during a single year in the mid 1970s.

In our previous work (1–5) we have reported the existence of severe psychopathology in a delinquent population. We have also reported that incarcerated delinquents have extremely adverse medical histories (6–8) and are characterized by many neuropsychiatric signs and symptoms (9). Having found such a high prevalence of psychotic symptoms, neurological impairment, and serious medical problems in incarcerated youngsters, we wondered what factors might influence a psychiatrically disturbed adolescent's being sent to a correctional facility rather than a treatment facility. We hypothesized that the behavior of incarcerated adolescents would be characterized primarily by aggression toward others, whereas the behavior of psychiatrically hospitalized adolescents would be more withdrawn, self-destructive, or, if violent, would be expressed within the home. We also wondered whether such factors as race and sex affected where a child was sent.

METHOD

Samples

Our two samples consisted of 1) all adolescents from an urban area of Connecticut who in a given year were sent to the only correctional school in the state and 2) all adolescents from the same area who in that same year were admitted to the adolescent unit of the only state psychiatric hospital serving the same area. Since both populations came predominantly from the lowest socioeconomic sectors of the area, private hospitalization was seldom available. The few residential treatment facilities for the area had waiting lists and tended to accept younger children. Except for a general-hospital-based crisis unit, there were no other readily available alternative placements for this population.

The sample of adolescents sent to a correctional school consisted of 63 youngsters, and the sample sent to an adolescent unit consisted of 35. Six children had been to both institutions and were included in the correctional school sample because this was their ultimate placement.

Because of differing acceptance criteria regarding age, the average age of the correctional school sample at the time of our study was 15.56 years and that of the adolescent unit children was 17.01 years, a significant difference ($t=6.151$, $p<.001$). To correct for possible bias due to age differences between the two groups, all data regarding medical histories were analyzed for two time periods, before age 4 years and through age 16 years.

Settings

The two institutions were situated in the same town, approximately 25 miles from the urban area being studied and only minutes from each other by car. Thus they were theoretically equally accessible to the youngsters. Both institutions had open and closed fa-

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cilities and were equipped to care for aggressive children. The adolescent service had 16 beds reserved for boys and 12 for girls. The correctional school had 4 cottages of 30 beds each for boys and 2 cottages of 30 beds each for girls.

Sources of Data

Data were gathered from the records of the adolescent unit of the state hospital, the records of the correctional school, all available previous psychiatric evaluations, and the records of the largest general hospital serving the urban area. This hospital had been used by 83.9% of the correctional school sample and 88.2% of the adolescent unit sample. The advantages and disadvantages of using these hospital records to gather medical information have been discussed in another report (7).

The following factors were explored: 1) violent and nonviolent behaviors influencing institutionalization, 2) quality of psychiatric symptoms mentioned, 3) number of general hospital visits, 4) use of particular services, such as the emergency room, clinics, and wards, 5) the nature of medical problems (e.g., perinatal difficulties, accidents and injuries, head and face injuries, and respiratory infections), 6) age, 7) sex, and 8) race. Although socioeconomic class could not always be established from the records, the great majority of adolescents in both groups came from the lowest socioeconomic sectors of the urban population.

For purposes of assessing the presence or absence of violent behaviors, violence was defined as any mention in the records of arson and/or serious physical violence directed against other people. That is, murder, serious assault in which injury to another person occurred, sexual assault, and threatening with a weapon were classified as violent.

FINDINGS

Behaviors

Our assumption that the psychiatrically hospitalized adolescents would be demonstrably less violent than their incarcerated peers was refuted by the data available. Of the 33 hospitalized adolescents about whom information regarding behaviors was available, 25 (76%) had extremely violent acts documented in their charts. In fact, according to their clinical charts, 19 of these hospitalized adolescents were known to the police or to the courts for a variety of more or less violent antisocial acts; however, for reasons that were not explicit, the courts had not sent them to the correctional institution.

The extreme violence manifested by the psychiatrically hospitalized youngsters is evident in the following examples:

One boy had been arrested for sexual assault, vandalism, indecent exposure, and beating an aged relative. Another had committed armed robbery, had sold drugs, and had a great number of minor charges against him. One boy had knifed another boy and had stated, "I get real violent. I don't stop until I see they're bleeding." One boy had threatened his mother's life, was arrested, released on $2,500 bail, and then sent to the hospital after injuring a policeman. Another boy, charged with burglary, fire-setting, larceny, and reckless endangerment, had allegedly homosexually attacked a small child and had taken his father's gun and tried to kill the father. Another boy had attempted to choke a younger sibling and had subsequently been ejected from a Salvation Army shelter when he assaulted another patron; a gun had been confiscated from him at one hospital, and bullets had been confiscated from him at another.

Physical aggression was characteristic of hospitalized girls as well as hospitalized boys, although it seemed to be somewhat less violent in nature. One girl had been expelled from a residential school because of violent behaviors and had subsequently assaulted a hospital staff member. Another had had a history of assaultiveness since second grade, had punched another patient in the mouth, and had threatened to harm staff. Another girl, who had also been expelled from school because of assaultiveness, had slapped around a child for whom she was babysitting and had physically assaulted the child's mother.

Of the 8 hospitalized adolescents not classified as violent about whom behavioral information existed, 5 had histories either of violence toward property (e.g., smashing furniture or breach of the peace) and/or threatening others with violence. Counter to our prediction, only 3 of the hospitalized adolescents were characterized primarily by self-destructive behaviors. Moreover, violent behaviors clearly were not limited to violence within the home.

Although it is not possible to compare exactly the prevalence and degree of violence in the two samples because the assessments of violence are inexact, a review of correctional school records indicated that 46 (73%) of the incarcerated sample had committed violent acts similar in nature to the acts of the 25 most violent hospitalized adolescents (e.g., assault, armed robbery, and sexual assault). Thus as far as we could determine, violence seemed to be as characteristic of the hospitalized sample as it was of the incarcerated sample. In fact, the majority of hospitalized youngsters had been arrested but not incarcerated for violent acts.

Psychopathology

Psychiatric hospital and correctional school records were maintained for clinical purposes and not for research; therefore, the clinical data were not uniform. All of the hospitalized children had been seen by a psychiatrist; some had also been seen by a psychologist. Almost all of the incarcerated children had received a

TABLE 1
Mean Number of Medical Contacts of 34 Psychiatrically Hospitalized and 63 Incarcerated Adolescents Through Age 16

Type of Hospital Contact	Hospitalized Adolescents	Incarcerated Adolescents	t[a]	Significance
Hospital visits	9.38	12.50	1.14	n.s.
Emergency room visits	4.71	7.62	1.94	p=.056
Clinic visits	4.94	4.22	0.50	n.s.
Admission to hospital ward	.27	.67	1.98	p=.051
Accidents or injuries	1.88	3.93	2.75	p=.008
Head or face injuries	.56	1.29	2.34	p=.022

[a]Two-tailed t test.

TABLE 2
Number and Percent of Psychiatrically Hospitalized and Incarcerated Adolescents with Particular Medical Problems Through Age 16[a]

	Hospitalized Adolescents		Incarcerated Adolescents	
Medical Problem	N	%	N	%
Perinatal problems[b]	9	6.0	19	61.3
Child abuse	6	17.6	16	27.1
Psychiatric symptoms	16	47.1	23	39.0
Psychiatric/social work consultation	9	26.5	19	32.8

[a]All of the comparisons were nonsignificant according to chi-square.
[b]Data on perinatal problems existed for 15 hospitalized and 31 incarcerated adolescents. Data in the other categories are based on 34 hospitalized and 59 incarcerated adolescents.

psychological evaluation, and many had had psychiatric evaluations as well. Both groups had received social work assessments. The majority of children in both groups had received previous psychiatric or psychological assessments in schools, clinics, or other residential settings, and these were also reviewed.

Of the 33 psychiatrically hospitalized adolescents for whom adequate clinical data existed, 13 were described as showing psychotic symptoms and 10 were described as having borderline or equivocal manifestations of psychotic disorders. Of the 60 incarcerated children for whom adequate clinical data existed, 14 were described as demonstrating psychotic symptoms and 17 as showing equivocal evidence of psychotic symptoms.

Because of the variability in the quality of clinical data available, we felt that it made little sense to make statistical comparisons between the prevalence of psychosis or borderline psychosis in the two groups. Suffice it to say that psychotic symptoms were reported to be prevalent in both groups.

Medical Histories

As can be seen in tables 1 and 2 hospitalized and incarcerated adolescents made similar numbers of hospital contacts and were equally likely to have sustained perinatal difficulties. Incarcerated adolescents, however, had significantly more accidents and injuries, specifically head or face injuries. It is noteworthy that the incarcerated children were as likely as the psychiatrically hospitalized children to have had psychiatric problems noted at the general hospital and to have been referred for psychiatric or social work evaluations.

Comparison of Medical Histories of Adolescents from Each Sample Born at the General Hospital

Although the correctional school youngsters had more adverse medical histories through age 16 years than did the adolescent unit youngsters, it was possible that differences in numbers of visits and numbers of injuries reflected length of residence in the area rather than actual numbers of medical events. More correctional school youngsters (N=28) than adolescent unit youngsters (N=9) had been born at the general hospital. Although this difference was not significant, it could conceivably have skewed our findings. We therefore decided to compare the medical histories of adolescents in each sample who had been born at the general hospital in question.

Tables 3 and 4 show the similarities and differences between the medical histories of children in each sample born at the general hospital studied.

Again, the two groups had similar numbers of visits and a similarly high prevalence of perinatal difficulties. However, once more, accidents and injuries, specifically head or face trauma, significantly distinguished the groups: incarcerated children had the greater number of injuries. Of note is the finding that the incarcerated group made significantly more hospital visits and had somewhat more accidents and injuries before age 4 years.

Sex and Race of Entire Samples

As can be seen in table 5 girls were more likely to be hospitalized and boys were more likely to be incarcerated. Furthermore, a significantly greater proportion of black youngsters were sent to the correctional institution than were hospitalized. When the data were analyzed for sex and race, the ratio of black to white girls did not differ significantly within the two institutions. On the other hand, the ratio of black to white boys was significantly greater in the correctional school than in the adolescent unit of the hospital.

TABLE 3
Mean Number of Medical Contacts of 9 Psychiatrically Hospitalized and 28 Incarcerated Adolescents Born at the General Hospital

Type of Hospital Contact	Hospitalized Adolescents	Incarcerated Adolescents	t[a]	Significance
Hospital visits through age 16	13.44	19.07	1.20	n.s.
Hospital visits before age 4	1.67	6.04	2.47	p=.019
Emergency room visits through age 16	7.22	11.44	1.59	n.s.
Clinic visits through age 16	5.78	6.67	.34	n.s.
Admissions to hospital ward through age 16	.44	.93	1.12	n.s.
Accidents and injuries through age 16	1.89	5.46	2.73	p=.010
Accidents and injuries before age 4	.22	.89	1.55	n.s.
Head and face injuries through age 16	.67	2.11	2.06	p=.048
Head and face injuries before age 4	.22	.43	.84	n.s.

[a]Two-tailed t test.

TABLE 4
Number and Percent of Psychiatrically Hospitalized and Incarcerated Adolescents Born at the General Hospital with Particular Medical Problems Through Age 16[a]

	Hospitalized Adolescents[b]		Incarcerated Adolescents[b]	
Medical Problem	N	%	N	%
Perinatal problems	6	75.0	17	60.7
Child abuse	3	33.3	10	35.7
Psychiatric symptoms	5	55.6	15	53.6
Psychiatric/social work consultation	3	33.3	11	39.3

[a]All of the comparisons were nonsignificant according to Fisher's exact test.
[b]Percents are based on the actual number of subjects on whom information was available for each category. Data on perinatal problems existed for 8 psychiatrically hospitalized adolescents. All other data on psychiatrically hospitalized adolescents were based on 9 subjects. Data in all categories for incarcerated adolescents were based on 28 subjects.

Multiple Regression Analyses

Having found the quality and prevalence of violence to be similar in the two groups, we wondered which of the medical or demographic variables that we were able to investigate distinguished the correctional school sample from the adolescent unit sample most clearly. We therefore conducted a multiple regression analysis, using group membership as the dependent variable (i.e., correctional school sample born at the hospital versus adolescent unit sample born at the hospital) and the following independent variables: 1) perinatal problems, 2) number of emergency room visits through age 16, 3) number of ward admissions through age 16, 4) number of accidents and injuries through age 16, 5) number of head and face injuries through age 16, 6) number of respiratory infections through age 16, 7) sex, and 8) race.

The most powerful variable distinguishing the groups was race, which accounted for 18.1% of the variance. Numbers of accidents and injuries through age 16 years accounted for 11% of the variance; the correctional school sample had the greater number. Sex accounted for 6.8% of the variance. Perinatal difficulties accounted for 4% of the variance: the hospitalized sample had more perinatal problems. The above factors together accounted for 39.9% of the entire variance (R=.632).

When we repeated the multiple regression analysis using the variables of accidents and injuries and head and face injuries as discontinuous variables (i.e., accident or injury through age 16, yes or no, and head or face injury through age 16, yes or no), the picture changed. The most powerful factor distinguishing the two groups was the fact of having received a head or face injury before age 17 years. This factor accounted for 30.2% of the variance: a greater proportion of correctional school adolescents had at one time or another sustained a head or face injury requiring hospital care. Race accounted for 16.7% of the variance. Perinatal difficulties accounted for 6.1% of the variance: the adolescent unit group had a greater prevalence of perinatal problems. Sex was the fourth most powerful predictor, accounting for 4.8% of the variance. These four factors accounted for 57.8% of the variance (R=.760).

Factors Distinguishing Incarcerated from Hospitalized Adolescents Before Age 4

We wondered whether a constellation of medical and/or demographic factors existed that distinguished the two groups early in life. We therefore conducted the same kinds of multiple regression analyses using age 4 as the cutoff point. Our dependent variable, again, was group membership (i.e., correctional school sample born at the hospital versus adolescent unit sample born at the hospital), and our independent variables were 1) perinatal problems, 2) number of emergency room visits before age 4 years, 3) number of ward admissions before age 4 years, 4) number of accidents and injuries before age 4 years, 5) number of head and face injuries before age 4 years, 6) number of respiratory illnesses before age 4 years, 7) sex, and 8) race.

We found that, again, race was the most powerful predictor of group membership, accounting for 18.1% of the variance. Sex predicted 8.3% of the variance, and number of emergency room visits accounted for 6.4% of the variance: correctional school children had

TABLE 5
Sex and Race of 35 Psychiatrically Hospitalized and 63 Incarcerated Adolescents

Item	Hospitalized Adolescents N	Hospitalized Adolescents %	Incarcerated Adolescents N	Incarcerated Adolescents %	χ^2	Significance
Sex						
Male	22	62.9	52	82.5		
Female	13	37.1	11	17.5		
Comparison					3.709	.01<p<.05
Race						
Black	10	28.6	42	66.7		
White	25	71.4	21	33.3		
Comparison					11.625	p<.001
Sex by Race						
Black and male	4	18.2	35	67.3		
White and male	18	81.8	17	32.7		
Comparison					13.061	p<.001
Black and female	6	46.2	7	63.6		
White and female	7	53.8	4	36.4		
Comparison					.198	n.s.

more emergency room visits. These three factors accounted for 33.3% of the total variance (R=.577).

When accidents and injuries before age 4 years and head and face injuries before age 4 years were treated as discontinuous variables (i.e., accident or injury before 4 years, yes or no, and head or face injury before 4 years, yes or no), the following findings emerged: race accounted for 18.1% of the variance, sex for 8.8%, and having had a hospital-treated accident or injury before age 4 years an additional 7.2%. Incarcerated adolescents were more likely to have been treated at the hospital for an injury or accident before age 4. These three factors together accounted for 34.1% of the total variance (R=.584). Thus the two groups could be distinguished from each other before their fourth birthdays.

DISCUSSION

Counter to our initial hypothesis, the results of this study suggest that the majority of adolescents hospitalized in the state psychiatric facility were as violent as their incarcerated peers. Whether or not the two samples had equally severe psychiatric symptomatology was difficult to ascertain, but our clinical and epidemiological findings suggest that both samples were essentially psychiatrically similar. Clinical descriptions suggested that a few of the hospitalized youngsters were more flamboyantly and therefore obviously psychotic than the incarcerated group. Consonant with psychiatric findings was the fact that both groups had similarly poor medical histories. Paradoxically, incarcerated adolescents were significantly more likely to have sustained head injury requiring hospital care. It is ironic that head injury, a medical event that one might assume would correlate with subsequent psychiatric treatment, was strongly associated with subsequent incarceration.

Our findings regarding the disposition of violently aggressive adolescent girls represents one of the few examples in our society of the operation of what would seem to be a reverse sex bias. Aggressive behaviors that in adolescent boys are often treated as the deliberate acts of healthy youngsters are more likely to be recognized as psychologically aberrant when performed by girls.

A most striking factor distinguishing the two groups was neither behavior nor psychopathology. It was race. In the lower socioeconomic sectors of the urban area studied, violent, disturbed black adolescents were incarcerated; violent, disturbed white adolescents were hospitalized.

There are several plausible explanations for the fact that aggressive, psychiatrically disturbed black adolescents are less likely than similarly disturbed and aggressive white adolescents to receive psychiatric treatment. The history of psychiatry in the United States bespeaks a tendency for white psychiatrists to overlook or rationalize as normal the signs and symptoms of psychopathology in black individuals (10–12). The exclusion of black patients from white psychiatric facilities was justified in the past by the presumption that black people were essentially different from white people—that they were cheerful and not prone to psychiatric illness and therefore were to be assessed by means of different standards of mental health (13).

These early-twentieth-century rationalizations have persisted. For example, Fischer (14) stated, "What is called sick or deviant behavior for one group may actually be adaptive—or functional—in another." Similarly, White (15) asserted, "What the dominant culture deems deviant or antisocial behavior might indeed be the functioning of a healthy black psyche."

To this day psychiatric professionals are taught to guard against the possibility of diagnosing pathological deviance in a different racial population. It is our experience that such advice and training have often led white professionals to dismiss the signs of psychotic and/or organic disorders in black patients as culturally appropriate. Hallucinations have been dismissed as

excessive religiosity, paranoid ideation and behaviors as healthy adaptation to hostile surroundings, and learning disabilities based on CNS dysfunction as evidence of cultural deprivation. As Comer (16) said so well, "Well-meaning social scientists . . . attempting to explain the circumstances in the black community, have sometimes reinforced the stereotypes instead of demolishing them" (p. 173).

The choice of whether to send a violently disturbed adolescent to detention or to the hospital is often made by police, probation officers, and judges as well as emergency room physicians. Thus the medical and the law enforcement professions share in perpetuating the segregation of white and black violently disturbed adolescents.

Our clinical and epidemiological findings indicate clearly that many seriously psychiatrically disturbed, aggressive black adolescents are being channeled to correctional facilities while their equally aggressive white counterparts are directed toward psychiatric treatment facilities. As a result of this practice, correctional facilities in the United States are being asked to function as the mental hospitals of the lower socioeconomic class black population. Conversely, it is likely (in view of the behaviors documented in the records of hospitalized white adolescents) that some of our state hospitals are required to function as jails for the disturbed, aggressive, lower socioeconomic class white population. The failure of white mental health professionals to recognize, diagnose, and treat psychopathology in their black adolescent clients may explain in great measure the fact that our prisons and reform schools are becoming increasingly populated by members of minority groups.

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BRIEF COMMUNICATIONS

Disulfiram Encephalopathy as a Cause of the Catatonia Syndrome

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The authors describe a case of the catatonia syndrome associated with disulfiram therapy. Although useful in the management of chronic alcoholism, disulfiram is being increasingly associated with a wide spectrum of side effects and untoward medical sequelae, which now include catatonia. The authors note that catatonia is a clinical syndrome associated with multiple medical conditions as well as psychiatric disorders.

Disulfiram became a popular drug in the management of chronic alcoholism after 1948, when Hald and associates (1) noted that disulfiram interfered with the metabolism of ethanol by blocking liver aldehyde dehydrogenase. The buildup of high blood levels of acetaldehyde in an individual who ingests both disulfiram and alcohol leads to the toxic reaction of vasodilation, hypotension, tachycardia, vomiting, headache, and sometimes death. Acute organic brain syndrome is a reported side effect of disulfiram taken without alcohol (2). Liddon and Satran (3) reviewed 52 cases of disulfiram-associated psychotic reactions and noted a wide spectrum of clinical symptoms, including confusion, anxiety states, depression, mania, paranoia, and frank convulsions.

Other investigators have pointed out the possible serious medical sequelae of extended disulfiram use. Plotnick (4) and Yodaiken (5) warned that workers who are exposed to ethylene dibromide (used in fuels and insectides) and are receiving disulfiram are at considerable risk for developing malignant tumors. Rainey (6) proposed that carbon disulfide may be the metabolite of disulfiram that is responsible for the drug's side effects and that alcoholic individuals receiving as little as 125 mg/day of disulfiram may have a four times greater risk of developing arteriosclerotic cardiovascular disease than do control subjects. However, Faiman and associates (7) question Rainey's assumptions and suggest that his conclusions are speculative. Lake and associates (8) noted small but significant increases in plasma norepinephrine and blood pressure in hospitalized alcoholic patients receiving 500 mg/day of disulfiram.

Gottesfeld and associates (9) reported what is to our knowledge the first case of acute catatonia associated with disulfiram therapy; they and others (10, 11) also have reported schizophrenic-like symptoms in other patients taking disulfiram. Reisberg (12) recently reported a second case of disulfiram-associated catatonia.

Catatonia was first described by Kahlbaum in 1874 (13) as a "symptom complex" consisting of negativism, catalepsy, mutism, stereotypies, and verbigeration (stereotyped language). Kahlbaum considered catatonia to be the manifestation of pathological brain structure. Kraepelin (14) included excited and retarded forms of catatonia as subtypes of the mental illness that he named "dementia praecox." Bleuler (15) redefined and expanded on Kraepelin's systematic classification of the psychoses and introduced the term "schizophrenia." Bleuler maintained catatonia as a schizophrenic subtype. Both Kraepelin and Bleuler taught that catatonic signs could be secondary to other conditions and were not exclusive symptoms of dementia praecox or schizophrenia, although Kraepelin

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may have overvalued catatonia as evidence of a functional psychosis (16). Gelenberg (17) pointed out that the catatonic syndrome has come to be associated with schizophrenia in teaching and clinical practice since Kraepelin and that other causes of the syndrome have been understated. He noted that catatonia may also be a manifestation of neurological disorders involving focal lesions in the basal ganglia, limbic system, temporal lobes, and diencephalon. Drugs that have been documented to induce catatonic reactions include aspirin, ACTH (17), the neuroleptics (18, 19), and now disulfiram. Other causes of catatonia are infections and metabolic and toxic conditions, as well as such psychiatric disorders as schizophrenia, manic-depressive illness, and the neuroses (17).

Abrams and Taylor (20) studied prospectively 55 psychiatric patients who exhibited one or more of the following catatonic motor signs: mutism, stereotypy, posturing, catalepsy, automatic obedience, negativism, echolalia/echopraxia, and stupor; only 4 patients (7%) satisfied criteria for schizophrenia, 39 (71%) had an affective illness, 9 (16%) had epilepsy, encephalitis, or drug-induced or toxic psychoses, and 3 patients (5%) had a reactive psychosis. Interestingly, two-thirds of the patients showed marked improvement or full remission after short-term treatment. Other studies have demonstrated that 16%–19% (21, 22) of 250 catatonic patients qualified for the diagnosis of a primary affective disorder, and 13.5%–28% (23–25) of manic patients exhibited clinical signs of catatonia. The catatonic signs were not predictive of outcome in treated and untreated patients. In this paper, we will describe a third patient who manifested catatonic symptoms as part of a disulfiram-induced encephalopathy.

CASE REPORT

Mr. A, a 25-year-old engineer, had a 9-year history of alcohol abuse. At the insistence of his work supervisor and his wife, he entered an inpatient alcoholic treatment unit several years ago. He was discharged 1 month later and maintained on 250 mg/day of disulfiram, which had been started about 2 weeks after he entered the treatment unit. Mr. A refrained from alcohol use and within 1 month after his discharge was functioning well at work and home. About 10 days later a marked change in Mr. A's behavior occurred. He became quiet and withdrawn and began to complain of diminishing memory, headaches, and dizziness. Five days later (the day before his admission to a local community hospital) Ms. A noticed that her husband was staring for long periods, apparently awake but not responsive; he was intermittently agitated. Ms. A encouraged Mr. A to stop taking disulfiram and arranged for his immediate admission.

On the day that Mr. A was admitted to the hospital, Ms. A stated that in spite of the success of her husband's alcoholic treatment program, she thought he had been "worried a lot." She also reported that Mr. A's younger sister was a chronic schizophrenic. No other family members were known to have mental illness.

During his stay in the hospital Mr. A was intermittently withdrawn and excited. Results of his physical examination were unremarkable except for hypertension (blood pressure=200/110). Laboratory results were entirely within normal limits except for a serum sodium of 133 mEq/liter, hemoglobin of 13.8 g/100 ml, and ECGs showing nonspecific T-wave changes. Serum alcohol levels were negative.

On admission, Mr. A was given 100 mg i.m. of chlordiazepoxide immediately and 50 mg b.i.d. orally, in addition to thiamine, 100 mg b.i.d. orally. There was no immediate change in his behavior. He was negativistic and occasionally self-mutilating and sat immobile staring at a television set for hours. After 5 days of hospitalization and no clarification of diagnosis or evidence of improvement, Mr. A was transferred to our medical center for further evaluation.

At the time of the transfer, Mr. A was perseverating, disoriented to time and place, restless, and showed a profound memory loss, both recent and remote. His general physical examination was normal except for injected conjunctivae. A neurological examination was unremarkable except for the marked mental status changes already noted. A traumatic lumbar puncture was performed: the opening pressure was 120 mm water; there were 6 lymphocytes and 5000 red cells per mm^3; and the spinal fluid contained 53 mg/100 ml of protein and 42 mg/100 ml of glucose (serum glucose, 79 mg/100 ml). Assays for neurotropic viral antibodies had negative results.

During the next 5 days, Mr. A remained intermittently agitated and withdrawn. He pulled out his intravenous tube, exhibited posturing of his arms, and was negativistic, refusing to eat or have vital signs taken. There were no focal neurological findings. On the 4th day after his transfer Mr. A had an EEG that showed "diffuse disorganization and disruption of activity . . . consistent with a diffuse gray and white matter encephalopathic process." On the 5th day a neuroleptic was administered to control his agitation (75 mg i.m. of chlorpromazine and 25 mg orally q.i.d.)

A psychiatric evaluation on the 6th day after his transfer revealed that Mr. A was awake, staring, and with his arm raised in a fixed position. He was not responsive to questioning, his affect was flat, and he resisted passive movements of his extremities and neck. His orientation, thought process, judgment, and insight could not be further evaluated. The psychiatrists recommended that chlorpromazine be discontinued and haloperidol 2.5 mg/hour i.m. as needed be started; Mr. A received approximately 10 mg/day of haloperidol for the next 2 weeks. Mr. A continued to exhibit combativeness, negativism, posturing, withdrawal, mutism, and staring. No focal neurological signs appeared. A second EEG on the 7th day on the neurological ward showed a pattern "entirely monotone without well formed dominant patterns." A second lumbar puncture performed on his 8th day was unremarkable.

After 12 days Mr. A began to improve. He was less agitated and took oral fluids more easily. On his 13th day the third EEG remained abnormal; there was intermittent slowing over both hemispheres with asymetrical activity and the suggestion of periodic complexes, particularly over the right hemisphere, consistent with improvement. Two days later Mr. A was able to talk and respond appropriately to simple questions and commands. Nonetheless, he continued to sit

immobile most of the time and had an expressionless face. A CT scan 1 day later was normal, and haloperidol treatment was discontinued 2 days afterward because of Mr. A's improvement.

By his 21st day on the neurological ward Mr. A was fully oriented. He continued to improve and was discharged 5 days later. Extensive psychometric testing before discharge suggested a mild organic brain syndrome. When he returned to the clinic 10 days after discharge, no detectable deficits were observed. Mr. A returned to the alcoholic treatment program but no longer took disulfiram.

In summary, after Mr. A began disulfiram treatment, he experienced no detectable side effects from the medication for 2 months. When he began experiencing some dysphoria followed rapidly by changes in sensorium and behavior, disulfiram treatment was stopped and he became intermittently withdrawn and excited; 10 days after stopping disulfiram, Mr. A. exhibited signs of catatonia that persisted for 1 week and then resolved over the next 2 weeks. Follow-up over 2 years has demonstrated a consistently normal mental status exam, although he has had several episodic drinking binges.

COMMENT

We have described a patient known to be taking disulfiram and successfully abstaining from alcohol who developed a catatonic syndrome manifested by mutism, negativism, stereotypy, staring, and combativeness. The syndrome quickly reversed to normal behavior after disulfiram was discontinued. Given the patient's positive family history of mental illness, it is possible that the patient is genetically vulnerable to a schizophreniform illness, which may have been exacerbated by disulfiram (11). The increased susceptibility of schizophrenic individuals to the untoward side effects of disulfiram may stem from disulfiram's property as a known inhibitor of dopamine β-hydroxylase (26). Ewing and associates (27) hypothesized that disulfiram might be able to provoke a psychosis by means of blocking the production of norepinephrine from dopamine.

Disulfiram is an important and useful modality in the management of chronic alcoholism, a disease with considerable morbidity and mortality. However, clinicians should now add catatonia to the list of toxic conditions associated with disulfiram and carefully monitor disulfiram-treated alcoholic individuals for behavioral changes, particularly during the early treatment period and in patients who have a positive family history of psychiatric illness. Catatonia remains a clinical syndrome that is associated with multiple medical conditions as well as psychiatric disorders.

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Dialysis of Schizophrenic Patients: A Double-Blind Study

BY JOSE A. DIAZ-BUXO, MD., JOHN A. CAUDLE, M.D., JOE T. CHANDLER, M.D., CHARLES D. FARMER, M.D., AND WILLIAM D. HOLBROOK, M.D.

The authors designed a double-blind hemodialysis protocol consisting of hemodialysis or sham dialysis twice weekly for 10 weeks, respectively. Four patients were carefully selected and evaluated clinically and on the Clinical Global Impression Scale. Three did not show any signs of improvement, and one patient demonstrated questionable improvement but decompensated after 12 additional weeks of hemodialysis. This preliminary study does not confirm the hypothesis that hemodialysis is effective in controlling psychotic symptoms. The authors consider other possibly beneficial treatments for schizophrenia.

Schizophrenia is characterized by progressive deterioration of affect, mentation, and conduct and has considerable negative socioeconomic impact. The report by Wagemaker and Cade (1) claiming a remarkable cure in 17 of 24 patients has aroused tremendous interest in the potential benefits of dialysis in the treatment of this disease. Despite universal enthusiasm, skeptics have pointed out the possible flaws of these studies (2): 1) there could be beneficial effects from the distress of undergoing dialysis in such impressive and strange surroundings, with venipunctures, extracorporeal circulation, and sophisticated monitoring systems; 2) the placebo effect or withdrawal of current drug therapy could cause improvement; and 3) the additional attention paid to these patients and the ancillary services given (occupational and recreational therapy) could be responsible for the improvement (3).

In an attempt to further characterize the potential benefits of dialysis in the treatment of schizophrenia, we designed and completed a preliminary double-blind study at Charlotte Memorial Hospital and Medical Center.

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METHOD

We selected four patients who had been diagnosed as having chronic schizophrenia by Bleuler's primary symptoms of looseness of association, inappropriateness of affect, and the presence of autistic logic. We did not feel that the thought disorder was secondary to their disorder of affect. Delusions and hallucinations, although present in most, were considered secondary symptoms. All patients had suffered a chronic thought disorder and disorder of affect and had been unresponsive to antipsychotic medications for at least 1 year. At any time during the study, if the patient was doing well antipsychotic medications could be reduced or stopped; however, if the patient worsened the medication could be increased and if necessary the patient could be hospitalized, with every effort made to continue the scheduled dialysis.

Before initiation of dialysis, all candidates were interviewed by one or both principal investigators (J.A.D.-B. and J.A.C.) and the nature, risks, and potential benefits of the procedure were discussed. If the investigators felt the candidate was unable to make a decision in his best interest, written consent was obtained from his guardian or first of kin. A pertinent history was taken and a physical examination was performed on each candidate and routine laboratory evaluations including hemoglobin, white blood cell count, hematocrit, serum sodium, potassium chloride, carbon dioxide, urea nitrogen, creatinine, alkaline phosphatase, CPK, SGOT, calcium, and phosphorus determinations were made before dialysis and monthly thereafter. An internal arteriovenous fistula was created in the nondominant upper extremity 4–6 weeks before initiation of therapy. All patients were evaluated on the Clinical Global Impression scale (CGI) and by extensive psychiatric interviews and reevaluated every 1–2 weeks during the study if possible.

Dialysis was provided with the Travenol RSP delivery system and a parallel flow, 85 M^2, 11 micra Cuprophane membrane dialyzer. Blood flows were maintained at 200 cc/minute and dialysate flows at 500 cc/minute. Each dialysis session lasted 4 hours.

During the control period (sham dialysis), the same equipment was used for the same length of time but only blood and not the dialysate was circulated, creating a situation of only extracorporeal circulation through the dialyzer. In order to keep the study blind to both patients and investigators, the dialysate lines were covered with dark material. Only the dialysis nurse was aware whether the patient was on real or sham dialysis.

Cards were drawn by the dialysis nurse for selection of treatment versus sham dialysis. The study protocol consisted of two phases: phase A, during which the patient received hemodialysis treatments, and phase B, during which the patient received sham dialysis. Each phase lasted 10 weeks, during which time hemodialysis or sham dialysis was administered twice weekly. After the subject had completed both phases of 20 weeks of study, the code was broken; if hemodialysis was considered beneficial, the patient was assigned to a maintenance phase (phase C) during which hemodialysis was administered for 4 hours weekly or as dictated by the patient's clinical status at the time.

The results were assessed after 20 weeks of study according to the data collected during the weekly CGIS determinations and individual psychiatric interviews.

CASE REPORTS

Case 1. Mr. A, a 45-year-old man, was admitted to the program while taking chlorpromazine, 100 mg q.i.d., amitriptyline, 50 mg q.i.d., and hydroxyline pamoate, 50 mg q.i.d. He had been hostile, paranoid, depressed, and exhibited suicidal thoughts. He had visual hallucinations of a headless man. The initial diagnosis was paranoid schizophrenia. Mr. A gradually became very depressed and required 8 ECT treatments every 3 months. He had received a total of over 150 ECT treatments in the 5-year period before he joined this study.

During the first 20 dialysis treatments Mr. A stopped hearing voices and was not depressed, and all his medications were stopped. During the second 20 dialysis treatments he relapsed and became very depressed and suicidal in spite of renewed medications. Eight ECT treatments between the 27th and 35th dialysis treatments were given because of suicide attempts. Once the code was broken, we noted that Mr. A had done much better during sham dialysis and had relapsed during real dialysis.

Case 2. Ms. B, a 31-year-old woman, had an 18-month history of recurrent episodes of psychosis characterized by auditory hallucinations, delusions, autistic thinking, inappropriate affect, religiosity, and grandiosity. At times she would become quite withdrawn, depressed, and noncommunicative. No response to ECT had been observed. Ms. B was selected for dialysis therapy and after 4 treatments her psychotic symptoms worsened and she had to be rehospitalized. At that time she was placed on thiothixene, 80 mg/day. Dialysis was continued and the thiothixene was gradually reduced to 10 mg t.i.d. during the second 20 dialysis treatments. She was not evaluated on the Clinical Global Impression Scale, but the psychiatric examiner (J.A.C.) believed that Ms. B did better during the second series of 20 dialysis treatments. The code revealed that Ms. B had the real dialysis during the first 20 treatments.

Case 3. Mr. C, a 36-year-old man with an 11-year history of schizophrenia, had been severely disturbed for 3 years, with aggressive outbursts, a very severe sleep disturbance, auditory hallucinations, grandiose ideas, and an inability to relate to others. Antipsychotic medication had been ineffective, and he had been unable to work for the 8 months before he entered the dialysis study. At the time he started dialysis he was taking fluphenazine, 5 mg h.s.

After the code was broken, we found that Mr. C had real dialysis during the first 20 treatments. At that time, he seemed to improve, and we reduced his fluphenazine to 2.5 mg h.s. However, during the sham dialysis he showed marked deterioration, and we increased fluphenazine to 5 mg h.s. Since Mr. C seemed to have done better on real than sham dialysis, he was restarted on real dialysis once a week. He seemed to slowly improve on real dialysis and became less hostile and delusional, although he continued to hear voices. He was able to relate and to control his psychotic symptoms. He remained on fluphenazine, 5 mg h.s. After 6 hemodialytic treatments Mr. C returned to work, although he continued to have psychotic symptoms that he could control. Within 48 hours Mr. C relapsed into a markedly psychotic state; he was hostile and belligerent, was out of touch with reality, heard voices once more, and was unable to function. He was restarted on fluphenazine but did not improve and had to stop working. After 12 dialysis treatments (1 per week), Mr. C was dropped from the study at his own request. We believe that he suffers from grandiose parancid schizophrenia; during the time that he seemed to get better he was not working and the usual life stresses that he had lived with were diminished. During the rest of the treatment, he showed signs of psychosis, and during phase C he relapsed into a severely psychotic state.

Case 4. Mr. D, a 35-year-old man, had an 18-year history of mental illness manifested by inappropriate affect, loose associations, and paranoid thinking that was primarily focused on his father. He had flat affect and significant anxiety and was not able to go out among people. We believed he was suffering from chronic undifferentiated schizophrenia. When he entered the treatment program, he was receiving thioridazine, 150 mg h.s. After 24 dialysis treatments the psychiatric examiner (J.A.C.) and the examiner in charge of the dialysis unit (J.A.D.-B.) communicated and the code was broken. Mr. D had been on real dialysis for the first 20 treatments and had not improved.

DISCUSSION

Although hemodialysis was first reported as a treatment for schizophrenia in 1960 by Feer and associates (4), the studies were uncontrolled and inconclusive and little attention was generated. The work of Wagemaker and Cade (1), revealing dramatic improvement in a large series of patients, has engendered new enthusiasm, but their study was also uncontrolled. The

possible identification of an abnormal polypeptide in schizophrenic patients that is recovered in high concentrations in the dialysate of these patients (5) may be of greater significance than the improvement noted by Wagemaker and Cade; however, these findings have not been duplicated. The mechanism by which hemodialysis removed this potential toxin is difficult to explain with our available understanding of membrane dynamics and renal physiology. Port and associates (6) and Kolff (2) have analyzed these problems in the light of our present knowledge and calculated the hypothetical clearance of endorphins by different modalities of therapy. It seems likely that if indeed abnormal endorphin accumulation is responsible for the symptoms of schizophrenia, other modalities of therapy such as diafiltration or peritoneal dialysis would be more effective than hemodialysis.

Our study, which is preliminary and very limited in its scope, was not intended to deter others from looking at this potentially important therapeutic and diagnostic tool. However, our results must be considered definitely negative, which emphasizes the importance of controlled and double-blind protocols if scientific conclusions are to be derived from such a costly and potentially hazardous study. We must also diagnose the type of psychosis of the individuals treated and report our findings as precisely as possible. If dialysis indeed ameliorates or cures psychotic symptoms, further studies should be funded that may lead to a better understanding of and therapy for schizophrenia.

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Cinderella Syndrome: Children Who Simulate Neglect

BY JEAN GOODWIN, M.D., M.P.H., CATHERINE G. CAUTHORNE, PH.D., AND RICHARD T. RADA, M.D.

The authors describe three adopted girls, aged 9 and 10, who falsely alleged that their adoptive mothers dressed them in rags, made them do all the chores, and favored their stepsiblings. Underlying these false accusations of abuse was a history that included 1) actual abuse of the child in a previous placement, 2) early loss of a mothering figure, and 3) emotional abuse in the adoptive home. Professionals involved in child protection need to recognize this syndrome because intensive family therapy and temporary placement of the child outside the home are required in all cases. The child's false accusation of abuse is a cry for help and should not be dismissed as a manipulative fabrication.

When professionals responsible for child protection discover that a child has exaggerated, fabricated, or simulated abuse, they tend to respond by simply closing the case. However, we have previously shown that false accusations of sexual abuse are often a child's way of calling for help in a family situation that has become desperate for other reasons (1). This report describes a similar pattern in false accusations of child neglect. In the three cases we report, the child's false accusation of neglect called attention to underlying family dysfunction that did require the intervention of a child protection agency.

In these three cases of simulated neglect, adopted girls had complaints remarkably like those of the fairy-tale heroine Cinderella. Each falsely alleged that her stepmother dressed her in rags, made her do all the chores, and showed preference for her stepsiblings. All three girls had been adopted by relatives after suffering the traumatic death of an early mothering figure.

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The children's Cinderella-like accusations functioned at several levels as a cry for help: 1) they were being emotionally abused and probably would have been physically abused had their false accusations not led to the involvement of a child protection agency and their temporary removal from the home; 2) they had never resolved experiences of abuse and abandonment that occurred before adoption; and 3) all three stepmothers had been abused as children and needed to integrate those past experiences before they could fully accept their stepdaughters. In addition, the Cinderella tale mapped inner conflicts that these children needed to explore in treatment. All of the families were hostile to psychotherapy. It was only under pressure of the legally mandated investigation triggered by the child's false accusation of neglect that these families allowed the troubled child to receive needed treatment.

CASE REPORTS

Case 1. Adele, a 9-year-old girl, was referred to a child protection agency when a policeman found her wandering outside school clad only in an undershirt and panties. She explained that because she had been unable to finish her chores at home, her adoptive mother had locked her out of the house. She said her adoptive parents often took her stepsiblings on outings, leaving her at home to do all the chores. A family interview established that Adele had taken off her own clothes and hidden them.

Adele's biological mother died when Adele was 1 year old. Adele was then placed with another relative, an alcoholic woman who neglected and physically and sexually abused her. This woman died when Adele was 6; she was then adopted by her current parents. Her adoptive father was also her uncle.

Adele's adoptive father, who held two jobs, tended to be distant and aloof. His wife, herself sexually abused and brutally beaten as a child, was an anxious woman with many compulsive rituals who bitterly resented Adele's uncontrollable behavior. The stepsiblings became angry with Adele for upsetting their mother. Adele said she wanted to be a member of the family and to get all her chores done.

The adoptive parents decided to voluntarily place Adele in foster care while she received therapy. They refused family therapy, but the mother agreed to participate in joint sessions with Adele's therapist. After 4 months Adele was reintegrated into the adoptive family.

Case 2. Betty, a 9-year-old girl, was referred by the school because she came to class in shabby clothes and without

shoes. Betty said her adoptive mother had thrown away her shoes as punishment for not cleaning under the bed. A family interview established that Betty had changed into shabby clothes on her way to school.

Betty had been adopted into this family at age 6. Her adoptive mother was her biological mother. Because Betty's mother was an unmarried schoolgirl when Betty was born, Betty was raised from birth by a friend of her mother, who was killed in an auto accident when Betty was 6. This foster mother had been overprotective and indulgent, keeping Betty on a bottle until age 4. After the foster mother's death, the girl's biological mother forcibly removed her from the foster home, not telling the child until a year later that she was her biological mother. Betty had many questions about her biological father, but her mother refused to speak of him.

The mother's present husband was a volatile man who refused to become involved with Betty. The mother felt frustrated about disciplining Betty, saying that the child responded to reprimands by tearing up clothes and toys. Betty's adoptive mother had been the victim of sadistic abuse as a child. The stepsiblings resented Betty and physically abused her by locking her in the doghouse and feeding her dog food. Betty had frequent stomachaches, had one episode of hysterical paralysis, and daydreamed that a race car driver would come and rescue her. After a year of play therapy Betty was able to be in the home only on weekends. Her adoptive parents still refused to become involved in her treatment and finally agreed to place Betty permanently with other relatives.

Case 3. Carol, a 10-year-old girl, was referred to a child protective agency by a passerby. Carol had run up to this man and told him that her adoptive mother had kicked her and was keeping her home from school to do all the household chores. Carol used terms such as "child sexual abuse" in describing her problems. A family interview revealed that the bruise Carol offered as evidence of being kicked had been sustained in a baseball accident and that she had refused to go to school because she had not been given the part of the princess in a school play. At age 9 Carol had claimed that her adoptive mother was trying to drown her in the bathtub. That complaint had been dismissed as fiction, and therapy was not offered.

Before her adoption, Carol had been an abused child raised in a psychotic family situation. Carol's psychotic mother had abused her by tying her hands and forcing her head into the toilet. Carol first lived with her adoptive parents at ages 5 and 6 while her mother, a relative of the adoptive father, was psychiatrically hospitalized. At age 7 Carol returned to live with her mother, who had remarried. The new stepfather began a sexual relationship with Carol. Within a year Carol's mother committed suicide.

When Carol was 9 her current parents adopted her; their only biological child, a blind and severely retarded son, had been institutionalized shortly before. Soon after her adoption, Carol had one episode of hysterical blindness.

The adoptive father held two jobs. He was ashamed of the mental illness of his relative, Carol's natural mother, and refused to allow Carol to speak of her. The adoptive mother had been sadistically beaten as a child by her mother and had been sexually abused by her father, but never complained about these incidents because she feared that she would not be believed. She felt defeated by Carol, something she denied having felt with her severely handicapped son, and she spanked Carol hard enough at times to leave bruises. Carol was not certain that her natural mother was dead and daydreamed about returning to her.

Carol was placed in a foster home. The foster mother complained that Carol refused to go to school or to do chores and that she made false accusations against her foster siblings. After 3 months of family therapy Carol was able to be with her adoptive parents on weekends.

DISCUSSION

These three cases were the only documented instances of simulated neglect seen over a 3-year period in a child protection agency that screens 150 complaints of neglect each month. Since the children were treated by different therapists, their remarkable similarities were not recognized until a systematic review of the agency's cases.

Child protection workers are understandably reluctant to make the judgment that a child is lying about parental abuse. One case report (2) described a 3-year-old whose allegation that his mother beat and burned him was dismissed as a false accusation planted by his father, who was estranged from the mother. Four months later this child was beaten to death by his mother. The cases reported here make the additional point that even if the worker is correct in judging a child's accusation to be a lie, other kinds of abuse may still be occurring in the home.

These three cases are remarkably similar and appear to represent a specific syndrome. All three children were 9- or 10-year-old girls who had entered their adoptive homes at about age 6. Each had experienced the traumatic death of an earlier mothering figure before adoption. Two of the girls had been sexually abused in their earlier homes; two had developed hysterical symptoms after adoption (one paralysis and the other blindness). The adoptive fathers were emotionally distant and unavailable to the children. The adoptive mothers had been severely abused as children and were appalled at being regarded as "bad" mothers, in part because that was how they viewed their own mothers. There was intense rivalry with stepsiblings in all cases. Betty was physically abused by her stepsiblings, and Carol developed the symptom of blindness, which mimicked the handicap of her stepbrother (3).

The false accusations made by these girls closely resemble the complaints of the fairy-tale heroine Cinderella. There is one report (4) of a 5-year-old girl who briefly took on the role of Cinderella, complaining that her mother made her do the hardest chores, made her sleep in the ashes, and favored her younger sister. The three girls reported here had similar complaints, but they were unable to deal with their grievances in fantasy or in play; instead, they presented them as realities.

In the original fairy tale, Cinderella's grief for her dead mother is a central issue. Early Italian versions of the tale described an incestuous relationship between Cinderella and her father. The normal rivalry of the

oedipal-age girl with her mother is intensified by actual incest with the father, and, should the mother die at the height of this intense competitive conflict, the child may experience the unconscious fear that she caused the mother's death (5). The girl must blot out all negative feelings about the mother in order to avoid intolerable guilt about her mother's death. In the Cinderella story, these negative feelings were displaced and projected onto the stepmother, and the dead mother was idealized.

Sibling rivalry is particularly intense for the Cinderella child, partly because she feels that her parents prefer her siblings to someone guilty of incest and murder and partly because she may provoke victimization by her siblings to punish herself and assuage her guilt. The therapeutic solution prescribed in the fairy tale was for Cinderella to complete the grief work for her mother—in the story she watered a tree on her mother's grave with her tears daily—and to accept the tasks requested both by the "bad" stepmother and the "good" fairy godmother. This acceptance of chores is a nucleus for the identification with the mother that must supplant rivalry.

Treatment of these cases is complex. Strategies include family therapy, individual therapy for the adoptive parents, and play therapy for the children. Family therapy defines the situation as a family problem and resists the family's effort to find a scapegoat. In our cases, individual play therapy focused on helping the child to mourn the idealized dead mother and to give up fantasies of reunion with her. It was critical for the therapist to visit the child in her home—as did the prince in the fairy tale. These children tended to shamefully conceal from the therapist the rejecting and lacerating family battles that constituted the actual abuse; the false accusation was a face-saving version of the actual situation revealed in the home visit.

The therapist had to monitor the family situation carefully throughout treatment because premature therapeutic interventions tended to trigger new cycles of simulated abuse. When the child felt rejected or blamed because of a therapeutic confrontation, she would provoke or invent a situation in which she felt unfairly abused by the family. This process assuaged the child's guilt and renewed her hopes that she might be rescued by a perfect, fantasized family, perhaps with the therapist. However, this acting out also provoked actual rejection and abuse by the outraged adoptive family. Ultimately, the "wicked stepmother" had to be brought into the child's treatment in order to break this cycle.

Therapy with the adoptive parents focused on their unresolved feelings about the child's lost parent and about their own parents. Two of the adoptive fathers—Carol's and Adele's—felt intense rage and shame about their psychotic female relatives who had abused these girls and thus would not allow the girls to speak of these mother-figures. Similarly, the adoptive mothers needed help in resolving their anger and guilt about their own abusive mothers and childhood experiences of sexual abuse. The adoptive mothers' guilt about childhood incest fostered their identification with the girls' guilt-ridden attempts to degrade themselves as well as their mothers. They seemed unconsciously gratified that the adoptive daughters were voicing complaints that they themselves had never dared to make; at the same time, the false accusations freed these mothers from the danger of surpassing their own mothers by being "good," nonabusing mothers (6). As the adoptive mothers' fear and rage lessened, they were better able to consistently enforce the performance of chores and to allow the girls to identify with them in more positive ways.

Although they are rare, cases involving simulated child abuse should be actively evaluated by professionals. In each of these three cases of simulated abuse, actual physical abuse or neglect was subsequently uncovered. It is possible that more severe abuse would have occurred had the child's simulated abuse not brought her family to professional attention.

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Sex and the Boards

BY MARC H. JACOBS, M.D., AND LOMA K. FLOWERS, M.D.

In 1978 a questionnaire on the functioning of the American Board of Psychiatry and Neurology was mailed to a sample of APA members. The authors present the significant differences in responses between men and women psychiatrists. A higher percentage of women than men were among those who had experienced stress from the time and effort involved in taking the Board examinations and from fear of the oral examination; 45% of the women respondents who had received certification had done so since 1975, versus 23% for the men. The authors discuss possible explanations for the sex differences, including role strain, and suggest greater flexibility in where, when, and how Board examinations can be taken.

In 1976 the membership of APA approved by a wide margin two referenda concerning the American Board of Psychiatry and Neurology (ABPN). Our paper results from the referendum that called for the appointment of a task force, one of whose charges was to "actively seek and summarize the view of the APA membership with regard to ABPN functioning." The questionnaire designed for this task generated an enormous number of data, and this paper explores those data which relate to the differences in views between men and women psychiatrists.

METHOD

The questionnaire (available on request from the authors) was divided into six sections: background information, factors inhibiting certification, certification policies, recommendations regarding certifying exams, personal experiences with the ABPN, and additional comments. The 66-item questionnaire was sent to a random sample of 4,800 APA members (20%). The return rate was 66%. Not every question was answered by all respondents, so there were variations in the sample size for individual questions. All questions were cross-tabulated with the question "What is your sex?" and the data were examined by chi-square analyses to determine the significance of differences between responses of men and women psychiatrists. Differences were considered significant at the .05 level.

RESULTS

Background Information

Of the respondents, 88% (N=2,563) were men and 12% (N=351) were women. This is consistent with the current proportions of men and women psychiatrists in this country (personal communication from C. Robinowitz, C. Nadelson, M.T. Notman, 1979).

The training patterns reported by the two groups show that since 1960 the number of women completing their training has been steadily increasing: over half of the women respondents had completed their training since 1966, while the men were significantly more likely to have been trained before 1966 (see table 1). Twenty-five percent of the women were foreign medical graduates, which is significantly higher than the rate for men. They were also less likely to have been trained in Veterans Administration hospitals.

The background questions on Board certification revealed additional differences between the two groups. Women were less likely to have attempted any of the three stages of Board certification (application, written examination, and oral examination), but this trend is changing: 69.5% of the women respondents who had received certification had done so between 1966 and 1978; 45% had done so since 1975 (versus 55.3% and 23.2% for men) (see table 2). Ninety-five percent of the respondents who had participated in ABPN examinations were men, and far more men than women were certified in general psychiatry only. The two groups were equally likely to be certified in both general and child psychiatry or in both neurology and general psychiatry.

Factors Inhibiting Certification

In this section we found only one significant difference between the two groups: more women than

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TABLE 1
Time Period in Which Men and Women Questionnaire Respondents Completed Psychiatric Residency

Period	Men (N=2,559[a])		Women (N=346[b])	
	N	% of Men	N	% of Women
1973–1978	515	20.1	118	34.1
1967–1972	607	23.7	95	27.5
1960–1966	580	22.7	58	16.8
Before 1960	799	31.2	66	19.1
New residents	44	1.7	8	2.3
Not finished nor enrolled in residency	14	0.5	1	0.3

[a]Four questionnaires from men respondents did not contain this information.
[b]Five questionnaires from women respondents did not contain this information.

TABLE 2
Time Period in Which Men and Women Questionnaire Respondents Received Board Certification

Period	Men (N=2,542[a])			Women (N=340[b])		
	N	% of Men	% of Certified Men	N	% of Women	% of Certified Women
1975–1978	363	14.3	23.2	68	20.0	45.0
1966–1974	504	19.8	32.1	37	10.9	24.5
Before 1966	701	27.6	44.7	46	13.5	30.5
Not certified	974	38.3		189	55.6	

[a]Twenty-one questionnaires from men respondents did not contain this information.
[b]Eleven questionnaires from women respondents did not contain this information.

men cited performance fear of both the oral and written examinations as reasons for not taking the Boards.

Certification Policies

Men and women respondents differed in their recommendations for changes in the Board certification policies. Slightly more women than men indicated that the system needs reworking. For example, a greater percentage of women thought that residency programs and the ABPN together should be responsible for certification and that there should be separate boards for neurology and psychiatry. More men felt that the ABPN alone should be responsible for certification. Men were also more opposed to the unpopular idea of having one board with separate testing procedures for psychiatry and neurology candidates.

A greater percentage of the women psychiatrists were among the majority favoring the following proposals: 1) ranking residency programs with respect to pass-fail rates, 2) making these pass-fail statistics available to potential residency applicants, and 3) offering the written certifying examination during residency. However, a greater percentage of the men were represented in the majority of respondents who agreed with the following statements in the questionnaire:

1. Residency programs should be given available knowledge profiles of their graduates.
2. Character and emotional stability should be used as eligibility criteria.
3. Certification should not be granted upon satisfactory completion of the residency alone.

Men were also more likely to support additional subspecialty certification, e.g., forensic, administrative, and adolescent psychiatry.

Recommendations Regarding the Certifying Exams

There was only one significant difference between the two groups in their recommendations on the examinations. More men than women supported the popular recommendation to retain both the oral and written examinations and the neurological sections of each.

Personal Experience with ABPN

The majority of those who had taken the Boards experienced stress from the time and effort involved and from fear of the oral examination. A higher percentage of women than men respondents were among this group. Similarly, a higher percentage of women were among the minority who were stressed by monetary cost, difficult logistics, insufficient time for completing the examination, and fear of the written exam. More women than men, however, felt that they had been advised adequately of the judgment criteria for the examination. There were no other significant differences.

DISCUSSION

It has been stated elsewhere (1, 2) that 40% of the women psychiatrists in this country are foreign medical graduates (FMGs), which is many more than in our study (25%). This may be because only APA members were sampled, or perhaps the number of U.S.-trained women is increasing fast enough to offset the increasingly large number of FMG women psychiatrists (1, 2).

The increase in the number of women psychiatrists found in our sample has been noted elsewhere (2). In addition, we noted a striking increase in the percentage of women certified in the period 1975–1978 compared with 1966–1974 or pre-1966. It is possible that this is a sampling error caused by the likelihood that women who were recently certified were more likely to return a questionnaire such as this. However, both of these findings may be accounted for in part by the influence of the women's movement and its encouragement of ambition and competition in women. For example, the APA Board of Trustees established a Committee on Women in 1974, the goals of which include promoting the academic involvement of women psychiatrists and providing emotional support systems for them.

That fewer women than men seek Board certification may be explained by our finding that women have a greater performance fear of the examination than men, despite similar success rates. This performance

fear, or anxiety, was described by Moulton (3) in her survey of men and women graduate analysts invited to make public speeches. She found that 50% of the women versus 20% of the men refused to speak in public. Cost in time and effort was not cited differently by the two groups.

More women than men indicated that the certification process needed reworking, perhaps because the women experienced more stress from the certification process. Horner (4, 5) found that women's actual performance worsened under competitive achievement conditions, while the reverse was true for men. These findings may suggest an inherent bias against women in an examination process that involves competition to determine achievement. Alternative testing procedures based on a less competitive model could be explored further.

The difference between the two groups' opinions regarding eligibility criteria and residency training program profiles could be the result of women being less willing to be judged, especially by men, who fill most residency training directorships and Board examiner positions. Alternatively, women psychiatrists may wish only the more general information made public, while the men want specific individual information made public. This is consistent with the different responses to competition seen in men and women.

The greater desire among men than women for more subspecialty certification may be related to Horner's finding (4, 5) that men's motivation for success increases under competitive achievement situations while women's decreases because they are caught in the double bind of being reluctant to be either a failure or a success. Some studies (6) suggest that men who achieve learn to expect rewards, whereas women learn to expect punishment. Also, as Applegarth (7) has noted, some women experience fears about sexual identity and unattractiveness to men, as well as guilt and primitive fears of retaliation and object loss, when being competitive or successful. Some successful women also regard their position as deviant (8, 9).

The women respondents' reports of greater stress from the cost in time and effort involved in the certification process were unrelated to the outcome of the exam. Role strain may be a partial explanation of the greater stress that women psychiatrists reported (10 and personal communication from N. Kaltreider, 1975). This possibility has been discussed elsewhere (11), and further research is necessary to confirm whether this was indeed the case with our women respondents. However, it has been suggested (12) that the demands of two disparate roles require more than twice the effort than would be necessary to perform either alone. This degree of role strain does not exist for men (9). It is interesting that the timing of the Board examination—two years after residency completion—coincides with the time that many men psychiatrists are most heavily invested in their careers, while many women psychiatrists are likely to have small children to care for. More data are needed to substantiate the hypothesis that the coincidence of timing is causally related to fewer women being Board certified.

The Board could reduce this role strain for women by providing greater flexibility in where, when, and how the examination can be taken. This could be done by allowing psychiatrists to take Board examinations over longer periods of time and by providing day care for children during the examination.

It is our hope that these data will be useful in providing the framework for a design for change, such as that envisioned by the APA membership when they passed the 1976 referenda on the American Board of Psychiatry and Neurology. The Board has already begun to institute some changes over the last three years. Our data show major differences in men's and women's responses to the Board certification process, which can be an important guide to any future changes in that process.

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Transcendental Meditation Versus Muscle Relaxation: A Two-Year Follow-Up of a Controlled Experiment

BY DAVID C. ZUROFF, PH.D., AND J. CONRAD SCHWARZ, PH.D.

In this questionnaire survey the authors measured the outcome among 20 students randomly assigned to muscle relaxation training and 19 assigned to transcendental meditation at one year (the number of respondents in each group was 13 and 16, respectively) and two and one-half years (the number of respondents was 18 and 17, respectively). At both follow-ups there were no differences between the groups in frequency of practice or satisfaction. In both groups, less than 25% reported more than moderate satisfaction, and less than 20% practiced as much as once per week. Subjects' expectancies at nine weeks predicted their satisfaction and frequency of practice at two and one-half years. The authors conclude that although some subjects (15%–20%) do enjoy and continue to practice transcendental meditation, it is not universally beneficial.

Well-controlled studies of the short-term therapeutic effects of transcendental meditation have found it to have no more effect than placebo treatments (1), or at most to have moderate effects on trait anxiety (2). Related meditative techniques have not proven any better (3, 4). It is possible, however, that the wide range of benefits claimed by the technique's proponents are obtained only after long-term practice. The present study was designed to answer three questions concerning the long-term effects of meditation: 1) Do initiates in transcendental meditation perceive the practice to have been beneficial? 2) How high is the dropout rate after training? 3) Is it possible to predict who will continue to practice and benefit from meditation?

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METHOD

The 60 subjects in the original study (2) were students from an introductory psychology course who volunteered with the understanding that they would be randomly assigned to receive training in either transcendental meditation or muscle relaxation, or to a no-treatment control group. All procedures were carefully explained before subjects consented to participate. Training sessions in transcendental meditation and muscle relaxation were conducted by, respectively, experienced initiators from the Student International Meditation Society and graduate students in psychology. The relaxation technique was Paul's version of Jacobsonian muscle relaxation (5), which involves alternately tensing and releasing the major muscle groups of the body. Training in relaxation was designed to have the same potent, nonspecific effects as training in meditation. The nine-week treatment period of the original study was dictated by the length of the students' semester. Information was collected concerning numerous variables that were thought to be potential predictors of response to treatment. Two follow-ups were conducted, the first one year after training and the second two and one-half years after training.

One year after training, subjects in the two treatment groups were mailed questionnaires asking them to describe their current frequencies of practice and to rate on a 5-point scale their overall satisfaction with their technique. This procedure was repeated two and one-half years after training; the questionnaire contained the additional question of whether there had been any intervals of a month or more during which the respondent had practiced more frequently than he or she did at present. The respondents were offered payment for returning the second questionnaire. Of the 19 meditation subjects, 16 returned the first questionnaire and 17 returned the second; of the 20 relaxation subjects, 13 returned the first questionnaire and 18 returned the second.

RESULTS

Perceived Benefit

At the one-year follow-up only 25% (N=4) of the meditation subjects and 15% (N=2) of the relaxation subjects described themselves as more than moder-

ately satisfied. At the follow-up after two and one-half years the proportions were 25% (N=4) and 22% (N=4), respectively. The differences between the groups were not statistically significant at either follow-up.

Frequency of Practice

As would be expected, frequencies of practice were low for both groups. Nineteen percent (N=3) of the meditation subjects and 15% (N=2) of the relaxation subjects were practicing once per week or more at one year. At two and one-half years 18% (N=3) of the meditation subjects and 11% (N=2) of the relaxation subjects were practicing that frequently. There were no statistically significant differences between the groups' frequencies of practice at either follow-up. More than half of the subjects in each group practiced less than once a month or not at all. Clearly, the large majority of both meditation and relaxation subjects essentially discontinued use of the techniques and felt that they had not been particularly beneficial.

Several interesting results emerged when the meditation group's frequencies of practice at one and two and one-half years were cross-tabulated. First, no subject who practiced infrequently (less than once per week) at one year was practicing frequently (once per week or more) at two and one-half years. On the other hand, every subject who practiced relatively frequently at one year continued to practice frequently at two and one-half years, and some increased their frequencies of practice. Each of the frequently practicing subjects described himself or herself as more than moderately satisfied at both follow-ups. These results imply that there are no "late bloomers" among initiates in transcendental meditation or returnees to the fold, but that there is a small group of 15%–20% who value meditation and continue to practice it with some regularity. These conclusions apply equally well to muscle relaxation trainees.

A third group of subjects—the occasional users—can be distinguished from the subjects who practiced regularly and those who virtually discontinued use of their technique. Seventeen percent (N=3) of the relaxation group and 29% (N=5) of the meditation group reported that there had been periods of a month or longer between the two follow-ups during which they practiced more frequently than they were practicing at two and one-half years.

Prediction of Response to Treatment

Multiple regression techniques were used to determine whether the measures of outcome (satisfaction and frequency of practice) could be predicted, given knowledge of the trainees' personality and behavioral characteristics. The potential predictors included trait anxiety, locus of control, maladjustment, drug and alcohol use, expectancies for benefits, attitudes of significant others, frequencies of practice during the nine-week treatment period, and experiences and heart rate while practicing. The measures employed have been described elsewhere (2).

Hierarchical multiple regression analyses (6) were performed, regressing the outcome variables on group membership (meditation versus relaxation), a potential predictor variable, and the product term representing the interaction of group and the potential predictor. The effect of each independent variable was tested against the error term from the regression equation in which that variable was the last one entered (Model I error [6]).

None of the interaction terms approached significance, indicating that the relationships between potential predictors and outcome variables were the same for meditation as for relaxation. Of the potential predictor variables, only the measures of expectancies for benefits were significantly related to the outcome variables. Expectancies measured after nine weeks of treatment were significantly related to both satisfaction ($t=3.5$, $df=31$, $p=.002$) and frequency of practice at 30 months ($t=2.8$, $df=32$, $p=.01$). Expectancies measured immediately after training were also significantly related to frequency of practice ($t=3.2$, $df=32$, $p=.04$) and tended to be related to satisfaction ($t=1.7$, $df=31$, $p=.11$). These results indicate that satisfaction with and frequency of practice of meditation and relaxation can be predicted from expectancies measured as early as nine weeks after training and, to a lesser extent, from expectancies immediately after training. The personality variables were not predictive of the measures of outcome; however, one must remember that the small sample provided only modest power to detect effects.

Finally, in a further search for correlates of continued treatment use, we conducted a series of one-way analyses of variance of the predictor variables, with subjects divided into relatively regular users of meditation or relaxation (once per week or more), dropouts (less than once per month), and occasional users. As might be expected, the dropouts had significantly lower expectancies after nine weeks of treatment than did the other groups ($F=6.5$, $df=1,32$, $p<.01$). It is of interest that the occasional users' scores on Rotter's locus of control scale (7) (mean=8.2) were more internal than those of the dropouts and regular users (mean=13.4) ($F=4.1$, $df=1,32$, $p<.05$).

DISCUSSION

The results of the two follow-up studies can be summarized as follows: 1) most subjects radically reduced their frequencies of practice of transcendental meditation or muscle relaxation within a year of training; 2) there was a small group of regular, satisfied users who continued to practice even two and one-half years after training; 3) there was another small group who typi-

cally did not practice but who occasionally used their techniques more regularly; 4) subjects' expectancies after nine weeks were related to outcome at two and one-half years; and 5) the occasional users tended to score as internals on Rotter's locus of control scale (7).

The three questions posed in the beginning of this article can now be given reasonably clear answers. First, most initiates in transcendental meditation did not perceive the technique as having been of major long-term benefit. Second, the long-term dropout rate was very high. This finding is consistent with other reports (1, 8, 9) of dropout rates of 50% or more. Third, one can predict treatment response by asking subjects whether they expect to benefit; however, no predictive personality or behavioral characteristics of the high expectancy-good response group were found. Although internality on the locus of control scale was unrelated to satisfaction or frequency of practice, it did predict a pattern of occasional use of the techniques.

These rather negative findings contrast sharply with the encouraging results obtained by earlier investigators (10). The difference may well be a consequence of the superior design of the present study, which was a longitudinal study of randomly assigned subjects. Many studies reporting positive results have been cross-sectional investigations of self-selected meditators with unknown dropout rates. Although the present results are based solely on self-report, one would expect that the true dropout rate was, if anything, higher than that reported, since biases in responding would be in the direction of underreporting discontinuation.

One may conclude from the results of the present study and the few other well-controlled studies that transcendental meditation is far less effective and satisfying than its proponents claim. After the initial surge of enthusiasm and sense of well-being pass, the modal initiate ceases to practice with any regularity and no longer feels great satisfaction with the technique. Professionals who are considering the use of meditation or relaxation in clinical settings may wish to use measures of expectancy to select the subset of patients who are likely to find the techniques helpful.

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Neuroleptic Effect on Desipramine Steady-State Plasma Concentrations

BY J. CRAIG NELSON, M.D., AND PETER I. JATLOW, M.D.

The authors examined the effect of neuroleptic drugs on desipramine steady-state plasma concentration in 30 patients who received similar milligram per kilogram doses of desipramine. Fifteen of these patients also received a neuroleptic drug and had desipramine plasma levels twice that of the 15 patients who received desipramine alone. The authors discuss the implications of the magnitude of this difference for therapeutic response and incidence of side effects.

Tricyclic antidepressant drugs are often used in combination with antipsychotic agents to treat depressed schizophrenic patients (1) as well as patients with delusional depression (2). Use of this combination requires consideration not only of the therapeutic interaction of the two drugs but also of their pharmacokinetic interaction. Although Gram and coworkers (3–6) have extensively studied the effects of neuroleptic drugs on the elimination of single-dose nortriptyline and imipramine in man and rat, the clinical implications of this interaction during continued tricyclic administration are less clear.

Moody and associates (7) first described elevation of tricyclic plasma levels after chlorpromazine was administered to a patient receiving imipramine. Gram and associates (3, 4), using single dose ^{14}C-nortriptyline, demonstrated that perphenazine, haloperidol, and chlorpromazine increased the plasma concentration of nortriptyline and decreased the concentration of metabolites in plasma and urine. Subsequently, they demonstrated that perphenazine inhibited 2-hydroxylation of a single dose of ^{14}C-imipramine and that this effect was especially pronounced for desipramine (5, 6). Few studies have examined this effect on steady-state tricyclic levels. Olivier-Martin and associates (8) demonstrated that administration of levomepromazine resulted in a 30% rise in imipramine and a 200% rise in desipramine steady-state plasma concentrations. A similar effect on nortriptyline steady-state plasma concentration was recently noted by Vandel and associates (9) in patients receiving amitriptyline combined with a phenothiazine. Our report examines steady-state plasma concentrations of desipramine in patients being treated for depression with desipramine alone or in combination with an antipsychotic drug.

METHOD

Our sample consisted of 30 inpatients, 15 of whom had nondelusional unipolar depression, met Research Diagnostic Criteria (10) for major depressive episode, and were treated with desipramine alone (desipramine group); 15 delusional patients, all of whom were treated with a combination of desipramine and an antipsychotic agent (desipramine-antipsychotic group), met RDC for either major depressive episode, psychotic (N=10), or schizoaffective disorder (N=5). Although the desipramine group was slightly younger (table 1), the difference in mean age for the two groups was not significant. Furthermore, in this sample, the correlation for age and steady-state drug level was −.08. Desipramine levels were lower for the men; however, the sex ratio for the two groups was comparable. None of these 30 was receiving other drugs known to affect hepatic microsomal enzymes (e.g., barbiturates or methylphenidate).

Desipramine was administered orally in three divided doses at 10:00 a.m., 2:00 p.m., and 6:00 p.m. Dosage was calculated using a ratio of 2.5 mg/kg of body weight. The actual mean dosages received by the two groups, 2.55 and 2.50 mg/kg, were nearly identical. One-third the calculated dose was given the first day, two-thirds on the second day, and the full dose on the third day and thereafter. Plasma samples were drawn at 9:00 a.m. Heparinized glass syringes were used to avoid contact with rubber stoppers. Plasma concentrations of desipramine were determined in duplicate with a modification of a gas chromatographic

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TABLE 1
Desipramine Plasma Concentrations and Patient Characteristics for 30 Depressed Patients

					Desipramine			
	Sex		Age (years)		Dose (mg/kg)		Plasma Level (ng/ml)[a]	
Group	M	F	Mean	SD	Mean	SD	Mean	SD
Desipramine alone (N=15)	5	10	38	13	2.55	0.18	110	81
Desipramine-antipsychotic (N=15)	4	11	48	18	2.50	0.22	255	115

[a]p<.002, Mann-Whitney rank order test.

nitrogen detector method previously described by Bailey and Jatlow (11). The average steady-state concentration was determined from three separate consecutive samplings after the patient had been on a constant dose for at least 10 days.

All 15 delusional patients received the antipsychotic drug first in order to facilitate clinical management and to prevent the possible exacerbation of psychosis by the antidepressant (12); 12 patients were treated with perphenazine (16–64 mg/day), 2 with haloperidol (6 and 20 mg/day), and 1 with thiothixene (12 mg/day). The dosage of the antipsychotic drug was determined depending on the needs of the patient.

RESULTS

The mean steady-state desipramine concentrations were 110 ng/ml for the desipramine group and 255 ng/ml for the desipramine-antipsychotic group (table 1). A nonparametric Mann-Whitney rank order test indicated the samples differed significantly ($p<.002$).

Although during the study no patient received other medications known to affect microsomal enzymes, 13 patients (3 in the desipramine group and 10 in the desipramine-antipsychotic group) received medications such as an antibiotic, a diuretic, a digitalis preparation, or benztropine mesylate. One of these patients had used barbiturates until 4 weeks before plasma levels were obtained. This prior use of a known enzyme inducer may explain the patient's unusually low plasma level (67 ng/ml) within the desipramine-antipsychotic group. Another of these 13 patients was chemically hypothyroid (thyroxine, 3.6 μg/100 ml; estimated free thyroxine, 0.8 ng/100 ml), although there were no clinical signs of hypothyroidism. Whether this finding was related to her unusually high plasma level (322 ng/ml) within the desipramine group is unknown. Desipramine plasma levels of the 7 patients who received benztropine mesylate, 1–2 mg/day, with the antipsychotic drug did not differ significantly from the plasma levels of the 8 patients receiving an antipsychotic without benztropine.

In order to assure that the difference in desipramine plasma levels observed in this sample was not the result of medication other than the antipsychotic agent, plasma levels of the 17 patients receiving no other medications were examined. The mean (±SD) steady-state plasma concentrations were 95±61 ng/ml for the 12 patients receiving desipramine alone and 274±154 ng/ml for the 5 patients receiving desipramine and an antipsychotic but no other drugs; the difference between these two samples was significant (Mann-Whitney, $p<.01$).

DISCUSSION

In our patient sample, the magnitude of the effect of antipsychotic drugs on desipramine plasma concentration is considerable. The fact that the plasma level was more than double in patients receiving antipsychotic drugs suggests that the magnitude of this effect may be of clinical importance. The relationship of plasma levels of desipramine and therapeutic response has not been well-defined; however, if analogous to nortriptyline this drug has a therapeutic window, which Friedel and associates (13) suggest may be between 40 and 160 ng/ml, then the effect of antipsychotic drugs on plasma concentration after usual doses of desipramine would be sufficient to raise the drug level beyond the therapeutic range. The elevated plasma concentration might also increase the incidence of toxicity. In our sample the 4 patients with serious side effects—urinary retention (N=1), a grand mal seizure (N=1), and a toxic brain syndrome (N=2)—had desipramine concentrations above 200 ng/ml. While the concurrent use of an antipsychotic drug in these patients may have also contributed to toxicity, each of the patients improved following reduction of the antidepressant drug without a change in the antipsychotic agent.

Although previous reports (3–8) suggest the metabolism of various tricyclic drugs may be impaired by different antipsychotic drugs, our data, as well as those of others (7, 8), suggest that desipramine may be particularly sensitive to this effect. The design of this study does not exclude the possibility that delusional patients may metabolize desipramine differently; however, the data of Gram and associates (3–6) indicate that inhibition of microsomal enzymes by neuroleptics is a more likely explanation. Although the clinical value of determining tricyclic concentrations has been debated, the use of combination antipsychotic-antidepressant drug therapy may be one indication for their measurement.

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Brief Group Psychotherapy with Chronic Hemodialysis Patients

BY DAVID R. CAMPBELL, M.D., AND BIRENDRA K. SINHA, PH.D.

The authors selected 11 chronic hemodialysis patients for a brief group psychotherapy program, the primary objective of which was to produce cognitive and affective changes pertaining to the illness. Each of the 10 therapy sessions lasted 1 1/2 hours and was conducted while the patients were actually on dialysis. Three therapists rated outcome as "good," "fair," or "poor" on 10 objective criteria, and subjective impressions of the group experience were obtained from each patient. The study demonstrated the beneficial effects of group therapy, although there was no statistically significant difference between patients in the three outcome categories.

Several studies have suggested that hemodialysis patients experience a great deal of psychological stress manifested by a variety of behavioral symptoms (1–4). Specifically, hemodialysis patients are found to suffer from disturbances of body image (5–7), sexual fears including impotence (7, 8), and fear of death (9). Perhaps the most important observation has been the presence of suicidal tendencies and depression (3, 5, 8–11). Abram and associates (11) reported that the incidence of suicide among hemodialysis patients is greater than 400 times that of the general population. The patients who deteriorate and eventually die are those who demonstrate depressive symptoms, such as withdrawal, acting out behavior, or refusal to follow dietary restrictions. Depression and suicidal behaviors along with denial of illness require some form of psychotherapeutic intervention in order for the patients to cope with the problems of chronic illness (6, 10, 12, 13).

Although psychiatric treatment of hemodialysis patients has included both individual (14) and group (15–17) psychotherapy, group psychotherapy appears to be the preferred mode of treatment in many renal units of hospitals. Published reports indicate success with a variety of group psychotherapy methods that emphasize different dimensions of the therapy process. For example, Wijsenbeck (7) used group therapy while the patients were off dialysis, and the therapist's task was to "listen and observe." In a recent article, Buchanan (15) described a short-term, open-membership group

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therapy program that involved both kidney transplant patients and their families. The therapy process focused on group cohesiveness, imitative behavior, development of socialization techniques, demonstration of family dynamics, ventilation, interpersonal learning, instillation of hope, altruism, information dispersal, and universality. Although the group therapy did not produce expected change in all 10 dimensions, Buchanan observed that the group was helpful in providing emotional support to the patients and the opportunity to learn adaptive strategies from others in the group.

The main purpose of our study was to ascertain the effectiveness of brief group psychotherapy with long-term hemodialysis patients. We used objective criteria for selecting patients and determining therapeutic outcome. A major innovative approach used by us was to conduct group sessions while the patients were actually on dialysis. The therapist and cotherapists were active participants in all group sessions and focused on specified aspects of the therapy process.

METHOD

Patient Selection

We selected 11 dialysis patients (8 men and 3 women) ranging in age from 20 to 50 years for group psychotherapy out of 18 patients referred for psychiatric consultation by the nephrologist in charge of the renal unit of the University of Alberta Hospital. Depression and management problems were the primary reasons for psychiatric referral. All patients had high T scores (> 80) on the Depression and Hypochondriasis scales of the MMPI.

The demographic characteristics of the patients are presented in table 1. The majority of the patients were under 25 years of age. The younger patients had a tendency to act out more and to present management difficulties. Although all 18 patients initially referred for psychiatric consultation had problems related to depression, somatic complaints, and hostility, the following criteria were used for selecting patients for the group therapy program.

1. *Severity of depression versus management problems.* The patients exhibiting difficult management problems such as acting-out behaviors or defiance and hostility toward the staff and treatment regimen were included in the program. Severely depressed patients were referred for more intensive individual therapy and chemotherapy.

2. *Acceptance versus denial reaction.* It appeared to us that acceptance of illness was related to severe depression, and we excluded such patients from the program. However, we considered depression combined with hostility and acting-out problems suitable for group therapy.

3. *Sexual concerns.* The patients with primary psychological problems related to sexual effectiveness were excluded from the study because we felt they could be best treated with individual psychotherapy.

4. *Indication of psychotic process.* The patients exhibiting clearly identifiable psychotic symptoms were considered unsuitable for group therapy.

5. *Length of time on dialysis.* In order to introduce patients to group processes at an early stage of the disease, we excluded those who had been on dialysis for more than 6 months from this study.

TABLE 1
Demographic Characteristics of 11 Dialysis Patients Selected for Brief Group Psychotherapy

Characteristic	Number	Percent
Age (years)		
20–24	5	45.4
30–34	2	18.2
35–39	1	9.1
45–49	2	18.2
50–55	1	9.1
Marital status		
Single	5	45.4
Married	6	54.6
Employment status		
Employed	2	18.2
Student	3	27.3
Housewife	3	27.3
Unemployed	3	27.3
Socioeconomic status		
Middle class	9	81.8
Working class	2	18.2

Group Process

Two groups consisting of 6 and 5 patients each were formed. The brief group therapy sessions took place every other week and lasted 1½ hours. There were a total of 10 sessions.

The primary objective of the group therapy was to produce cognitive and affective changes pertaining to the illness. Specifically, cognitive changes were concerned with the patient's perception and explanation of illness. As suggested by Pritchard (18), a physically disabled person is likely to perceive his illness "as an enemy which is attacking him" (p. 55). Such a perception may, in turn, cause undesirable affective responses, such as hostility, anger, anxiety, and feelings of helplessness. Hence, the group psychotherapy aimed at changing the patient's perception so that illness could be viewed as a challenge rather than an enemy. We expected that the new perception of kidney failure as a problem-solving task would produce positive emotional responses in the patients.

The group sessions were conducted by a therapy team consisting of one psychiatrist and two nurses, one from the renal unit and the other from the hospital's psychiatry ward. The psychiatrist was the therapy leader, and the two nurses served as cotherapists. The group process focused on active verbalization and expression of feelings. As an active member of the

group, the group leader took a confronting attitude. Rational examination of feelings and cognitive restructuring were attempted with respect to specified psychological difficulties of the group members. At times, the therapist provoked the patients with direct interpretation of the initial complaints. Personal and social consequences of low motivation, hostility, and depressed attitudes with respect to the dialysis management routine were discussed in all sessions. While the therapist was active and direct, the cotherapists were supportive. Their supportive role appeared necessary for the depressed patients to express themselves and contribute to the group sessions.

The therapists did not attempt depth analysis of the patient's underlying motivations. In fact, healthy defenses, such as rationalization, suppression, or substitution, were reinforced. However, the group process provided some insight into unhealthy defenses, such as denial, displacement, and projection, that were frequently employed by the patients during initial therapy sessions.

Evaluation Criteria

We evaluated the effectiveness of the group therapy program after 10 sessions in terms of clinical judgment of the therapy team obtained through their ratings on 10 relevant criteria and the patients' evaluation of their group experience. We selected the following relatively objective criteria for evaluating the program because of their clinical importance in the management of dialysis patients: 1) control of diet and fluid control; 2) improved interpersonal communication with staff, patients, relatives, and friends; 3) return to work, including part-time employment and/or a suitable educational program; 4) acceptance of the kidney unit; 5) decreased fear of actually running on the machine; 6) reduced depression and/or demanding and acting-out behavior; 7) decreased somatic complaints, such as nausea, vomiting, headache, pain, during dialysis or ward activities; 8) ability to cope with minor (e.g., vein clot) and major stress (e.g., death of a patient on dialysis in the next bed); 9) changes in the patient's attitudes and feelings toward staff, relatives, and friends; and 10) changes in the attitudes and feelings of the staff, relatives, and friends toward the patient.

Each patient was rated "good," "fair," or "poor" by the psychiatrist and the two nurses independently. The three raters provided a total of 30 ratings for each patient. The most frequently rated category was used as the final score; e.g., if a patient received 15 good, 8 fair, and 7 poor ratings, he was placed in the good category. In case of equal distribution of the three categories the psychiatrist made the final judgment about the therapy outcome. Thus, the therapy outcome was based upon a composite evaluation by pooling the observations of the therapy team. Although the therapists rated the patients independently, the ratings cannot be considered truly independent because the raters worked together in group sessions. Hence, no attempt was made to establish interrater reliability.

We obtained the patients' evaluation of the group therapy program in the form of written comments about their overall subjective impressions. No attempt was made to quantify the patients' evaluations.

RESULTS

The initial group sessions revealed that denial of illness and fear of death were the primary areas of concern. Reference to poor kidney function and its fatal consequences was avoided in group discussions. When there was a death on the renal unit, the group therapy patients had a tendency to ascribe the cause to heart or lung failure rather than the kidney problem. As the group progressed, the members became increasingly supportive of each other. They became more objective in dealing with stressful situations, such as death of a dialysis patient.

Depression and apathy were other areas of major concern in the therapy sessions. However, increased verbalization, open communication, and close interaction between members were clearly noticeable during later group sessions. The patients showed greater concern for the welfare of other members and exchanged advice for better management of their kidney problem. The group therapy sessions seemed to promote the feeling of self-worth and hope in the future. Even sexual matters were discussed in a lighter vein in the form of jokes.

We felt that the degree of mutual trust and group cohesiveness among dialysis patients was much lower than that achieved by other successful therapy groups. The lack of trust was mostly with reference to doctors, nurses, and family members. These physically healthy persons were, perhaps, constant reminders to the patients about their dependence on others. During the period of our study, several dialysis patients had died in the unit. Death of fellow patients might have reactivated the group members' fear of death and dampened their faith in the effectiveness of the dialysis machine. The same psychological processes might account for the slow development of group cohesiveness. It was difficult to achieve open communication and close interpersonal relationships between group members when denial of illness was one of the frequently used defense mechanisms. However, as the group progressed, the patients tended to accept their handicap as well as the fact that their survival was contingent upon regular use of the dialysis machine.

Approximately 64% (N=7) of the patients were rated good, 18% (N=2) were rated fair, and 18% (N=2) were rated poor. A chi-square test did not reveal significant difference between the three outcome categories. However, when the combined good and fair categories are compared with the poor category,

the difference approaches statistical significance ($p<.10$). The lack of a statistically significant difference is probably due to our small number of cases.

All patients indicated in their written comments that they benefited from the group experience with respect to depression, acceptance of the illness, body image, and self-esteem. Even those who were judged poor by the therapy team had positive feelings about the group therapy program. In general, the patients became more cooperative in following the strict regimen imposed by the dialysis treatment program.

CONCLUSIONS

This study demonstrates the value of group therapy without taking the patients off the dialysis machine, providing a situation of direct confrontation since the machine is an immediate reminder to the patients that they have a serious physical disability. Our criteria for selecting the patients and evaluating the therapy outcome appear valid and promising; however, in view of a statistically insignificant difference in the therapy outcome as rated by the treatment team, further research is needed to identify the variables most sensitive to therapeutic intervention. Our study also does not compare the effectiveness of individual and group therapy. Such a controlled comparison on quantifiable dimensions would certainly help the consulting psychiatrist in planning therapeutic programs suitable for individual patients.

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Credibility and Incredibility: The Psychiatric Examination of the Complaining Witness

BY ROBERT LLOYD GOLDSTEIN, M.D.

Psychiatrists are called on to evaluate the credibility (and incredibility) of complaining witnesses in criminal proceedings despite a longstanding controversy about their ability to accurately make such determinations. The author discusses the history of such psychiatric activity from Freud through the Alger Hiss perjury trial, its current status in terms of benefits to the criminal justice system, the legal and social questions it raises (e.g., the competing issue of the complainant's right to privacy), and the court's restrictions on the psychiatrists. He presents two illustrative case histories.

In 1906, in one of his rare ventures into the area of forensic psychiatry, Sigmund Freud delivered a lecture before a University of Vienna law class titled "Psycho-Analysis and the Ascertaining of Truth in Courts of Law" (1). He suggested that the psychiatrist may have at his disposal scientific means by which the testimony of witnesses can be tested for truthfulness and reliability but cautioned that these scientific methods were of a highly experimental nature and for research purposes only. They were not intended to influence the verdict of the court.[1] He thus anticipated a controversy that has raged on for three quarters of a century regarding the psychiatrist's special ability to evaluate the credibility of the complaining witness in a legal proceeding.

Davidson (2) contended that the psychiatric appraisal of the witness represents a new field of forensic psychiatry. He described nine clinical conditions that the psychiatrist alone is qualified to discern and to relate to the credibility issue: 1) schizophrenia, 2) senile psychoses, 3) manic states, 4) paranoid states, 5) drug addiction, 6) mental deficiency, 7) alcoholism, 8) psychoneurosis, and 9) psychopathic personality. Davidson (3) also stated, "If the psychiatrist were given all the facts about the witness' background; if he were given time to do a thorough physical and mental examination; if he could take a psychiatric history on the witness, he could give the trial lawyer a solid opinon about testimonial capacity."

The best known use of psychiatric evaluation of a complaining witness' credibility came in the second Alger Hiss perjury trial in 1949. According to Slovenko (4), the judged ruled,

> It is apparent that the outcome of this trial is dependent, to a great extent, upon the testimony of one man—Whittaker Chambers. Mr. Chambers' credibility is one of the major issues upon which the jury must pass. The opinion of the jury—formed upon their evaluation of all the evidence laid before them—is the decisive authority on this question, as on all questions of fact. The existence of insanity or mental derangement is admissible for the purpose of discrediting a witness. (4, p. 45)

The judge allowed a psychiatrist, Dr. Carl A.L. Binger, to testify that, in his expert opinion, Chambers was a "psychopath with a tendency toward making false accusations" (4, p. 45). This expert opinion was based on Binger's personal observation of Chambers on the witness stand and on a reading of some of Chambers' poems, plays, and book reviews. No psychiatric examination per se was carried out. The jury chose to disregard the psychiatric testimony and found Hiss guilty as charged.

In some jurisdictions, when the credibility of the complaining witness in sex offense cases is in doubt a psychiatric examination is ordered "not to determine whether the witness is telling the truth, but to determine whether the emotional or mental condition of the witness *may* affect his or her ability to tell the truth" (5). A report of the American Bar Association (6) recommended psychiatric examination of all complainant women in sex offense cases to ascertain the presence of a psychiatric disturbance. Proponents of psychiatric involvement believe that psychiatry has firmly established itself as an inextricable cog in the machinery of the law and that it would be better if the system availed itself of psychiatric expertise when the alternative

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[1] In a memorandum prepared in 1922 that is not extant, Freud deprecated any half-baked application of psychoanalytic theories in legal proceedings.

might be to rely on uniformed ignorance, myths, and misconceptions (7, 8).

Critics of psychiatric involvement have raised serious questions in regard to the desirability, accuracy, and effectiveness of psychiatric evaluations of credibility. In 1909 Wigmore (9) asked, "Where are the exact and precise experimental and psychological methods of ascertaining and measuring the testimonial certitude of witnesses?" According to Slovenko (4), the judge in a famous rape case ruled that the jury alone would best decide the issue of the complaining witness' credibility. The judge refused to allow the introduction of psychiatric testimony:

> We are not convinced that the time-honored and well settled and undefined rule of impeachment of the veracity of a witness should thus be innovated upon. It is yet to be demonstrated that psychological and medical tests are practical and will detect a lie on the witness stand. (4, p. 49)

Other courts have been even more skeptical:

> The divergence of psychiatric opinion and its frequent inexactness render its value minimal in enabling the jury to decide the issue of credibility . . . the jury after being subjected to several conflicting, equivocating and highly technical psychiatric opinions may actually be more confused than before. (4, p. 59)

Another court failed to see the benefit to be gained from "an amateur's voyage on the fog-enshrouded sea of psychiatry" (4, p. 59).

There is a great deal of concern about reducing the prerogatives of the jury and giving a psychiatric expert the power to decide the issue of credibility (10), about the competing interests of witness credibility and privacy, and about the possible misuse of psychiatry by calling for psychiatric opinions based on questionable practices that are not firmly grounded in good clinical evaluation methods. Slovenko (4) concluded,

> To obtain an ideal climate for an effective psychiatric evaluation, a number of legal reforms would be necessary that might be unwise either from a social or legal point of view. If this is the case, then psychiatrists and jurists should realize the limitations that the legal procedure places on the accuracy and effectiveness of the psychiatric examination and, in turn, on the psychiatric opinion. (p. 56)

Following are case reports in which the court permitted testimony by a psychiatrist (myself) as to the mental state of the complaining witness. In these cases it was the contention of the defense that the mental disorder of the complaining witness and not reality was the source of the complaint. A direct psychiatric examination of the complainant, an approach that was promoted as being more accurate and rewarding, was requested in both cases. The court denied permission but did allow psychiatric testimony based on a review of the complainant's psychiatric hospitalization records. This might be viewed as a compromise between the competing issues of a need to evaluate credibility and a desire to infringe on the privacy of the complainant as little as possible.

CASE REPORTS

Case 1. A 74-year-old woman who lived alone claimed that the son of her building superintendent had assaulted and robbed her. He was arrested the same day. He had known the complaining witness for more than 10 years and lived in the same building. He emphatically denied the charges against him but had no solid alibi. He was unemployed and had a criminal record and a history of drug abuse (he was currently attending a methadone program). Shortly before the trial it came to light that the complainant had had several psychiatric hospitalizations since age 45 for paranoid schizophrenia, characterized by delusions, hallucinations, confusion, memory lapses, and antagonistic behavior toward friends and family. During these episodes she had delusional ideas of being robbed of cherished possessions. She even accused her relatives of stealing from her. As a result of these disclosures, the court ruled that a psychiatrist retained by the defense could review her psychiatric records and testify as to the possible influence of her mental condition on her credibility. I testified that in view of her documented psychiatric history and the particular nature of her disorder, there was a substantial index of suspicion that her testimony represented another example of delusional thinking. I conceded that my opinion would be more cogent if it were based on a direct psychiatric examination of the woman. The jury acquitted the defendant. It appeared that the impact of psychiatric testimony was facilitated by the overall weakness of the prosecution's case, which was based on the complainant's word without any other corroboration.

Case 2. A 40-year-old woman charged that her boyfriend had raped her. She stated that he had visited her to inform her that their liaison could not continue because his estranged wife was returning to live with him. She alleged that he forcibly raped her after they quarreled. She made no outcry about this until 3 days later. During a gynecological examination 2 days after the alleged rape, she made no mention of a sexual assault. It was learned that she had been hospitalized twice in recent years for depressive illness with hysterical features. One month before the alleged rape she underwent a hysterectomy. The court allowed me as defense psychiatrist to review her hospital records but not to examine her directly; the court stated that a direct psychiatric examination was an unacceptable invasion of her privacy. I testified as to the possible effects of narcissistic blows to the complainant as a result of her hysterectomy and being abandoned by her boyfriend. I talked about possible relationships between narcissistic injuries of this nature, a personality makeup including depressive and hysterical traits, attempting to maintain a shaken feminine self-image, and allegations of rape at length before the jury. The jury could not find the defendant guilty of rape "beyond a reasonable doubt," and he was acquitted.

DISCUSSION

A psychiatric evaluation of the complaining witness in a criminal proceeding may be necessary to allow for informed deliberation by the jury. In cases where doubt exists as to whether the complainant may be psychiatrically impaired, information regarding the mental state of the complainant is germane and should be made fully clear to the jury if the jury is to function meaningfully. The determination of the effect of any psychiatric condition on the credibility of the complaining witness remains the sole province of the jury; nonetheless, information provided by an expert witness permits the jury to make informed decision as to that witness' worth.

To enhance the truth-finding function of the jury, courts have ruled that they have an inherent power to order the complainant to cooperate. Not only is such testimony admissible, but the Court of Appeals in New York State has reversed convictions in which such information was not put before the jury (11, 12). The court held that the inability of the jury to appreciate the nature of the psychiatric infirmity was sufficient grounds to overturn the conviction. The purpose underlying the testimony of an expert is not to substitute his or her estimation of credibility for that of the jury. Rather, it is to provide a scientific perspective for the jury according to which it can then evaluate the complainant's testimony for itself.

Scrutiny of the psychiatric condition of the complaining witness is an area of forensic psychiatry that warrants continuing study. Is the psychiatrist invading the usual province of the jury? Is he or she really determining the credibility of the complaining witness or being used to discredit the complainant improperly? Slovenko asked, "Is psychiatry here being used more for its prestige value than for its probative value?" (4, p. 56). Is the privacy of the complainant being invaded to an unacceptable extent? A New York State court has ruled that although the right to psychiatric testimony clearly exists, the defendant must make a clear showing that it is warranted in view of the infringement on the complainant's right to privacy (13). Is it the complainant who will be tried instead of the defendant? Will this serve to discourage victims of crime from pressing charges for fear they may be exposing themselves to humiliation and vilification? Proponents of this trend have already tried to expand it further: it has been argued that the presentation of critical scientific data as to the psychiatric condition of a complainant should not depend on the chance that he or she has a history of psychiatric illness warranting closer scrutiny. Where there is no history, some have contended that there is an a priori right to launch such an inquiry before trial. This would certainly place on trial all complaining witnesses, whether or not they have a psychiatric history.

I have suggested the term "incredibility" to describe that quality in a witness which renders his or her evidence unworthy of belief. Although the invention of a technical term may help to clarify an issue and avoid confusion and distortion, it can also give the appearance of creating facts for the social sciences (14). Psychiatry already has plenty of such facts in the form of technical terms, and these are reified and often misused in the courtroom and other places to overawe those who are not used to scrutinizing authorities. The danger is that determinations of incredibility, if not managed judiciously and vigilantly, might be overused and misused to harass and stigmatize complaining witnesses. It remains to be seen what impact this direction in psychiatry's involvement in court proceedings will have, what specific guidelines and safeguards will evolve for its application, and what will result from renewed conflicts between the individual's right to privacy and the jury's right to know in order to ensure a fair trail and to protect the accused. This is but a further example of the constant need to adjust two conflicting ideals in the law so that one is not sacrificed to the other. Just as Freud cautioned against "wild analysis" as a reckless and blind misuse of psychoanalysis, so must the forensic psychiatrist avoid introducing any testimony based on incomplete, inadequate, or questionable methods of clinical evaluation in order to aid the legal process in reaching more informed and intelligent decisions.

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Plasma and Saliva Levels of Chlorpromazine and Subjective Response

BY THEODORE VAN PUTTEN, M.D., PHILIP R.A. MAY, M.D., DONALD J. JENDEN, M.D., ARTHUR K. CHO, PH.D., AND CORALEE YALE, M.A.

Newly admitted schizophrenic patients (N=48) were given a test dose of 2.2 mg/kg of chlorpromazine hydrochloride by mouth. Chlorpromazine was measured in plasma and saliva at 1, 2, 3 (saliva only), 4, 6, 8, 12 (saliva only), and 24 hours by gas chromatography-mass spectography. Subjective response at 4 and 24 hours after test dose was not significantly related to either plasma or saliva level even for dysphoric responders. Dysphoric responses to chlorpromazine appear to be idiosyncratic—rooted perhaps in altered receptor sensitivity or in a patient's very personal interpretation of drug effect.

The subjective response to the first dose of antipsychotic medication is important. If the patient finds the medication agreeable or helpful (a syntonic response), all is well. A dysphoric response, however, augurs poorly. In our experience an initial dysphoric response is a powerful predictor of drug noncompliance, no matter how hard the therapist tries to correct it or how understanding he tries to be (1, 2). Even if the patient can be persuaded to continue the medication, an early dysphoric response is modestly predictive of an inferior short-term outcome, at least at conventional dosage levels (1).

The dysphoric responders in the present study experienced the drug effect as unbearable. From the very beginning they reported that chlorpromazine made them "goofy," "lazy," "mummified," "dull," or "fuzzy." All experienced the medication as "too strong" and requested, in fact demanded, that the dosage be reduced.

Since there is enormous variability in blood levels after a single dose of chlorpromazine (3, 4), we hypothesized that dysphoric responders would have deviant pharmacokinetics—in particular, higher blood levels.

METHOD

The subjects were 48 schizophrenic patients newly admitted (or readmitted) to Brentwood Veterans Administration Medical Center who had given informed consent and for whom treatment with an antipsychotic drug was considered appropriate. They were suffering from a functional psychosis, schizophrenic type, but not from organic brain disease, significant physical disease, or major drug addiction or alcoholism.

In general, the patients were admitted voluntarily (79%). Most were in young middle age (mean age±SD=32.3±9.9 years), had never been married (79%), and had a chronic disorder (mean duration=6.4±6.93 years) and a fair prognosis. On a nursing observation scale (NOSIE) (5), their mean overall degree of severity of illness was 4.72 (4=moderately ill and 5=markedly ill).

The patients were rated on four different sets of diagnostic criteria for schizophrenia: the NIMH schizophrenia criteria (6), the New Haven Schizophrenia Checklist (7), the Diagnostic Rating Scale (8), and the World Health Organization criteria (9). For 40 of the 48 patients all 4 diagnostic criteria confirmed a diagnosis of schizophrenia; for 6 patients 3 of the 4 criteria confirmed this diagnosis. Of the remaining 2 patients, 1 was rated as "probably" schizophrenic, and the other as having schizoaffective psychosis. A 2- to 7-day washout period was provided; most patients, however, had not taken medication for several months and could be presumed to be drug-free on admission.

An initial test dose of 2.20 mg/kg of chlorpromazine hydrochloride was given to each patient as oral concentrate. They were given a light standardized meal 2-

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Jane A. Kirksey assisted in data processing, and the manuscript was typed by Rosa Surrence. Lorraine A. Wittman obtained the blood and saliva specimens and did the preliminary processing; Emma Di Stefano did the gas chromatography-mass spectography analyses; Wilfred J. Dixon, Ph.D., gave statistical advice; and A. Maniau, Ph.D., provided $^{2}H_{6}$-chlorpromazine.

3 hours after the test dose. Blood and saliva samples were taken at 0, 1, 2, 3 (saliva only), 4, 6, 8, 12 (saliva only), and 24 hours. Chlorpromazine was measured in plasma and saliva by gas chromatography-mass spectography with *N, N*-bisperideuteromethyl chlorpromazine ($^2H_6$-chlorpromazine) as an internal standard. A dental cotton roll was used to collect the saliva specimens in order to avoid a psychologically distasteful instruction to "spit." Details of the collection and assay methods are given elsewhere (3).

Subjective response was assessed by a semistructured interview in which the following questions were asked: "How does the medication agree with you?" "Does it make you feel calmer?" "Does it affect your thinking?" and "Do you think this would be the right medicine for you?" The subjective response was graded on a syntonic-dysphoric continuum on a scale from +44 (maximum syntonic response) through −44 (maximum dysphoric response), as reported previously (1).

Subjective response was measured at 4 and 24 hours after the test dose. Patients were divided according to their scores into "syntonic," "indifferent," and "dysphoric" responders: greater than 5=syntonic responder, +5 through −5=indifferent responder, less than −5=dysphoric responder.

RESULTS

Plots of the logs of plasma and saliva chlorpromazine against time for the dysphoric (N=10), indifferent (N=4), and syntonic responders (N=18) did not reveal any obvious relationship between subjective response and either blood or saliva level.[1] The dysphoric responders did not have higher blood or saliva levels than syntonic or indifferent responders; the shape of the curve for all three groups was similar. Pearsonian correlations of subjective response at 4 and 24 hours with the respective plasma and saliva levels (whether raw or logarithmic) were not statistically significant.

Different cutting points for dysphoric, syntonic, and indifferent responders still did not yield significant differences. Further, even the most extreme dysphoric responders were not different in their plasma and saliva levels: for example, the patient with the highest plasma and saliva levels was semicomatose at the time of the peak levels; at 24 hours, his plasma level had fallen to 137 pmol/ml, at which time he was only mildly dysphoric. (He went on to have a good objective and subjective outcome on a very low dose of chlorpromazine).

Subjective response to a test dose of chlorpromazine ranged widely, from +36 through −36 (mean score±SD=4.8±16.6) at 4 hours and from +33 through −44 (mean score=4.9±16.9) at 24 hours. There were no relationships between subjective response and age, duration of psychotic symptoms, diagnosis (paranoid versus nonparanoid schizophrenia), peak drowsiness ratings (subjective or objective), or initial clinical state (1).

COMMENT

Whatever the reason for dysphoric responses to chlorpromazine, our data indicate that pharmacokinetics is not the answer. When the dysphoric responder tells us that the medication is too strong or that we are "poisoning" him, the explanation is not an aberrant plasma chlorpromazine level. Dysphoric responses to chlorpromazine appear to be idiosyncratic, rooted perhaps in altered receptor sensitivity or a patient's very personal interpretation of drug effect.

[1]A figure showing subjective response versus blood and saliva levels is available from Dr. Van Putten on request.

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Priorities in Psychiatric Residency Training

BY CHARLES L. BOWDEN, M.D., FREDERICK J. HUMPHREY, D.O., AND MICHAEL G.G. THOMPSON, M.D.

The authors surveyed 22 psychiatric educators to determine their views of the basic knowledge and skills that should be required for all psychiatric residents. A comprehensive document of educational objectives facilitated practical differentiation of responses. Diagnostic, problem-solving, and treatment-dispositional skills were particularly highly ranked. Such data may be useful in defining the profession of psychiatry as well as in curriculum planning.

The core curriculum for residency training is one of the critical issues now facing psychiatry. As Beigel (1) stated, this curriculum "must define concepts and areas of knowledge that all psychiatrists should possess regardless of the specific application(s) which they later choose for this knowledge and be subject to evaluation according to defined criteria." Benefits may result from consensually derived curriculum objectives, especially if these are ranked as to importance. Psychiatric educators and residents would have objective information on which to base curriculum planning and clinical experiences. Accountability might be improved. With general agreement about the content of psychiatric residency education, individual programs should be better able to establish expected levels of proficiency and to develop evaluation methods to document that proficiency. The curriculum task force of the American Association of Directors of Psychiatric Residency Training surveyed psychiatric educators to determine their views about the degree to which a broad range of objectives is essential in residency training. The results are reported in this paper.

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The objectives used for the survey are those developed by the Canadian Coordinators of Psychiatric Education, a listing of more than 900 objectives in 19 topic areas describing knowledge and clinical skills relevant to psychiatric education (2). One advantage of such detailed objectives for this survey was that important differences in emphasis within a subject area might become evident. For example, skills in psychopharmacology almost certainly would be ranked high by all psychiatrists. However, by subdividing the area of psychopharmacology into enabling objectives, such as "ability to state . . . the general chemical formulae for the class of drug" and "ability to state theoretical sites of action in the brain," objectives that most respondents considered less important, we can begin to establish limits for the core levels of knowledge and skills to be attained in various subject areas during residency training.

METHOD

The questionnaire, which took 4 to 10 hours to complete, was distributed to 36 psychiatrists, 22 of whom responded: 15 were psychiatric residency directors, 3 were undergraduate psychiatric education directors, 2 were members of the American Board of Psychiatry and Neurology, and 2 were chief residents. Twenty respondents were affiliated with medical-school-based training programs, and 2 were directors of state hospital programs. Four were from the northeast, 5 from the southeast, 8 from the midwest, 3 from the southwest, and 2 from the west.

For each objective the respondent indicated whether it should be considered essential for all residents, moderately emphasized, or touched upon/optional.

RESULTS

Table 1 lists those objectives considered essential by at least 50% of the respondents. Table 2 lists illustrative examples of objectives ranked as essential by less than 50% of the respondents. A complete list of the rankings for all 900 objectives is available from the first author.

TABLE 1
Residency Training Objectives Ranked Essential by at Least 50% of 22 Psychiatric Educators

Objective	Essential		Moderate Emphasis		Touched Upon/ Optional	
	Number	Percent	Number	Percent	Number	Percent
Knowledge of major trends in contemporary psychiatry	14	63.6	7	31.8	1	4.5
Ability to assess normality and deviance from normality	18	81.8	2	9.1	2	9.1
Knowledge of effect of illnesses and medications on sexual behavior	14	63.6	7	31.8	1	4.5
Ability to compare male and female life cycles regarding achievement, motivation, affiliation, and sexuality	18	81.8	4	18.2	0	
Knowledge of function of limbic system	19	86.4	3	13.6	0	
Knowledge of function of hypothalamus, pituitary, and adrenal cortex	15	68.2	7	31.8	0	
Knowledge of function of lobes of cerebral cortex	13	59.1	8	36.4	1	4.5
Knowledge of relevance of neuroendocrine function to psychiatric disorders	14	63.6	6	27.3	2	9.1
Knowledge of biological concomitants of stress reaction	14	63.6	6	27.3	2	9.1
Knowledge of biochemical theories of schizophrenia and affective disorders	20	90.9	2	9.1	0	
Knowledge of distribution and metabolism of neurotransmitters	16	72.7	5	22.7	1	4.5
Knowledge of genetic transmission of psychiatric disorders	13	59.1	8	36.4	1	4.5
Knowledge of indications for CT scan, lumbar puncture, etc.	15	68.2	7	31.8	0	
Knowledge of indications for biofeedback	13	59.1	7	31.8	2	9.1
Knowledge of effect of brain syndromes on intelligence	11	50.0	9	40.9	2	9.1
Ability to clinically distinguish schizophrenic language difficulties	14	63.6	2	9.1	6	27.3
Knowledge of relationship of psychiatric disorders to social class	13	59.1	8	36.4	1	4.5
Knowledge of developmental milestones of children	13	59.1	8	36.4	1	4.5
Knowledge of indications for common child psychiatric treatment techniques	15	68.2	4	18.2	3	13.6
Knowledge of developmental tasks of adolescence	21	95.5	1	4.5	0	
Knowledge of indications for adolescent psychiatric treatment	19	86.4	2	9.1	1	4.5
Ability to differentially diagnose mental retardation	16	72.7	4	18.2	2	9.1
Knowledge of genetic basis of psychiatric disorders	15	68.2	7	31.8	0	
Knowledge of family's role in patient's condition	17	77.3	4	18.2	1	4.5
Ability to present a comprehensive case history of all major diagnostic groups	20	90.9	1	4.5	1	4.5
Knowledge of basic psychoanalytic concepts	21	95.5	1	4.5	0	
Ability to outline the mental mechanisms commonly used in neuroses and psychoses	21	95.5	1	4.5	0	
Skill in psychiatric consultation	21	95.5	0		1	4.5
Ability to competently interview (including mental status exam), differentially diagnose, and establish treatment plan for all *DSM-III* disorders	22	100.0	0		0	
Ability to conduct complete physical exam	20	90.9	2	9.1	0	
Ability to use laboratory tests in diagnosis	19	86.4	3	13.6	0	
Ability to determine characterological coping patterns in patient under stress	21	95.5	1	4.5	0	
Ability to assess patient's resources, problem-solving ability, and flexibility of defenses	21	95.5	1	4.5	0	
Knowledge of main characteristics of crisis reactions	16	72.7	4	18.2	2	9.1
Ability to assess and manage psychiatric emergencies	22	100.0	0		0	
Knowledge of basic epidemiologic data on suicide	16	72.7	6	27.3	0	
Ability to assess and treat grief reactions	21	95.5	1	4.5	0	
Knowledge of biological correlates of psychophysiologic disorders	15	68.2	6	27.3	1	4.5
Knowledge of role of life stress and learning theory in psychophysiologic disorders	15	68.2	6	27.3	1	4.5
Ability to assess and treat psychophysiologic disorders	21	95.5	0		1	4.5
Ability to assess and treat psychosocial complications of physical illness	20	90.9	2	9.1	0	
Knowledge of psychological theories of neuroses	15	68.2	7	31.8	0	
Skill in treating neuroses	21	95.5	1	4.5	0	
Ability to describe drug treatment for personality disorders	20	90.9	2	9.1	0	
Ability to describe group treatment for personality disorders	19	86.4	3	13.6	0	
Ability to describe behavior therapy for personality disorders	16	72.7	6	27.3	0	
Knowledge of treatment for alcohol and drug abstinence syndromes	19	86.4	1	4.5	2	9.1
Knowledge of treatment approaches for sexual deviations	16	72.7	4	18.2	2	9.1
Knowledge of etiological hypotheses for functional psychoses	22	100.0	0		0	
Ability to treat functional psychoses	20	90.9	1	4.5	1	4.5

TABLE 1 (continued)
Residency Training Objectives Ranked Essential by at Least 50% of 22 Psychiatric Educators

Objective	Essential		Moderate Emphasis		Touched Upon/ Optional	
	Number	Percent	Number	Percent	Number	Percent
Ability to define and describe clinical features of borderline states	18	81.8	3	13.6	1	4.5
Ability to assess and manage postpartum psychoses	16	72.7	2	9.1	4	18.2
Knowledge of features of normal aging	21	95.5	1	4.5	0	
Knowledge of clinical features of organic brain syndromes	20	90.9	2	9.1	0	
Ability to make a thorough mental status, neurological, and psychological assessment of organic mental functioning	22	100.0	0		0	
Knowledge of classes and clinical features of psychotropic drugs	22	100.0	0		0	
Knowledge of chemical formulae for each class of drug	14	63.6	3	13.6	5	22.7
Ability to define the placebo response	18	81.8	4	18.2	0	
Knowledge of indications, efficacy, and risks of ECT	20	90.9	2	9.1	0	
Knowledge of classical and operant conditioning	15	68.2	6	27.3	1	4.5
Ability to assess appropriateness of behavior therapy for a patient	13	59.1	5	22.7	4	18.2
Psychodynamic understanding of psychotherapeutic relationship	22	100.0	0		0	
Ability to assess suitability of a patient for psychotherapy	21	95.5	1	4.5	0	
Skill in basic psychotherapeutic techniques	22	100.0	0		0	
Ability to recognize biases imposed on therapeutic relationship by sexual stereotyping	19	86.4	3	13.6	0	
Skilled execution of individual and supportive psychotherapy	21	95.5	1	4.5	0	
Knowledge of indications and limitations of group therapies	15	68.2	6	27.3	1	4.5
Ability to systematically assess a family	20	90.9	1	4.5	1	4.5
Skilled execution of family therapy	12	54.5	7	31.8	3	13.6
Ability to define primary, secondary, and tertiary prevention	20	90.9	2	9.1	0	
Ability to describe the team approach to psychiatric care	16	72.7	4	18.2	2	9.1
Ability to assess a geriatric patient	21	95.5	1	4.5	0	
Knowledge of special features of psychotropic drug use in the aged	20	90.9	1	4.5	1	4.5
Knowledge of legal requirements of fitness to stand trial	18	81.8	4	18.2	0	
Ability to assess competency	16	72.7	5	22.7	1	4.5
Working knowledge of commitment law and procedures	18	81.8	2	9.1	2	9.1
Ability to define common statistical and epidemiologic terms	12	54.5	4	18.2	6	27.3
Ability to critically analyze a research paper	12	54.5	7	31.8	3	13.6
Ability to critically evaluate one's own training	18	81.8	2	9.1	2	9.1

TABLE 2
Selected Residency Training Objectives Ranked Essential by Less than 50% of 22 Psychiatric Educators

Objective	Essential		Moderate Emphasis		Touched Upon/ Optional	
	Number	Percent	Number	Percent	Number	Percent
Knowledge of historical development of moral treatment of the insane	1	4.5	8	36.4	13	59.1
Clinical skill with sleep disorders	8	36.4	14	63.6	0	
Knowledge of frequency and type of language disorders in a grade school population	2	9.1	7	31.8	13	59.1
Knowledge of general systems theory	6	27.3	10	45.5	6	27.3
Knowledge of characteristics of nuclear and extended families	3	13.6	7	31.8	12	54.5
Therapeutic skill with the mentally retarded	2	9.1	7	31.8	13	59.1
Ability to discuss insulin therapy, sleep therapy, and vitamin therapy	3	13.6	4	18.2	15	68.2
Ability to conduct behavior therapy	8	36.4	8	36.4	6	27.3
Knowledge of indications and principles of Gestalt therapy	3	13.6	10	45.5	9	40.9
Ability to work as a therapist in a group	10	45.5	8	36.4	4	18.2
Experience in community consultation	9	40.9	7	31.8	6	27.3
Clinical experience in a geriatric setting	8	36.4	4	18.2	10	45.5

DISCUSSION

Highest emphasis was given to practical clinical objectives, such as being able to describe the clinical manifestations and clinical course, evaluation, differential diagnosis, and treatment of the major psychiatric disorders. Objectives focusing on the vicissitudes of normal development and phase-specific conflicts,

especially those during childhood, adolescence, and old age, were also given high rankings. In terms of treatment skills, emphasis was placed on individual psychotherapy and psychopharmacology. From a theoretical standpoint, psychodynamic objectives were given greater emphasis than learning theory and behavioral modification objectives. The latter, however, were emphasized more than less commonly used approaches, such as Gestalt and rational-emotive therapies. Child and adolescent psychiatry objectives were consistently ranked lower than similar adult objectives. This held true for skill objectives, such as differential diagnosis and treatment, as well as theoretical constructs. It suggests that the emphasis in basic training is considerably more on adult psychiatry than on general psychiatry. Much of the detailed information from biological, psychological, and sociological sciences was not considered to be essential. Several respondents indicated that many of these objectives were more suitable as precursory experiences.

Regardless of the content area, certain kinds of objectives were generally considered outside the essential experience of the resident. These included listing the contributions of famous people, detailed knowledge of less common treatment techniques, levels of mastery suitable for a psychiatric subspecialist, and mastery of theoretical information, especially if not objectively established.

The current study indicates those objectives which psychiatric educators consider essential. The respondents were a select group in several ways. The majority were directors of psychiatric residency training who were sufficiently motivated to devote several hours to the survey. Their knowledge and concern about curricular matters was probably above average. One might expect such a group to have unusually broad definitions of the scope of training. Their parsimonious view of what constitutes essential knowledge and skills is therefore noteworthy.

The small number of psychiatrists surveyed clearly marks this as a pilot study that would benefit from some form of replication with a larger and more diverse group of psychiatrists or psychiatric residents. The American Association of Directors of Psychiatric Residency Training is surveying residents completing training to determine how well their programs met these core objectives.

The data presented here can facilitate establishing a core curriculum in content and experiences. The rankings should aid in identifying discrepancies between what psychiatric educators consider important and the experiences actually provided residents. The data may also be useful in professional boundary sharpening—defining those things for which psychiatrists are uniquely qualified, areas of overlap with other medical specialties and mental health practitioners, and areas largely outside or peripheral to the basic competencies of psychiatrists.

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Should Psychic Studies Be Included in Psychiatric Education? An Opinion Survey

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The authors received 228 responses to a questionnaire on psychic phenomena mailed to professors and residents in psychiatry, other medical faculty, and deans of medical schools. Fifty-eight percent of the respondents believed that an understanding of psychic phenomena is important to future graduates of psychiatry, and 44% believed that psychic factors are important in the healing process. The authors believe that including psychic studies in psychiatric education would attract more medical students to psychiatry.

Meta psychiatry refers to the confluence of psychiatry and psychic phenomena. In addition to and including parapsychology, which deals primarily with ESP, telepathy, precognition, psychokinesis, reincarnation, and related fields, metapsychiatry includes many other paranormal manifestations that affect human behavior, e.g., altered states of consciousness, meditation and trance, out-of-body experiences, nonpsychotic hallucinations, postmortem survival, mind manipulation, charisma, voodooism, placebo effects, energy fields, and "Kirlian photography," cults, faith and the power of suggestion, voluntary control of autonomic functions (as originally practiced by esoteric sects and now confirmed by biofeedback techniques), and, above all, the scientific modus operandi of so-called miraculous healing. Already pointing in that direction are recent discoveries that endogenous histochemical mechanisms involving neurotransmitters, mood regulators, analgesics, and other reparative agents play an important role in the healing process, thus raising the strong possibility that exceptional emotional responses such as those associated with faith, suggestion, prayer, and placebo effects may greatly accelerate the speed and concentration of natural reparative biochemicals in somewhat the same manner as the well-known adrenal fight or flight mechanism.

Our own experience (1-3) and various surveys by others (4) have revealed an unprecedented degree of contemporary interest in psychic research among physicians and the general public. In his Presidential Address at the 1979 annual meeting of the American Psychiatric Association, Dr. Jules Masserman stated, "As to the third human proclivity for the arcane and occult, there is also a growing interest among us in what we call metapsychiatry" (5, p. 1015).

Unfortunately, little or no top level academic training in psychic studies has been available thus far; therefore, most educators are not aware of the added rich dimension such studies may provide in the therapeutic process. The purpose of this survey was to obtain an interprofessional consensus on the desirability of including psychic subjects in psychiatric and ancillary medical education. Our survey team consisted of a psychiatrist (S.R.D.), a specialist in family practice (C.O.P.) who was a former member of the Anglo-American Conference on Continuing Medical Education, and a marketing analyst (M.L.D.), who analyzed our computer-processed results.

METHOD

We used a national mail survey questionnaire to gather data because it was the most practical way to reach a broad-based, difficult-to-reach sample, was well suited to extensive rating scales, and assured anonymity to the respondents—a salient point in dealing with innovative subject matter (6). The questions were close-ended, with open-ended write-in options available for personal comments (7). Questionnaire completion time was minimized through the use of simple-to-record check-mark answers.

Questionnaires were mailed to 1,150 individuals: 293 deans of medical schools, 109 heads of departments of psychiatry, 261 professors of psychiatry, 419 residents in psychiatry, and 68 other medical faculty. Medical school deans and faculty members were mailed questionnaires directly. Psychiatric residents received

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them for the most part through secondary redistribution by heads of departments. That procedure for residents was necessitated by their relatively unspecific listings and may account for their low rate of response. The 1,150 questionnaires were distributed during September and October 1978. By December 20, 1978, we received 228 useable responses, representing a response rate of 19.8%. That figure compares favorably with reported average mail response rates (8). The response rate was highest among professors of psychiatry (31%), followed by deans (14%) and residents (12%).

RESULTS

Our questionnaire consisted of 6 divisions, which are summarized with their responses.[1]

1. Importance of psychic studies to future graduates. Fifty-eight percent of the respondents believed that an understanding of psychic phenomena is important to future graduates of psychiatry programs, compared to 22% who considered it unimportant. An additional 17% were neutral, and 4% were undecided. Opinion was mixed, however, regarding psychic studies in nonpsychiatric education, with the majority viewing that need as unimportant or remaining neutral.

2. Need for research on nonmedical factors in healing. More than half of the respondents (52%) believed that additional research is needed in this area, 35% were neutral, 4% were undecided, and 8% believed that additional research is not needed. In short, there was a strong consensus on such need, with no significant differences among sample groups.

3. Importance of specific healing factors. Nature's own reparative processes were rated as important factors in healing by 97% of the respondents; appropriate diagnosis was rated important by 96%. The patient's expectations, faith, and cooperation followed closely (94%), with specific medical-surgical intervention in fourth place, surprisingly (92%). Those leaders were followed by very high rates for the practitioner's therapeutic personality, 90%, and the power of suggestion, 82%. Another higher than expected percentage (44%) believed that psychic factors, i.e., some kind of unknown augmentation of the healing process, were important. Energy-field transfer from practitioner to patient was perceived as important by the smallest percent (11%). However, about half of the respondents were neutral or undecided about this factor, seemingly indicating ambivalence or some uncertainty about the meaning of the term "energy-field transfer."

4. Forms of psychic phenomena. Respondents were asked whether they believed that academic study of 12 specific psychic phenomena would be valuable. There was considerable variation in perceptions, possibly due in some cases to definitional uncertainty. Voluntary control over autonomic functions (biofeedback, etc.) and transcendental or religious meditation received the highest percentages of positive responses (39% and 21%, respectively). Mind control, psychic healing, psychokinesis, ESP, mysticism, nonpsychotic hallucinations, survival after death, and cosmic states of consciousness each received positive responses from 5% to 12% of the respondents. Reincarnation and energy fields received support from less than 5%. There was agreement among the respondent groups in all of the above areas except nonmedical healing, which, interestingly enough, professors of psychiatry accepted more than either psychiatric residents or medical school deans.

5. A proposed psychic aptitude test as a diagnostic instrument. Thirty percent believed such a test would be useful as a diagnostic aid, 43% felt it would be useless, and 27% were neutral or had no opinion.

6. Demographic prevalence of psychic phenomena. Relatively few respondents (15%) denied the existence of psychic abilities, latent or overt. Most (42%) acknowledged the possibility of their existence. Twenty-one percent believed that some people possess psychic abilities in some degree, and 14% believed all people possess them in some degree. The remainder were neutral. In addition, 35% had themselves or knew of someone who had had occult or paranormal experiences, chiefly ESP, telepathy, precognition, and altered states of consciousness.

DISCUSSION

Our survey was intended to serve two functions: providing directions for further research and indicating current attitudes about psychic phenomena in education. We believe that despite certain shortcomings in any questionnaire of this kind, the opinions of 228 working scientists, the majority of whom are academic administrators, have intrinsic value.

Our results indicate a high incidence of conviction among deans of medical schools and psychiatric educators that many psychic phenomena may be a reality, psychic powers are present in most or all of us, nonmedical factors play an important part in the healing process, and, above all, studies of psychic phenomena should be included in psychiatric education and, to a lesser extent, in other branches of medicine. The relatively high percentage of neutral and undecided opinions suggests that the attitudes of many are still open but cautious. This seems to indicate that their attitudes may become increasingly supportive as interest becomes more manifest—a hypothesis that awaits further research and trend analysis.

Would the inclusion of psychic studies add to the scope of psychiatry and attract more medical students

[1]Percentages are rounded off to the nearest whole number. Copies of the questionnaire and tables of the responses are available from the authors on request.

o the profession? We believe it would. In the past few ears there has been a substantial progressive drop in he percentage of medical students entering psychiary—a reduction of more than 12%. According to ardes, "The nature of the educational experience ofered the student is one of the major factors. . . . Admissions committees must recognize the importance of he *psychosocial, humanistic* side of medicine (italics dded). . . . Psychiatry has to elaborate more clearly n the precise role of psychiatrists vis-à-vis other medcal specialties by enlarging the armamentarium of sychiatric clinical approaches" (9). In short, our profession must expand its horizons to include important, resh dimensions. We submit that psychic studies are t least one such dimension and trust that our findings vill provide useful information to educational decision nakers in planning a comprehensive, contemporary ealth care curriculum in keeping with the times.

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Consultees' Representations of Consultants' Psychiatric Diagnoses

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In an attempt to characterize consultees' representations of consultants' psychiatric diagnoses, the authors reviewed the medical records from 190 psychiatric consultations in a general hospital. Using specific outcome criteria, they rated 50% of representations as concordant and 39% as nonconcordant. Five variables, including the primary medical diagnosis at discharge and the referring service, were significantly related to concordance. Surprisingly, concordance was independent of the psychiatric diagnosis, indicating that consultees did not respond differentially to varying magnitudes and implications of the different disorders. This work raises the question of possible strategies to improve receptivity to psychiatric diagnoses in the consultation setting.

Efforts to measure the effectiveness of psychiatric consultation in the medical-surgical hospital should address the responses of the consultee as initial arbiter of the consultant's perceptions. We have previously characterized consultees' responses to the recommendations of psychiatric consultants with regard to psychotropic drugs (1) and diagnostic actions (2). As a further step in the development of our Consultation-Liaison Outcome Evaluation System, which we designated CLOES, in the present study we examined consultees' representations of consultants' psychiatric diagnoses.

METHOD

We retrospectively examined consultations performed by the Adult Psychiatric Consultation-Liaison Service of the University of Minnesota Hospital during the period January–June 1977. The service provides psychiatric interventions, support, and diagnostic skills to the medical, neurological, and surgical inpatient services of the hospital. A member of the requesting service is contacted, if possible, before and after the initial patient evaluation. These contacts are intended to clarify the reasons for the request and subsequent impressions. Whenever possible, the patient is seen within 24 hours of submission of the request. After the initial interview a handwritten evaluation is placed in the progress notes of the medical chart. Although a specific format is not used, the resultant psychiatric diagnosis is usually placed at the end of the note. The written consultations were authored by the service's attending staff, third- or fourth-year residents, or others serving in an emergency capacity. The principal diagnostic nosology used by the service at the time of the study was *DSM-II*.

We reviewed the medical records of patients seen by the service during the study period. For each case we identified and recorded all psychiatric diagnoses offered by the consultant in the initial note. We then screened the discharge summary for the same hospitalization (dictated by a house officer and signed by the attending physician) for the consultee's representation of each psychiatric diagnosis. Each case was rated as follows: nonconcordant if no mention of the diagnosis was made; concordant if a verbatim or near verbatim representation of the consultant's diagnosis was found; and uncertain if the representation was not rated concordant or nonconcordant.

For each case rated uncertain, a second procedure followed. Each psychiatric diagnosis offered by the consultant and the corresponding representation by the consultee were presented independently to two psychiatrists who were not members of the service during the study interval. Each representation was rated according to the following criteria: concordant if it accurately conveyed the descriptive portion of the diagnosis, such as affective disorder or organic brain syndrome, and if it included modifying statements judged critical to understanding the severity or implications of the disorder; partially concordant if the descriptive portion was correct but a critical modifier was absent; and nonconcordant if the descriptive portion was inaccurate. In those cases in which the consultant offered more than one diagnosis, the case was given the rating of the least concordant representation. This procedure was repeated 72 hours later with an altered sequence of case presentations. Raters had not been informed of the repetitive design of the study.

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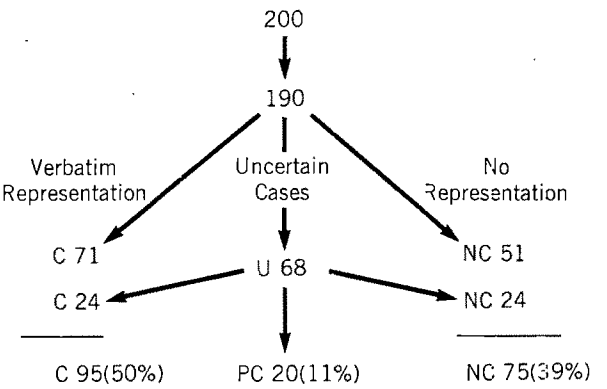
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TABLE 1
Significance of Variables in 190 Psychiatric Consultations[a]

Characteristic	p
Patient	
1. Number of previous hospitalizations at present facility (none, ≥1)[b]	.083
2. Number of problems on problem-oriented medical record (0-19)[b]	.290
3. Formal past psychiatric history (no, yes)[b]	.521
4. Current psychotropic drug usage (no, yes)[b]	.935
5. Primary medical diagnosis at discharge (functional, other)	.006
Consultee	
6. Referring service (general medicine, general surgery, neurology, others)[b]	.001
7. Written indication of psychiatric intervention in ensuing progress notes (no, yes)	.356
Consultant	
8. Consultant (N=9)[b]	.167
9. Consultant's status (staff, resident, student)[b]	.750
Consultation	
10. Days hospitalized at time consultation was performed (1-71)[b]	.004
11. Days hospitalized after consultation was performed (0-149)	.047
12. Total days hospitalized (1-151)	.003
13. Timing of the consultation (performed in the first half of the hospitalization or second half)	.160
14. Resultant psychiatric diagnosis (9 categories)[b]	.445
15. Resultant psychiatric diagnosis—collapsed grouping (organic brain syndrome, other)[b]	.116
16. Contact subsequent to initial consultation (no, yes)	.682
17. Presence of psychotropic drug recommendations (no, yes)[b]	.260
18. Concordance with psychotropic drug recommendations (nonconcordant, concordant)	.536
19. Presence of recommendations for diagnostic action (no, yes)[b]	.595
20. Concordance with recommendations for diagnostic action (nonconcordant, concordant)	.729

[a]By individual chi-square analyses or t tests.
[b]Prospective variable.

FIGURE 1
Final Concordance Assignments on 200 Psychiatric Consultations[a]



[a]C=concordant, U=uncertain, PC=partially concordant, and NC=nonconcordant.

Thus, a total of four ratings was available for each case initially classified as uncertain. Each rating was scored according to the following system: concordant=+1, partially concordant=0, and nonconcordant=−1. Final concordance assignments for each case were based on the composite score of the four ratings: 4 and 3=concordant; 2, 1, 0, −1, and −2=partially concordant; and −3 and −4=nonconcordant.

Consultees' concordance was then examined as a function of 20 variables that characterized the patient, consultee, consultant, or the consultation (see table 1). Individual chi-square analyses or t tests were used to identify those variables which were significantly ($p<.05$) related to concordance with consultants' psychiatric diagnoses.

RESULTS

Of the 200 consultations performed in the study interval, 190 (95%) were reviewed. Records for four cases had not been retrieved three months after they were requested and were excluded from the present analysis. Six cases that resulted in inpatient psychiatric transfers were also excluded. Of the remaining 190 consultations, 68 (36%) were initially rated uncertain. Forty-eight of these cases were subsequently assigned a concordant or nonconcordant rating. All four component ratings were identical in 39 of these cases. After concordance ratings were assigned to the uncertain cases, the overall results (N=190) yielded 95 cases of concordance (50%), 20 cases of partial concordance (11%), and 75 cases of nonconcordance (39%). Of the 75 cases of nonconcordance, 51 (68%) failed to include the psychiatric diagnosis in the discharge summary and 24 (32%) contained inaccurate representations (see figure 1). To highlight the differences between concordant and nonconcordant cases, the 20 partially concordant cases were eliminated. Thus, subsequent statistical analysis involved 170 cases of which 95 (56%) were rated concordant and 75 (44%) nonconcordant.

Of the 20 variables examined (see table 1), 5 were significantly related to concordance. They were primary medical diagnosis at discharge, consultee service, days hospitalized at time consultation was performed, days hospitalized after consultation was performed, and total days hospitalized. Concordance rates for these variables are detailed below. With respect to primary medical diagnosis at discharge, a concordance rate of 79% was observed when the consultee's diagnosis was functional, compared with 50% for all other diagnoses combined ($\chi^2=7.60$, $p=.006$). Concordance rates also differed widely according to referring service (i.e., 11% concordance in a general surgery group versus 63% in general medicine; $\chi^2=16.61$, $p=.001$). The concordant group averaged 3.9 fewer days hospitalized at time of consultation ($t=2.96$, $p=.004$), 3.3 fewer days after consultation ($t=2.00$, $p=.047$), and 7.2 fewer days in total ($t=3.00$, $p=.003$).

DISCUSSION

We assessed consultees' representations of consultants' psychiatric diagnoses in this study using discharge summaries from medical records. Written at the conclusion of the hospital sequence, the discharge summary recapitulates and offers perspective on the completed hospitalization. It sets forth the responsible physician's account of what has transpired, together with what has been determined or planned. Accordingly, the discharge summary may be expected to address such consultations as have been carried out. Such attention might ideally include the consultee's reflections on and integration of respective consultants' perceptions. Since diagnostics are pivotal in reasoned clinical management and disposition, the adequacy of consultees' diagnostic representations should constitute a critical outcome factor.

In only 50% of the discharge summaries reviewed were consultees' representations judged concordant with the psychiatric diagnoses made by consultants. Of those rated nonconcordant, two-thirds omitted the psychiatric diagnosis entirely and the remaining one-third provided inaccurate representations. Overall, more than one-quarter of the discharge summaries made no mention of the consultants' psychiatric diagnosis. These results, comparable to Froese's findings in a pediatric population (3), demonstrate that neither accurate consultee representation nor even inclusion of consultants' psychiatric diagnosis in discharge summaries can be taken for granted.

The surprisingly low overall concordance rate appears to involve two factors. First, the failure of consultees to include the psychiatric diagnosis in the discharge summary suggests that consultees perceive psychiatric diagnoses as having minimal usefulness or importance. Oversights aside, the omissions may also reflect disagreement, lack of comprehension, or a devaluation of the consultant's opinion. Second, when efforts at representation are made, the failure to depict accurately the consultants' diagnosis calls into question consultees' grasp of psychiatric nosology. Errors of omission occurred twice as often as inaccurate representations. Taken together, the omissions and inaccuracies reflect the wide latitude for improvement in consultant-consultee communication. This is especially so given our emphasis on dialogue with the consultee both before and after initial patient evaluation.

The examination of selected patient, consultee, consultant, and consultation characteristics (table 1) identified five variables that were significantly associated with consultees' diagnostic concordance. Among these was the consultee's primary medical diagnosis at the time of discharge. A concordance rate of 78.8% emerged for functional disorders (26 of 33 diagnoses), versus 50.4% for all primary medical diagnoses combined (69 of 137 diagnoses). This striking difference demonstrates a heightened receptivity to psychiatric diagnosis when the consultee has been apparently unable to identify or document pathophysiology. At such a juncture, it appears that psychiatric diagnosis suddenly achieves credibility, perhaps vindicating the absence of organic findings and confirming the consultee's diligence. Most distressingly, this sequence appears to indicate that consultees employ a functional-organic dichotomy in their conceptual and clinical approach to patients and tend to diagnose psychiatric disorder by exclusion.

A second variable found to be related to concordance with consultants' psychiatric diagnoses was the consultee's service (e.g., medical, surgical, neurology). Concordance rates differed strikingly by service, from a disturbing low of 11.1% in a general surgery group (2 of 18 diagnoses) to 63% in a general medicine group (34 of 54 diagnoses). These findings may be indicative of intrinsic differences in specialty training with respect to both psychiatric disorders and their implications. Most nonconcordant responses in the surgery group consisted of omissions rather than inaccuracies. One might argue that these omissions represent no more than a problem in documentation; however, it is more probable that they represent oversight or haste. In our studies of concordance with consultants' recommendations for psychotropic drugs (1, 4) and diagnostic actions (2), we did not find that responses differed by type of consultee service. This seems to imply that the response of the surgical consultees in this study is specific to psychiatric nosology and its representation, rather than embodying a more general response to actions of psychiatric consultants. It may be that the low concordance of the surgical group involved a question of the perceived relevance of diagnostic labeling when unaccompanied by clinical actions. Surgeons' lack of interest or their discomfort regarding psychiatric diagnosis merits further study.

The remaining three variables associated with concordance involved the patient's length of stay. Patients representing concordant cases averaged fewer days of hospitalization at the time of consultation than did patients representing nonconcordant cases. They also spent fewer days in the hospital after consultation and had fewer total days of hospitalization. These data suggest that the more quickly consultees perceive the need for psychiatric intercession, the more receptive they are to the resulting observations of the consultant. Having promptly recognized psychopathology, consultees may find psychiatric diagnoses more useful or tenable. Although this may represent another facet of the consultees' reliance on a functional-organic dichotomy, it cannot be said to involve psychiatric diagnosis by exclusion. Patients with lengthier post-consultation and total hospitalization periods may be at risk for omission of psychiatric diagnosis or inaccurate representation due to the time lag from consultation to discharge. It should be noted that our previous studies have found heightened concordance phe-

nomena when the consultation occurred early in the hospitalization.

Of the 20 variables we examined, 15 exhibited no significant relationship with concordance. These variables included the resultant psychiatric diagnosis itself, the patient's psychiatric history, the consultant's identity and status, and the presence of and concordance with other recommendations of the psychiatric consultants. It is possible that a larger sample might reveal relationships not appreciated in the present study. Factors not explored here, such as the clarity, uniformity, and brevity of the consultant's presentation, may prove to be important determinants of consultees' representations of psychiatric diagnoses. It should also be emphasized that this study did not concern itself with the accuracy of the consultants' diagnoses.

One finding that should be particularly emphasized is that consultees' concordance with consultants' diagnoses is not related to the resultant psychiatric diagnosis in question. This finding appears to point to a generalized lack of interest or discomfort regarding psychiatric nosology rather than toward differences in the perceived usefulness of specific diagnostic entities or differences in the presentation of diagnostic statements. The overall concordance rate of 50% in this study is comparable to that reported in an earlier study of concordance by case with consultants' recommendations for diagnostic action (2) but appears somewhat lower than the overall 68% concordance found with consultants' recommendations for psychotropic medications (1). This implies that consultees may be more concerned with management than with evaluation and assessment.

This work extends our quantitative description of consultees' response to psychiatric consultation and anticipates the development of specific consultation strategies incorporating or derived from quantitative outcome studies. For example, this might include major revisions or simplifications of our nosological schema. The results should be contrasted with consultees' representations of diagnoses made by consultants from other medical specialties. They demonstrate several major challenges confronting the psychiatric consultant in the general hospital and reflect the opportunity for marked improvement in the accomplishment of consultant task functions. The findings also speak to the hazards of assuming either adequacy of communications or shared conceptual frameworks between consultant and consultee. This may be particularly instructive with the advent of *DSM-III*.

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CLINICAL AND RESEARCH REPORTS

This section contains 1) new research findings, including preliminary data from pilot studies, either clinical or laboratory; 2) worthwhile replication studies; 3) case reports that describe a truly new syndrome or cast new light on established ones; and 4) case reports that indicate a new therapeutic procedure of potential value or call attention to adverse effects of drugs or previously unreported complications of therapeutic interventions. Program descriptions and literature reviews cannot be printed in this section. Criteria for format are listed in "Information for Contributors" in each issue; papers that do not adhere to these criteria will be returned to the author.

Effect of Psychiatric Intervention on Use of Antihemophilic Factor Concentrate

BY H. ALLEN HANDFORD, M.D., DENNIS CHARNEY, M.D., LARRY ACKERMAN, M.D., M. ELAINE EYSTER, M.D., AND EDWARD O. BIXLER, PH.D.

The development of antihemophilic factor (Factor VIII) freeze-dried concentrate, which can be infused by patients outside a hospital, has radically changed the care and treatment of hemophilic patients. With the advent of home treatment, however, the staffs at hemophilia centers who provide Factor VIII concentrate and monitor hemophilia treatment programs have observed that the amount of Factor VIII concentrate used varies widely among patients, even when the patients have comparable levels of severity of the disease and frequency of bleeding.

The potential effect of emotional status on a patient's use of Factor VIII concentrate is suggested by Agle and Mattson (1), who discussed the psychological component of hemophilia. They emphasize the importance of understanding the hemophilic patient's relationships with his family, home, work, and social environments, and they encourage psychiatric intervention when indicated. Similarly, LaBaw (2) reported the successful use of suggestibility and hypnosis in controlling the pain and bleeding of hemophilia.

The purpose of our study was to evaluate the effects of emotional status on a patient's use of Factor VIII concentrate by assessing the relationship between psychiatric intervention and a patient's use of Factor VIII concentrate over a given time period. In the following case report we describe an adolescent with severe hemophilia, and we compare the levels of Factor VIII concentrate he used during three 15-month periods of time—before, during, and after psychiatric intervention. We analyzed these data by using the Time Series Analysis originally described by Box and associates (3, 4). The model fitting and analysis were done with a computer program developed by Glass and associates (5). These methods have been found appropriate for statistical analysis of large quantities of data that relate to a single subject and have been gathered over time. For each 15-month period of time we also compared by month the number of bleeding episodes the patient experienced, his total number of Factor VIII concentrate infusions, and the number of days he was infused.

We found that this patient reduced his use of Factor VIII concentrate during and after the psychiatric treatment phases, which suggests that psychological factors affect Factor VIII concentrate use and that patients with severe hemophilia may benefit from psychiatric intervention.

Case Report

When first referred for psychiatric evaluation, the patient was a 15-year-old with severe classic hemophilia who had a Factor VIII level of less than 1% (normal range, 50%–150%).

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TABLE 1
Antihemophilic Factor (Factor VIII) Concentrate Use in a Hemophilic Adolescent Who Received Psychiatric Intervention

	Monthly Factor VIII Concentrate Determinations					
			Number of Infusions			
15-Month Periods	Total (U)	Corrected for Weight[a] (U/kg)	Total	For Hemorrhage	Days Infused	Number of Bleeding Episodes
Baseline	22,680	426.6	23.3	5.5	22.5	2.4
During intervention	15,008[b]	283.1[b]	17.3[b]	2.7	16.9[b]	1.5
After intervention	12,970[b]	229.5[b]	16.0[b]	2.4[c]	15.5[b]	1.4[c]

[a]Body weight factor was linearly interpolated between actual weighings.
[b]Significantly different from baseline, $p<.01$.
[c]Significantly different from baseline, $p<.05$.

Before age 10 he had often been confined to a wheelchair because of frequent and severe bleeding. After he was placed on regular treatment with Factor VIII concentrate at age 10, however, he became active in tennis and swimming. Our hematology staff noted that he was using an excessive amount of Factor VIII concentrate, presumably out of anxiety and fear of bleeding rather than simply for the bleeding episodes as they occurred. At this point he was referred for psychiatric evaluation and possible treatment.

A mental status examination revealed that the young man was anxious about possibly having to reduce his use of Factor VIII concentrate. He had pressure of speech but no thought disorder. His memory was good, and he was well oriented in all spheres. His thought content revealed some adolescent conflicts about dealing with parental anger, meeting girls, and coping with teachers. He denied being unusually worried about his bleeding, and some disorganization under stress was noted.

The patient's mother was concerned that less frequent Factor VIII concentrate treatment would jeopardize the nearly normal lifestyles the patient and his family had been able to develop. Her husband expressed similar concerns that less frequent infusions would result in their son having more frequent bleeding incidents. Psychiatric intervention was offered, and the young man and his family were fully cooperative. All sessions were conducted by the same psychiatrist, who remained blind to the patient's Factor VIII concentrate use. Other medical staff exerted no pressure on him or his family to reduce concentrate use.

During the 15-month period before psychiatric intervention the patient administered Factor VIII concentrate to himself 5–6 days each week; he used almost four times as much concentrate as did the average home-therapy patient in our hemophilia program. His baseline mean (±SEM) of Factor VIII concentrate use was 22,680±994 U/month. In this program state funds provide for the total cost of Factor VIII concentrate used by each patient, without regard to financial circumstances, and federal funds provide for psychiatric and other staff services.

The patient then received 15 months of psychiatric treatment. The treatment began with 7 family therapy sessions over a 3-month period with an emphasis on the development of communication skills and the open expression of feelings and problems among family members. His Factor VIII concentrate use for this period dropped to 19,367±1,755 U/month, a 15% reduction from baseline. During the next 4 months, one of his siblings was depressed and was seen in 14 individual psychotherapy sessions. The patient's mean Factor VIII concentrate use during this period was 14,796±895 U/month, a 35% decrease from baseline. Three more family therapy sessions were held during the next 2 months; the main theme was the young man's separation and independence from his family. His use of Factor VIII concentrate for this period increased slightly (5%) over the preceding 4 months to 15,897±10 U/month. Over the next 4-month period he participated in 5 adolescent group therapy sessions in which the main themes were dating and jobs. His mean Factor VIII concentrate use for this period was 13,364±848 U/month, a decrease of 41% from baseline. After 3 self-hypnosis sessions during the next 2 months, his psychiatric treatment was discontinued. His Factor VIII concentrate use for this final treatment period was 11,295±1,719 U/month, a decrease of 50% from the baseline mean.

Communication among family members improved during this 15-month treatment period. Feelings of optimism replaced those of depression in all of the family members. In addition, the patient learned to use self-hypnosis techniques at times of stress, such as before school examinations or when he sensed impending bleeding episodes. Subsequently, he made a successful college adjustment while living away from home.

During the 15 months following termination of psychiatric therapy, his use of Factor VIII concentrate rose slightly to a final mean figure of 12,970±621 U/month, for an overall reduction of 43% from the original baseline. As shown in table 1, throughout the study the patient showed a decline in the amount of Factor VIII concentrate he used per kilogram of body weight (corrected for weight gain), the number of hemorrhages he had, the number of infusions he used for hemorrhage and nonhemorrhage episodes, and the number of days he used infusions.

Discussion

Over the 15-month period of psychiatric treatment, this young man significantly reduced his excessive use of Factor VIII concentrate. This resulted in appreciable savings to the family and our program (approximately $10,885 after deducting psychiatric costs) without compromising his care and treatment or interfering with his activity level. Moreover, he maintained his reduction in Factor VIII concentrate use for 15 months following the end of psychiatric intervention.

Factors other than the effects of psychotherapy may have played a role in reducing this patient's Factor VIII concentrate use. We may have observed the Hawthorne effect, where any new therapy brings about change because of the novelty of the intervention as suggested by LaBaw (2) in his work with hemophilic patients. Also, the patient's desire to

please his therapist may have caused a reduction in his use of concentrate. It is our belief, however, that his continued low rate of Factor VIII concentrate use during the 15-month follow-up period substantiates the effectiveness of psychotherapy in reducing his use of Factor VIII concentrate.

We believe that as the patient's activity levels increased through the use of Factor VIII concentrate treatment, which he used in anticipation of bleeding rather than after its onset, he and his family began to rely on Factor VIII concentrate until he used the concentrate almost without limit to reduce his fear of bleeding episodes. When psychiatric intervention relieved this chronic stress in this patient and his family, he dramatically reduced his use of Factor VIII concentrate.

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Psychotic Reaction Caused by Proprietary Oral Diet Agents

BY CHARLES B. SCHAFFER, M.D., AND MARTHA WELTY PAULI

Self-medication with over-the-counter oral diet aids is increasing. Several of these diet aids contain phenylpropanolamine as the active ingredient. Phenylpropanolamine is available without a prescription in a variety of weight reduction formulations such as Dexatrim and Permathene. These anorexic agents contain as much as 75 mg of phenylpropanolamine per dosage unit; most contain 25–50 mg (1). The American Medical Association (2) reports that phenylpropanolamine is probably an ineffective anorexic agent. Phenylpropanolamine is also one of the most frequently used oral nasal decongestants, usually marketed in combination with an antihistaminic drug. Acute psychotic episodes in three patients following their use of phenylpropanolamine as an oral decongestant were reported by Kane and Greene (3); and Wharton (4) reported one case of paranoid psychosis related to the use of phenylpropanolamine as a nasal decongestant. To our knowledge there are no reports of psychotic reactions secondary to the use of phenylpropanolamine as an oral anorexic agent currently available in the literature. In the case report below we describe a woman who experienced a psychotic episode while taking oral diet aids.

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Case Report

Ms. A was 23 years old, married, and the mother of one child. She was brought into the psychiatric emergency service by her husband and sister because she had been taking too many "diet pills." About five days before her admission she began taking one or two pills a day to lose weight. She had purchased these pills over the counter at a local drug store. About three days before her admission she began taking copious amounts of these pills. During that time she began to sleep less and displayed a change in behavior that included making bizarre statements, feeling people were going to harm her, running out of her house without a specific destination, and allowing the maintenance of her house to decline.

According to her husband and sister Ms. A had no previous similar episodes or psychiatric history, and she had functioned well as a mother and housewife. She had no recent episode of depression nor a history of significant drug or alcohol usage. There was no positive family psychiatric history.

When Ms. A was brought to the hospital she was noted to be alert but disoriented and somewhat agitated. Her speech was sparse and her thoughts were disorganized. She made several inappropriate responses to questions. Her attention and concentration were impaired; her judgment and insight were poor. At times she giggled and at other times she was combative. She also made some fragmented paranoid statements. She was admitted to the hospital, and her family was asked to bring in the diet pills she had been taking. Her husband brought in a bottle of Permathene and a bottle of Dexatrim. He believed that his wife had been taking 3–5 pills a day from each bottle during the few days before her admission.

On the first day of hospitalization Ms. A remained dis-

organized and disoriented, and she displayed regressive, intrusive, hyperactive behavior requiring restraints. After 3 days of hospitalization her symptoms improved and she was considered by her husband to be her normal self. She was not given any daily medication during this period.

We ordered routine laboratory tests at the time of Ms. A's admission (electrolyte, BUN, creatine, calcium, phosphorus and glucose levels, complete blood count, and liver function tests); all of these were within normal limits. Ms. A did not cooperate for a physical examination when she was admitted, but she did cooperate later and there were no remarkable physical findings.

Discussion

The acute psychotic episode experienced by this patient was most likely related to the ingestion of more than the recommended amount of the oral anorexic agents Dexatrim and Permathene. Ms. A's psychiatric symptoms began shortly after her indiscreet self-medication, and her symptoms resolved quickly without treatment after these agents were discontinued. She lacked any history of primary psychiatric disorder that might have played a role in the production of her psychotic symptoms. The drug-induced psychosis secondary to amphetamine use is widely publicized and well known (5). However, there is much less awareness about similar, if less frequent, complications of the amphetamine-related compounds, the sympathomimetic amines. There have been two reports of psychosis caused by ingestion of the sympathomimetic amine ephedrine (6). Even though phenylpropanolamine is regarded as one of these agents with fewer central nervous system effects, it should be noted that it is not without hazard. A pharmacy reference warns that untoward central nervous system effects, such as nervousness, restlessness, insomnia, and headache, may occur with the use of phenylpropanolamine (7), but there are no warnings of possible psychotic reactions even with excessive usage. Likewise, there are no warnings on the packages of Dexatrim or Permathene of possible psychotic reactions with excess use.

Caffeine is also an ingredient in Permathene (75 mg phenylpropanolamine, 140 mg of caffeine) and Dexatrim (50 mg phenylpropanolamine, 200 mg of caffeine). It has been reported that a high intake of caffeine (caffeinism) can cause symptoms such as nervousness, irritability, tremulousness, and insomnia, but there have been no reports of psychotic symptoms resulting from an excess intake of caffeine (8), even when up to 5,000 mg/day is taken. It is therefore unlikely that caffeine played a role in our patient's psychotic episode.

The wide use of phenylpropanolamine as a proprietary decongestant for colds, hay fever, and sinusitis, plus its increasing use as an oral over-the-counter anorexic agent, indicates a need to increase our awareness of its possible serious psychiatric side effects, including psychotic reactions. Indeed, the risks of these anorexic agents as diet aids may outweigh their benefits because there is no reliable evidence that phenylpropanolamine can help obese patients achieve a long-term weight reduction (9).

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Elevated Platelet Monoamine Oxidase Activity in Patients with Nonendogenous Depression

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Platelet monoamine oxidase (MAO) activity has attracted psychiatric research because the enzyme's role in metabolizing neurotransmitter amines ties it into biochemical theories of psychopathology and psychotropic drug effect. Increase or decrease of MAO activity may lead to depletion or accumulation of brain amines; conversely, excess or deficiency of these amines from other causes may lead to inhibition or enhancement of enzyme activity. MAO activity in blood platelets has received most psychiatric study because we have easier access to this tissue than we have to the brain. The case for the relevance of platelet MAO assay to psychopathology rests mainly on empirical findings of altered activity in various psychiatric conditions.

The greatest amount of research has focused on patients with schizophrenia, in whom the majority of studies have found lower MAO activity than in normal control subjects (1). However, low MAO activity has been reported in several other disorders including bipolar manic-depressive illness (2, 3).

In contrast, higher than normal MAO activity has been reported mainly in depressive patients outside the bipolar group (2–8). However, patients with unipolar depression comprise a large, heterogeneous group, and which of these patients may show elevated enzyme activity remains a major question. Orsulak and associates (8) singled out a syndrome they named schizophrenia-related depressions, which may relate more closely to character disorder than to schizophrenia, although their diagnostic category certainly denotes a chronic disorder. Robinson and associates (5) found that patients with high-MAO depression were more symptomatic and less responsive to treatment than other patients with depression. Landowski (6) reported that the elevation of MAO activity in patients with unipolar depression related to intensity of anxiety. These few findings suggest that elevated MAO activity may occur within the group of depressed patients sometimes labelled atypical or nonendogenous. One of the few dichotomies to emerge consistently from studies attempting to classify depressed patients on the basis of clinical data (9) is the separation of endogenous depressed patients from nonendogenous depressed patients. Therefore, it seems important to investigate this classification in relation to the biological parameters of affective disorder.

Method

Blood specimens were obtained from 96 subjects: 52 outpatients who met the Research Diagnostic Criteria (RDC) (10) for major depressive episode, 34 hospital staff matched roughly for sex and age (their specimens were drawn and analyzed concurrently with those of the outpatients), and 10 hospitalized patients with schizophrenia who were also diagnosed according to RDC. All RDC diagnoses were made by two clinicians. All specimens were drawn in the morning; the subjects had not taken medications for at least 1 week. The outpatients were further subclassified according to RDC as patients with bipolar depression, patients with nonbipolar endogenous depression, or patients with nonbipolar nonendogenous depression.

Platelet MAO activity was assayed according to the following procedure. First, 20 cc of whole blood was drawn from the antecubital vein into a plastic syringe; it was then transferred to a plastic tube containing 1 cc of 3.8% sodium citrate as an anticoagulant. The specimen was centrifuged at 600 rpm for 15 minutes and at 20,000 *g* for 5 minutes to yield a platelet pellet. The pellet was washed with 2 cc of 0.9% sodium chloride and spun again at 20,000 *g* for 5 minutes. After 2–8 weeks of storage at −20° C, the pellet was sonically treated in 1 cc of 0.05 M phosphate buffer (pH, 7.0). About 300 μg of the resulting protein was incubated with ^{14}C-phenylethylamine with a final concentration of 5 μM; after 30 minutes the reaction was stopped with 0.1 cc of 6 N hydrochloric acid. The mixture was passed through a 4.0×0.6-cm column of Rexyn 101-Na^+ and washed with 2 cc of water. The eluate was combined with 10 cc of Aquasol and counted in a Beckman LS-335 liquid scintillation counter using automatic external standardization. Enzyme activity was expressed as nanomoles of the product formed per milligram of protein per 30 minutes.

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TABLE 1
Demographic Characteristics and Mean Platelet MAO Activity in 62 Patients with Schizophrenia or Depression and 34 Control Subjects

Diagnostic Group	Male to Female Ratio	Age (years)		MAO Activity (nmol/mg protein/30 minutes)					
				Men		Women		Total	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD
Schizophrenia (N=10)	1:1	36.7	15.2	0.42	0.33	0.52	0.20	0.47	0.26
Bipolar depression (N=10)	1:1	28.1	8.3	0.40	0.22	0.45	0.18	0.43	0.19
Endogenous depression (N=7)	0.75:1	36.7	14.8	0.29	0.18	0.56	0.33	0.44	0.24
Nonendogenous depression (N=35)	1.5:1	34.7	9.4	0.44	0.22	0.66	0.33	0.53	0.29
Control (N=34)	1.1:1	34.0	11.1	0.37	0.16	0.45	0.18	0.41	0.17

Results

Table 1 shows means and standard deviations of platelet MAO activities for the diagnostic groups, broken down by sex. In all cases, analysis of covariance was employed; sex, age, race, and diagnosis were treated as covariants when appropriate. Overall, women had a significantly higher enzyme activity than men, $F(1,91)=7.58$, $p<.007$. Women also showed higher mean activity than men within each diagnostic group, although this difference reached statistical significance only for patients with nonendogenous depression, $F(1,32)=9.52$, $p<.004$. For all subjects age showed a significant positive correlation with MAO activity ($r=0.24$, $p<.01$). We failed to show any consistent relationship between race and enzyme activity, $F(3,89)=0.42$, $p>.7$. In comparing the various groups, statistically significant differences emerged only between those patients with nonendogenous depression and the control subjects, with the depressed patients showing higher than normal MAO activity, $F(1,67)=5.50$, $p<.03$. Interestingly, this diagnostic difference held true for the women, $F(1,26)=8.71$, $p<.007$, but not for the men, $F(1,35)=0.82$, $p>.3$.

Discussion

Our results support the hypothesis that elevation of MAO activity may occur in the group of patients with nonendogenous depression. The limitation of this finding to women raises the possibility of a relationship between menstrual variations in depressive symptoms and MAO activity (4); however, we lack the data to confirm this. We note that our results are consistent with prior reports of higher enzyme activity in women than in men and of enzyme activity increasing with age (5). Although we failed to replicate the findings of other researchers who showed that patients with schizophrenia have lower enzyme activity, we note the small size of our schizophrenic patient group and the fact that we compared inpatients with schizophrenia to outpatients with other diagnoses.

Our finding of a biological alteration in patients with nonendogenous depression has special interest. This group of patients, normally associated with the less specifically defined group of neurotic depressed patients, has also been generally associated with psychological rather than biological etiology and treatment (9). Thus, patients with nonendogenous depression have received correspondingly less attention in biological research than patients with endogenous depression. Our findings call this tendency into question. Moreover, these findings raise the intriguing possibility that elevated MAO activity is related to the responsiveness of patients with nonendogenous depression to MAO-inhibiting antidepressant medications.

However, nonendogenous depression remains a category only fractionally smaller than unipolar depression as a whole. Also, the enzyme activity findings of our patients with nonendogenous depression overlapped considerably with those of all other diagnostic groups. In order to find a closer correlation between elevated MAO activity and depressed patient subtypes, we may need more phenomenological studies contrasting the clinical features of depressed patients with high versus low MAO levels.

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Neuroendocrine Dysfunction and Blood Levels of Tricyclic Antidepressants

BY JUAN RAMON DE LA FUENTE, M.D., AND ALAN H. ROSENBAUM, M.D.

There is increasing interest in developing scientific laboratory procedures to help psychiatrists document their clinical impressions and to make their therapeutic interventions more rational. Psychoendocrine strategies (1) and determination of plasma concentrations of psychotropic drugs (2) appear most promising.

Despite significant advances in classification and pharmacologic treatment of affective disorders, precise documentation of depression in adolescent psychiatric populations remains conspicuously absent (3). Traditional views of adolescent turmoil and phenomenologic features somewhat distinct from those of depression in adults seem to have contributed to this state of affairs (4). Adolescents with affective illness, like the subject of our case report, present an opportunity to test the clinical value of laboratory aids, particularly when such adolescents exhibit phenomenologic features that do not fit the usual pattern of a major affective disorder.

Case Report

Malcolm, a 16-year-old boy who was the product of a second marriage of both parents, came to our hospital with a 2-month history of abnormal behavior and changes in his mental functioning. He had been examined at a local hospital, where the diagnoses of catatonic schizophrenia, acute organic brain syndrome, and possible seizure disorder had been made. Malcolm had reportedly been in good health until the onset of his current illness, which was marked by rapid heartbeat, perspiration, and feelings of fear and anxiety. He had also begun to suffer from initial insomnia and subsequent confusion and inability to concentrate on his studies. Malcolm was first seen by his family physician and was given imipramine hydrochloride, 25 mg orally at bedtime, and flurazepam hydrochloride, 15 mg orally at bedtime. He improved slightly for 4 or 5 days, but then all of his symptoms recurred and in a more aggravated form. His parents noted that he was becoming more withdrawn, fearful, and overly concerned about irrelevant matters. While on a trip with his father, Malcolm ran away because he thought that he was being taken away from his mother forever. He was subsequently admitted to another psychiatric facility. The facility's records described Malcolm as paranoid, with auditory hallucinations and convulsions. However, the symptoms were vaguely described, and neither Malcolm nor his parents were able to confirm them. Nevertheless, Malcolm occasionally complained of headaches and experienced dysphagia and twitching of the cheeks. Treatment with phenytoin sodium, primidone, and a small dose of haloperidol produced little improvement.

Interviews with the parents revealed that they had both suffered from periods of depression in the past. Malcolm's mother had responded well to lithium carbonate. Malcolm had always looked younger than his peers and when this difference became more apparent in 10th grade, he was teased by his peers and had fewer friends.

At admission, Malcolm was sleepy and did look younger than his stated age. He had sparse facial hair, his facies were somewhat masked, and his eyelids drooped slightly. A neurological examination revealed no significant abnormalities. Malcolm was oriented to time, person, and place, his speech was soft, his affect was very bland, and motor behavior was decreased. His cognitive functions were adequate. His thought content was marked by self-reproach, self-depreciation, and some rather vague nihilistic ideas, but Malcolm did not elaborate delusionally on any of these. He admitted that something was wrong with him and that he was not behaving as he should. He denied perceptual abnormalities.

Results of laboratory studies with extensive endocrine testing and tomograms of the sella were normal or negative unless otherwise specified. Malcolm's bone age was determined to be 14 years, and his plasma testosterone level was 2.5 ng/dl (normal range, 300–1,200 ng/dl). Three sequential EEGs showed no evidence of seizure activity. Using Jiang and associates' technique (5), we determined plasma corticosteroid levels. At 8 a.m. and midnight these levels were abnormally elevated (48 μg/dl and 40 μg/dl, respectively). The next day, after a 2-mg dose of dexamethasone given orally at midnight, the level was still elevated and showed minimal, if any, suppression (see table 1).

We used comprehensive psychometric, neuropsychologic, and personality assessments, including the Wechsler Memory and Intelligence Scales, Bender Visual-Motor Gestalt, Auditory-Verbal Learning Test, KNOX Cube Test, Tactual Performance Test, Kinetic and Static Steadiness, Rorschach Protocol, and MMPI, to rule out an organic brain disorder. However, these test results were inconclusive regarding the nature of Malcolm's psychiatric disturbance. Our working diagnoses were primary affective disorder and benign delay of adolescence. After completing the workup, we initiated treatment with imipramine hydrochloride. Plasma levels of the tricyclic antidepressant, determined according to Biggs and associates' method (6), were measured approximately 2 weeks later and were close to therapeutic range. We expected such a finding because Malcolm had improved considerably and showed total suppression after a new dexamethasone suppression test on hospital day 19.

However, Malcolm gradually regressed after a weekend visit with his mother. We slightly increased his dosage of imipramine hydrochloride and, despite what various authors (7–9) would consider therapeutic blood levels, his deterioration advanced and his ability to maintain suppression after

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TABLE 1
Postdexamethasone Corticosteroid Levels in a Depressed Patient Treated Initially with Imipramine Hydrochloride and Then with ECT

Hospital Day	Plasma Corticosteroid Levels (ug/dl)		
	8 a.m.	4 p.m.	12 a.m.
2	48.0	12.0	35.0
19[a]	1.2	1.0	1.0
31[b]	1.8	9.2	16.0
44[c]	3.0	2.9	3.6

[a]Daily dose of imipramine was 75 mg; blood level of imipramine and desipramine, a metabolite of imipramine hydrochloride, was 174 ng/ml.
[b]Daily dose of imipramine was 85 mg; blood level of imipramine and desipramine was 264 ng/ml.
[c]After ECT.

another dexamethasone dose was impaired, showing an "early escape" from dexamethasone suppression on day 31. We discontinued imipramine hydrochloride on day 35.

Because of Malcolm's increasing withdrawal, recurrence of his ruminative self-depreciatory thoughts, and appearance of new dramatic symptoms (incontinence of feces and urine), we administered 7 sessions of ECT (series 7). Malcolm's improvement was as dramatic as his deterioration had been. He quickly became spontaneous and cheerful, even boisterous, engaging in some roughhousing. Before discharge, results of another dexamethasone suppression test showed that Malcolm had regained his ability to suppress.

Discussion

Malcolm's clinical presentation seemed to go beyond the border of a major affective disorder, as defined in *DSM-III*. However, he did have some psychiatric symptoms that, together with his family history of depression, an abnormal dexamethasone suppression test, and an initial response to tricyclic antidepressants, were suggestive of an affective disorder, albeit atypical.

The dexamethasone suppression test proved a valuable diagnostic aid in this case. Its ability to discriminate between depression and other psychiatric syndromes (mainly schizophrenia) is quite good (10) and thus could be of further benefit if used in larger samples of adolescents with confusing symptomatology.

An abnormal response to dexamethasone in a depressed patient should revert to normal when the patient's condition improves. This may be of additional value since the pharmacokinetic properties of tricyclic antidepressants have made interpretation of plasma concentration values within a therapeutic context very difficult. This test might be used to establish therapeutic dosages of antidepressants in nonsuppressor patients. Not all depressed patients are nonsuppressors and we are not suggesting routine monitoring by this means of all depressed patients; but for research purposes, this strategy might be worth pursuing.

In retrospective assessment of Malcolm's case, an accepted clinical practice would have been to increase the dosage of imipramine hydrochloride when Malcolm's condition deteriorated after his initial response to a modest dosage. However, when we considered the severity of the relapse, the length of the hospitalization, the increasing anxiety among the patient's relatives and the medical staff, and the presumably therapeutic blood levels of the medications, ECT seemed the most expedient approach. This case adduces further testimony that prediction of outcome on the basis of plasma concentration of tricyclic antidepressants is limited and that development of methods to identify high-plasma-concentration responders should receive priority in research.

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Erotomania: A Separate Entity

BY MARIE RUDDEN, M.D., MARGARET GILMORE, M.D., AND ALLEN FRANCES, M.D.

Erotomania has been described in two different ways: as a secondary symptom of paranoia or schizophrenia, or as a distinct atypical psychosis. This case suggests the existence of a primary form of erotomania that manifests itself as a rare atypical psychosis most often reported in women with a characteristically intermittent and more benign course than schizophrenia.

Case Report

Ms. A., a 30-year-old professional woman, happened to meet an attractive man at a party and was very disappointed when he did not call her. She soon became convinced he was using car horns to communicate indirectly with her, was following her, tapping her phone, broadcasting her thoughts over the television, and wiring her bed to excite her sexually and to monitor her masturbation. Her work performance declined, she talked to walls, and begged her coworkers to stop relaying his messages. Ms. A held these beliefs almost continuously for a year and then reluctantly entered psychotherapy. She did not respond to phenothiazines and angrily stopped seeing her psychiatrist when he failed to confirm her suspicions.

Ms. A then consulted another psychiatrist and displayed prominent suicidal wishes and Schneiderian first-rank delusions but no derailment, hallucinations, or affective disturbance. She refused hospitalization and, in an effort to avoid involuntary commitment, requested an opportunity to confront her tormentor. At the meeting the "persecutor" denied Ms. A's charges and Ms. A's symptoms dramatically disappeared. She expressed considerable insight and relief, abandoned her delusions, and apologized with dignity. She has continued in outpatient psychotherapy and now functions well professionally without medication. She periodically develops delusions about various men she meets and about her male therapist. However, she is able to correct these delusions and to continuously maintain reality testing for a month or more at a time. Even when delusional, Ms. A shows a normal range of affect, a surprising warmth, maturity, and sense of humor. She is still often delusional about her male psychiatrist, claiming that he is monitoring her thoughts or indirectly communicating with her. However, she calls this her "daydream" and distinguishes the "part of myself that believes it." Sometimes she really does believe these delusions, especially when she is not in the psychiatrist's office.

When Ms. A was 8 years old, her father died after a prolonged illness. Ms. A had been his favorite daughter and for two years she was unable to accept the reality of his death. Instead she believed he might suddenly communicate with her or appear on the street. Eventually she was able to experience a delayed mourning. It is of interest that several other erotomanic patients reported in the literature had also lost parent figures in early childhood.

Ms. A had had a very active fantasy life as a child, but had no imaginary companions or transitional objects. As a woman, she has been somewhat shy but has experienced satisfying friendships and heterosexual relationships. She does not abuse alcohol or drugs and she gives no indication of factitious illness or malingering.

Discussion

Hollender and Callahan (1) divide erotomania into two types: 1) a primary form distinguished by the sudden onset of a delusional belief that focuses on one object and becomes a chronic, fixed delusional system; and 2) a secondary form in which the delusion has a gradual onset superimposed on other symptoms of schizophrenic psychosis. The first type is considered a subset of paranoia; the second of paranoid schizophrenia. Patients with both types of erotomania fail to respond to psychotherapy. Doust and Christie (2) collected eight additional cases, most of which fell into one of the above categories. Several cases with sudden onset were thought to be related to specific organic or toxic precipitants, but virtually all persisted chronically and were unresponsive to treatment. None of the patients had an intermittent course like the one Ms. A experienced.

Seeman's classification (3) differs from Hollender and Callahan's and includes a category that closely fits Ms. A. Seeman divides her patients into two groups: 1) women with constant, fixed delusions in conjunction with other symptoms of severe psychiatric illness, e.g., schizophrenia or schizoaffective disorders; and 2) women with recurrent and short-lived delusions in the absence of other symptoms of schizophrenia. The latter group of three women followed a characteristic and unusual pattern: they developed delusions about men they had actually met (rather than strangers) and often were able to give up their delusions when confronted by these men. Like Ms. A, these patients quickly repeated the pattern with a different man. They led active heterosexual lives, tended to be cyclothymic, competed with men, were better integrated than schizophrenic patients, and were diagnosed as having borderline personality, hysterical psychosis, or bipolar affective illness. In a personal communication to us, Dr. Seeman noted that one woman, who developed insight in therapy and had abandoned her delusions, would experience a recurrence when Dr. Seeman was on va-

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cation and a male psychiatrist was covering her practice. When a female colleague provided coverage, the patient's delusions did not recur.

Raskin and Sullivan (4) described a woman who had been treated for affective illness and then developed a delusional system when her treatment ended. She believed her therapist was in love with her, monitored her activities, and tested her through the clinic staff. After three years of psychotherapy and phenothiazine treatment, she relinquished her delusions. Subsequently she occasionally retreated into erotic fantasies but was apparently able to test their reality. The authors concluded that erotomania can occur in women with varying levels of illness and ego functioning.

This brings us to the differential diagnosis of our patient. When first evaluated, Ms. A clearly met *DSM-III* criteria for schizophrenia, paranoid subtype, but her ability to correct the delusion with reality confrontation, and her normal affective range and interpersonal relatedness are not uncharacteristic of schizophrenia. Although her illness resembled paranoia, it did not meet the *DSM-III* criteria because of Ms. A's Schneiderian symptoms and because her delusions were not "unshakeable" and were more erotic than persecutory in content. Ms. A resembled patients with brief reactive (sometimes called hysterical) psychosis in the reactivity of her symptoms, heterosexual conflicts, and good premorbid functioning. However, the chronic course distinguished Ms. A's illness from brief reactive psychosis in which symptoms persist for no more than two weeks.

Ms. A's illness more closely resembles that of the erotomanic patients described by Seeman than any specific *DSM-III* category. She exhibits an intermittent delusional conviction of being sexually pursued by various men with whom she is acquainted. She had no affective disturbance and remains remarkably intact in other areas of personality functioning. The most unusual and specific aspect of her illness is her capacity to abandon delusions in response to reality confrontation. This suggests a particular deficit in her object relations and reality testing—a man must actually be present before Ms. A can test the reality of her perceptions about him. Investigators should examine the long-term efficacy of such reality confrontations. Only the collection of additional cases will determine to what extent this type of erotomania constitutes a rare, separate entity.

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Serial Cognitive Testing in Cancer Patients Receiving Chemotherapy

BY THOMAS E. OXMAN, M.D., AND PETER M. SILBERFARB, M.D.

Previous research by our Cancer Psychiatry Division (1, 2) has suggested the need for an investigation of mild cognitive impairment in patients with cancer. Mild cognitive impairment may go undetected, yet it may influence a patient's ability to perform routine daily functions and to comply with treatment protocols. It was reported in one of these studies (2) that of 50 oncology patients who were admitted consecutively, those patients receiving chemotherapy demonstrated a significantly lower level of cognitive functioning than did nonchemotherapy patients. This was true regardless of the primary site of the cancer, whether there were cerebral metastases, or which chemotherapeutic agents were used. Despite these significant results several confounding variables were not controlled in the study, and they deserve closer scrutiny, namely, the existence of previous cognitive impairment and the possible effect of prior or concurrent brain irradiation (3).

In the present study we controlled for the above variables by testing each patient's cognitive capacity before chemotherapy treatment was begun, and we also eliminated from the study all patients who demon-

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strated prior cognitive impairment. Also excluded from this study were patients who received radiation therapy. Serial cognitive testing was used, which made each patient his or her own control and allowed us to document a relationship between chemotherapy and cognitive impairment.

Method

As part of the cancer-psychiatry liaison service, patients admitted for chemotherapy to the oncology service of the Mary Hitchcock Memorial Hospital were seen routinely on rounds. From patients admitted consecutively we selected a group who demonstrated no obvious psychiatric illness or organic brain syndrome and asked them to participate in this study and to give informed consent. None of the patients had a history or symptoms of depression, mania, or schizophrenia using the criteria of Feighner and associates (4). We evaluated 20 patients; 3 patients were subsequently excluded because they received concurrent radiation therapy, 6 patients were excluded because they received follow-up care at distant locations, and 1 because he developed toxic septicemia immediately after chemotherapy.

The final study group consisted of 10 patients: 9 women and 1 man. The mean age was 52.8 years (range, 36-67 years). Eight of the patients had a high school education and the remaining two patients had some college education. Six of the women had adenocarcinoma of the breast; for the other 4 patients the primary sites of the disease were ovary (papillary adenocarcinoma), colon (adenocarcinoma), uterus (leiomyosarcoma), and prostate (adenocarcinoma). All patients had some evidence of metastatic disease, but the disease had not spread to the central nervous system. Five patients were having their first course of chemotherapy, and 5 were having repeat courses.

Eight of the 10 patients received intravenous doxarubicin with varying combinations of cyclophosphamide, cis-platinum, DTIG, and vincristine. One patient received cyclophosphamide, methotrexate, and fluorouracil and one patient received tamoxifen and bleomycin. Eight patients received haloperidol or perphenazine as antiemetics before and during chemotherapy (average neuroleptic dose, equivalent of 8 mg of haloperidol). Five patients received 100 mg of intravenous secobarbital before chemotherapy, and 5 patients received oral narcotics after the initial testing (average narcotic dose, equivalent of 17 mg of morphine per 24 hours).

Patients were tested for cognitive impairment before chemotherapy by the vocabulary, reading, and information subtests of the Wechsler Adult Intelligence Scale (5) and the Halstead-Reitan Battery (6). Four additional subtests of the Halstead-Reitan Battery (digit symbol, trails B, digit span forward, digit span backward) were used to determine the patients' sensitivity to mild cognitive impairment (6).

TABLE 1
Serial Cognitive Testing in 10 Cancer Patients Before and After Chemotherapy

Cognitive Test	Mean Raw Scores: Before Chemotherapy	After Chemotherapy: 24 hours	After Chemotherapy: 1 month	F[a]
Digit symbol	50.6	49.2	52.2	1.054
Digit span forward	6.1	6.3	6.3	.014
Digit span backward	4.3	4.0	4.4	1.83
Trails B	113.7	109.7	105.8	.153

[a]No analyses were significant.

Test results for all 10 patients before chemotherapy were within the norms expected for age and education, confirming the absence of significant cognitive impairment. The four subtests sensitive to mild cognitive impairment were repeated 24 hours after chemotherapy and 1 month after chemotherapy.

Results

Analysis of variance showed no significant difference on any of the subtests before chemotherapy, 24 hours after chemotherapy, or 1 month after chemotherapy (see table 1). These results were in agreement with clinical observations of these patients.

Discussion

A generalized interpretation of these results is limited because of the small number of patients in this sample and the possibility of a practice effect on serial testing (particularly on trails B). Nevertheless, over time the patients had nearly identical mean scores on each of the four subtests, which make the negative results in this population important.

In contrast to our earlier study most of the patients in this study were women and younger (mean age in the earlier study, 59.4 years; range, 22-86 years). These distinctions are important in view of the higher incidence of preexisting organic brain syndromes (primarily dementia) in older men (7). People with compromised cerebral function are more susceptible to delirium from further biological or psychological stress (8). The cognitive impairment found in patients in the earlier study may have been secondary to preexisting impairment or to an unmasking of subclinical impairment by chemotherapy. In addition, ectopic production of parathormone or vasopressin is known to occur in tumors, particularly lung cancers. These substances can produce delirium (9). Because 20% of the patients in the previous study had lung cancer, these hormones may have played a role in the higher occurrence of impairment found in that study.

The present study does not cast any doubt on the high occurrence of cognitive impairment in cancer patients nor on the fact that chemotherapy can cause cognitive impairment (3, 10). This study does suggest that factors other than chemotherapy are important in

causing cognitive impairment in cancer patients and that serial, rather than random, cognitive testing should be done to further elucidate these factors. Ideally, this testing should be done on patients who have the same cell type, stage and site of the disease, treatment, and premorbid cognitive status. In most cancer treatment centers stratifying a population to this extent will make it difficult to identify an adequate number of subjects. However, the multiinstitution cancer groups offer an opportunity to study the important problem of cognitive impairment in cancer patients in a controlled manner. Several studies of this type are currently being conducted in Cancer and Leukemia Group B, one of the multicenter study groups.

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Strategy for the Study of Patients at High Risk for Tardive Dyskinesia

BY JOHN KANE, M.D., FREDERIC A. STRUVE, PH.D., PAUL WEINHOLD, PH.D., AND MARGARET WOERNER, PH.D

Despite a considerable increase in research that is relevant to tardive dyskinesia, our understanding of etiological factors of the disorder remains limited. The epidemiologic data implicating neuroleptics is quite convincing; however, possible risk factors relating to drug history variables are still difficult to identify (1).

Age and sex have frequently been implicated in the development of tardive dyskinesia—older female patients have been shown in several retrospective analyses (2-4) to have increased risk. Surprisingly, dosage and length of neuroleptic exposure have been inconsistently related to the risk of tardive dyskinesia development (5). It may be that the lack of evidence for such intuitive relationships is merely a methodological problem given the difficulties involved in retrospective data analyses, particularly in dating the onset of the syndrome, and given the complexities of drug metabolism. An alternative hypothesis is that individual vulnerability or sensitivity may be the most important single factor in producing high risk.

Our previous work (6) suggested that electroencephalographic abnormalities, particularly the B-mitten pattern, may indicate increased risk for tardive dyskinesia. The B-mitten pattern, which can be detected only during moderately deep sleep, consists of a sharp transient wave (often formed by the last wave of a frontal spindle) followed by a higher voltage slow wave; the resulting complex resembles the thumb and hand of a mitten. These signals occur over the bilateral fronto-parietal region. The B-mitten pattern is rare among normal subjects, occurs primarily among psychiatric patients, and does not appear to be caused by neuroleptic medication because it also occurs in patients not receiving neuroleptics.

In order to address the problem of determining a patient's risk for developing tardive dyskinesia, we identified and studied a group of patients who demonstrated a high risk for the development of the syndrome.

Method

During a 3-year period we reviewed 99 consecutive case histories of patients with presumptive tardive dyskinesia and identified 7 patients who satisfied our definition of high risk. We arbitrarily defined a high-

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TABLE 1
Characteristics and Treatment Data for 7 Patients at High Risk for Tardive Dyskinesia

Patient	Age (years)	RDC Diagnosis	Drug Exposure (months)	Predominant Drug Class	Maximum Dose[a] (mg/day)	EEG Evaluation[b]	Tardive Dyskinesia Data	
							Severity	Follow-Up
1[c]	31	Bipolar II	9.0	Butyrophenone	1000	Normal (drug free); severe generalized and diffuse paroxysmal slowing (lithium)	Mild	Persistent after 15 months
2	15	Major depressive disorder, schizotypal features	7.0	Piperazine	2250	B-mitten dysrhythmia	Moderate	Remitted after 2 months
3	20	Other psychiatric disorder	5.5	Piperazine	80	B-mitten dysrhythmia	Severe, functionally impaired	Persistent for 2 years; neuroleptics withdrawn for 3 months
4	28	Other psychiatric disorder	4.0	Aliphatic	1200	Anterior brady-rhythmia	Moderate	Persistent after 9 months
5	59	Bipolar I	8.0	Piperazine	300	Normal[d]; no sleep recording	Moderately severe, functionally impaired	Persistent for 16 months; neuroleptics withdrawn for 10 months
6	47	Major depressive disorder, alcoholism	9.0	Piperazine	700	B-mitten dysrhythmia	Mild	Persistent after 6 months
7	29	Manic disorder	8.2	Butyrophenone	200	B-mitten dysrhythmia	Mild	Remitted after 1 month

[a]Maximum daily dose was received for a minimum of 1 month; doses in chlorpromazine equivalents.
[b]Evaluations made blind to tardive dyskinesia diagnosis.
[c]Only female patient.
[d]May be false negative for B-mitten dysrhythmia.

risk patient as anyone who developed presumptive tardive dyskinesia after less than 1-year cumulative lifetime exposure to neuroleptics.

Although no established criteria for the diagnosis of tardive dyskinesia have been developed, these patients had definite symptoms of at least mildly severe presumptive dyskinesias and several had moderate to severe symptoms. The diagnoses were made on a longitudinal rather than cross-sectional basis, and they were confirmed on several occasions by different examiners, including a neurologist. Two patients had dyskinesias of such severity that they were functionally impaired.

Results and Discussion

The characteristics of these patients are presented in table 1. Several findings should be noted: the patients' mean age was 33 years, 6 of the 7 patients were men, none met the Research Diagnostic Criteria (7) for schizophrenia, 6 of the 7 patients received predominantly high potency neuroleptics, and 4 patients received a maximum dosage for at least 1 month that was less than 700 mg/day of chlorpromazine equivalents.

The dyskinesias of 2 patients were reversed within 2 months after discontinuation of drugs; therefore, these patients might be considered in a different category. It would be difficult to consider these patients as having withdrawal dyskinesias because their dyskinesias emerged without reduction in or discontinuation of neuroleptic dosage. It is impossible to determine whether or not these patients would have developed persistent dyskinesias had their neuroleptics not been withdrawn following early signs of the syndrome. It is striking, however, that 4 of these patients have continued to manifest involuntary movements for at least 6 months following drug discontinuation; in three patients the movements continued to be at least of moderate severity, suggesting that these patients had considerable vulnerability to the disorder.

The most severely impaired patient received very low doses of neuroleptics, which suggests he was extremely vulnerable. He may have had some form of minimal brain dysfunction as a child; when he was 12 years old he had a brief exposure to chlorpromazine, but he had no history of psychostimulant exposure. It is conceivable that this history played a role in his apparent sensitivity to tardive dyskinesia.

EEG evaluations were made without any knowledge of a tardive dyskinesia diagnosis; 5 patients showed an abnormal recording and 4 patients had the B-mitten dysrhythmia (see table 1). The one patient who had no evidence of EEG abnormality had shown evidence of cognitive deterioration without gross neurological abnormalities. This suggests some degree of CNS dysfunction before the development of involuntary movements.

Possibly some degree of preexisting CNS dysrhythmia predisposes individuals to develop tardive dyskinesia. It is also noted that affective illness appears predominant in these patients. Although patients with all diagnoses are susceptible to dyskinesias, two anecdotal reports (8, 9) suggest that patients with affective illness may be at greater risk. The previously reported association of the B-mitten pattern with affective dysregulation is also of interest (10).

Two of these patients were 15 and 20 years of age. Because of their young age it is possible that they were exposed to neuroleptic medication in utero resulting in their unusual vulnerability. We questioned both patients' mothers, but we were unable to establish a history of neuroleptic use for antipsychotic purposes. However, antiemetic administration is difficult to rule out with certainty.

It is impossible to draw conclusions from this report except to suggest that persistent dyskinesias are not always tardive (that is, occurring late in exposure to neuroleptics) and that some patients appear exquisitely sensitive to this drug effect. The patients in this study may represent one subgroup of patients who develop tardive dyskinesia, and therefore, generalizations about the risk factors in this group cannot be made. Our findings are reported here for heuristic purposes; we are in the process of collecting data from a larger group of patients. We feel, however, that caution should be used when considering neuroleptics for conditions other than schizophrenia.

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Tardive Tourette Syndrome in an Autistic Patient After Long-Term Neuroleptic Administration

BY STEPHEN M. STAHL, M.D., PH.D.

Gilles de la Tourette syndrome is a hyperkinetic movement disorder of unknown cause with onset during childhood and characterized by multiple chronic motor and vocal tics (1). Tourette syndrome patients may also exhibit some rather remarkable language and behavior disorders such as coprolalia, echolalia, copropraxia, echopraxia, and compulsive, organized stereotypies such as touching, kissing, and jumping (1, 2).

A number of recent investigations point to a genetic and biological basis for idiopathic Tourette syndrome (3). Pharmacologic observations suggest that idiopathic Tourette syndrome may be characterized by a relative imbalance between striatal dopamine and acetylcholine with dopaminergic hyperactivity and cholinergic underactivity (4, 5). Tourette syndrome symptoms are exacerbated by dopamine agonists (such as amphetamine, methylphenidate, and L-dopa), relieved by dopamine receptor blockers (i.e., neuroleptics). Evidence for the role of a dopamine-acetylcholine imbalance in idiopathic Tourette syndrome is strengthened by observations that dopamine agonists can induce motor and vocal tics as well as stereotypies in patients who do not have Tourette syndrome (6). Furthermore,

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it now appears that Tourette syndrome can be induced in patients by long-term neuroleptic therapy; in that respect Tourette syndrome is analogous to tardive dyskinesia (7). In the case report below we describe a residual autistic adult who had an onset of Tourette syndrome after long-term neuroleptic administration.

Case Report

Mr. A, a 28-year-old man, came for an evaluation because he had uncontrollable motor and vocal tics that had lasted 2 years. He was the product of a full-term pregnancy with normal labor and low forceps delivery. Mr. A's growth and developmental milestones were normal up to 14 months of age, when his parents noted he made bizarre sounds and had paucity of speech and poor understanding of spoken language. By age 3 he avoided eye contact; developed stereotyped hand movements, flapping, and rocking; was fascinated by spinning objects; and would stand at the sink and run water through a funnel endlessly. When he was 5 years old, he could communicate simple thoughts with single words or short phrases in a staccato speech with pronomial reversal. He attended public school for 3 years, where his bizarre behavior, delayed language skills, and functional retardation were noted; his IQ was in the range of 45–55. At age 11 he entered a program for the mentally retarded. An examination at this time by numerous consultants revealed normal neurological findings with no abnormal movements except for the rocking motions of his trunk. An EEG revealed mild left temporal slowing. At age 13, he entered a state hospital because he failed to improve. He received neuroleptics (200 mg/day of chlorpromazine or thioridazine) continuously over the next 13 years; he also received diazepam or phenobarbitol on rare occasions for sleeplessness or agitation.

When Mr. A was 26 years old, he was returned home to his parents, and they requested that the thioridazine be discontinued. Approximately 2 to 4 weeks after the gradual tapering of thioridazine, he developed facial tics, grunting, and involuntary limb movements. A neurological evaluation at this time revealed the presence of frequent bilateral lower facial tics, sniffing, grunting, barking, occasional whispered coprolalia, and tic-like movements of the neck, torso, abdominal muscles, and diaphragm, the latter especially accompanying vocalizations. An EEG again showed Mr. A had left temporal slowing. A CT scan of the brain was normal. Phenytoin and primidone were prescribed for Mr. A, but he did not improve. Over the next 2 years his movement disorder persisted but was nonprogressive. Mr. A's tics were exacerbated by agitation or stress and relieved by sedation; he had little or no voluntary control over them.

A neurological examination of Mr. A at the age of 28 yielded findings that were unchanged from earlier neurological examinations. A comprehensive review of Mr. A's medical records, as well as psychological, speech and language, and psychiatric assessments showed that he met the *DSM-III* criteria for infantile autism, residual state. A brief trial of haloperidol completely suppressed his motor and vocal tics; d-amphetamine severely exacerbated these symptoms. Mr. A received an intravenous infusion of physostigmine salicylate, 4 mg over 60 minutes, after pretreatment with 6.25 mg of propantheline bromide i.v. An observer who was blind to Mr. A's treatment studied videotapes taken of Mr. A during this time and counted the number of tics he had; the observer noted that Mr. A's tics were completely suppressed for 6 hours starting 30 minutes after the infusion.

Discussion

Although the motor and vocal tics present in this patient are identical to those of idiopathic Tourette syndrome, several features of his case clearly distinguish it from classical Tourette syndrome. First, according to the criteria of *DSM-III* and Shapiro and associates (2) the age of onset (26 years) was atypically delayed. Second, the movement disorder was superimposed on the syndrome of residual infantile autism. Also, the onset of the syndrome in Mr. A occurred shortly after discontinuation of chronic neuroleptic treatment. Furthermore, Mr. A's tics were under no obvious control and fluctuated relatively little over time.

To our knowledge, this represents the first reported case of concurrent Tourette syndrome and infantile autism. Caine and associates (8) reported a patient with Tourette syndrome who was treated for a long time with haloperidol and who developed psychosis upon withdrawal of the medication. Klawans and associates (7) reported a patient with schizophrenia who was treated for a long time with chlorpromazine and who developed Tourette syndrome upon withdrawal of medication. Our patient thus appears to be the second reported case in which the manifestation of neuroleptic withdrawal dyskinesia has taken the form of Tourette syndrome. We have coined the term "tardive Tourette syndrome" to distinguish this particular syndrome from classical Tourette syndrome. We have also coined the term "toxic Tourette syndrome" to designate Tourette syndrome induced by dopamine agonists. Thus, we can now distinguish two unique forms of Tourette syndrome from classical Tourette syndrome, namely tardive Tourette syndrome and toxic Tourette syndrome.

Our observations thus have several important implications. 1) Tardive dyskinesia can take the form of Tourette syndrome as well as classical oro-buccal-lingual dyskinesias. 2) The pharmacology of tardive Tourette syndrome is identical to that of both tardive dyskinesia and classical Tourette syndrome, namely, exacerbation by d-amphetamine and amelioration by haloperidol and by physostigmine. 3) The pathophysiology of both Tourette syndrome and tardive dyskinesia can be characterized by dopamine-acetylcholine imbalance, with relative dopamine excess and relative acetylcholine deficiency. 4) The etiopathologies of tardive dyskinesia and Tourette syndrome might also be linked, so that data and hypotheses relevant to one syndrome may be applicable, by analogy, to the other. Thus, supersensitivity of postsynaptic dopamine receptors, possibly the cause of tardive dyskinesia (9), may also be important in Tourette syndrome. 5) Finally, clinicians should be aware of the possibility that the emergence of Tourette syndrome in a given patient might be secondary to withdrawal of neuroleptics and

that patients treated with chronic neuroleptics may be at risk for tardive Tourette syndrome as well as tardive dyskinesia.

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Psychogenic Oligodipsia with Hypernatremia in a Psychotic Patient

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Hypernatremia often causes lethargy, confusion, stupor, or coma and occasionally causes muscle irritability and convulsions. The common causes of hypernatremia include central nervous system lesions of the hypothalamus or supraoptico-hypophysial tracts, circumstances in which the patient has been unable to freely obtain water (this includes impairment of sensorium), or nephrogenic diabetes insipidus (1).

We report below a case of psychogenic oligodipsia with hypernatremia in which a psychotic patient drastically reduced his liquid intake apparently in response to psychological conflicts. The diametrically opposite syndrome, psychogenic polydipsia with hyponatremia, is well documented in psychotic patients (2, 3). As a result, it has been suggested that medical staffs monitor serum sodium levels routinely in psychotic patients when polydipsia is suspected (3, 4). An increase in gross neurobehavioral signs, such as agitation or confusion, or an increase in psychotic symptoms may reflect polydipsia (2).

The patient in this case was discovered when routine electrolytes were taken during his stay on a psychiatric unit; the tests indicated the patient had a serum sodium level of 178 mEq/liter (normal range, 135-145 mEq/liter). Surprisingly, the patient was alert and ambulatory, showing none of the above mentioned symptoms of hypernatremia. He refused to drink water and was transferred to the intensive care unit where intravenous fluids were initiated.

Case Report

Mr. A, a 58-year-old man, had the first of several psychiatric admissions 18 years ago; he has been continuously hospitalized for 8 years. The treatment team diagnosed him as having schizophrenia, chronic undifferentiated type. Mr. A had been treated for neurosyphilis, but subsequently he had a nonreactive serum and cerebral spinal fluid VDRL. There were no neurological stigmata of syphilis; tardive dyskinesia was the only neurological abnormality noted.

Mr. A had the appearance of a regressed psychiatric patient. He had difficulty communicating, rambling speech, and a short attention span; he was agitated, negative, and manneristic. Immediately before the discovery of Mr. A's dehydration it was noted that his usual level of agitation and hostility increased. No hallucinations or delusions were evident. He was taking 50 mg q.i.d. of hydroxyzine hydrochloride; thioridazine had been discontinued during the previous month.

The physical findings that were relevant in Mr. A's case included minimal dryness of the tongue and skin and normal blood pressure. He reported an absence of thirst. After three days of parenteral fluid therapy, his serum sodium level decreased from 178 to 145 mEq/liter. During this period, as Mr. A went from severe to mild hypernatremia, several urine osmolality determinations exceeded 600 mosmol/kg. These osmolality data reflect adequacy of the urine-concentrating mechanism and indicate normal secretion of antidiuretic hormone, normal kidney functioning, and the absence of any

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adverse drug effects. Thus, central nervous system lesions or nephrogenic diabetes insipidus were excluded as causes of the hypernatremia (5). Also, had he wished to do so, Mr. A was able to freely obtain water. No predisposing medical illness was found to account for his decrease in fluid intake, but there were obvious psychological reasons for his reluctance to take fluids.

Mr. A's self-induced dehydration was associated with a recurring psychological conflict. The first manifestation of this conflict had occurred 30 years earlier, when Mr. A had a hemorrhoidectomy. At that time he became preoccupied with bowel movements and accidentally defecated while riding in a bus. He was embarrassed and subsequently spent inordinate amounts of time in the bathroom for fear of fecal incontinence. At the peak of his disturbance, he remained in the bathroom for 2 years. He slept there, and his mother and wife brought him food. Mr. A's bizarre behavior persisted for more than a decade before his family brought him in for psychiatric treatment. In recent years Mr. A was spending much less time in the bathroom.

The conflict reemerged in a new form when Mr. A developed benign prostatic hypertrophy with urinary incontinence. The staff frequently found it necessary to ask him to change his clothing and to shower, and again he was quite embarrassed. His fear of urinary incontinence continued despite the fact that his control improved following prostatic surgery. He rushed to the bathroom to urinate, showered several times daily, and reduced his fluid intake. Even though he was severely dehydrated he reported an absence of thirst; he refused to drink anything except about two cups of coffee per day and ate dry cereal without adding any liquid. After the parenteral fluid therapy, Mr. A was eventually coaxed into drinking about two more cups of coffee per day. He still refused to drink any other liquid but willingly ate jello (seemingly unaware of its high water content). Thus, Mr. A's serum sodium ranged from 138–147 mEq/liter during the next six months.

Discussion

We have reported a case of severe hypernatremia in which a psychotic man drastically reduced his fluid intake. No medical conditions were found to account for his reduction of fluid intake, but it was associated with a recurring psychological conflict. Dehydration is a common problem in psychiatric patients who are stuporous (e.g., in catatonic states), but our patient was alert, ambulatory, and had no eating problems. To our knowledge this is the first reported case of psychogenic oligodipsia, a syndrome diametrically opposite to the frequently reported psychogenic polydipsia. Drinking is one of our most primitive drives, and indeed, only in a severely disturbed person could this powerful drive be thwarted. The prevalence of psychogenic oligodipsia is a matter of speculation. In view of the possibilities of morbidity and mortality associated with hypernatremia and hyponatremia (1), we suggest monitoring serum sodium levels in all psychotic patients to identify those patients who may have oligodipsia or polydipsia.

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Misidentification of a City: Delimiting Criteria for Capgras Syndrome

BY MARIE INGRAM THOMPSON, M.D., KENNETH R. SILK, M.D., AND GERALD L. HOVER, PH.D.

In 1923 Capgras described a symptom of delusional misidentification wherein the patient believes that a familiar, emotionally charged other person has been replaced by a duplicate or double of that person (1). The patient appears to retain recognition of the significant other person's external appearance but then delusionally denies the other's identity, claiming the other person has been duplicated by an imposter. This misidentification classically occurs with a clear sensorium (2).

The Capgras delusion is usually limited to those persons closest to the patient (a spouse, child and/or parent). Often there is some explanation for the disappearance of the real person (kidnapped, imprisoned, murdered). The imposter is frequently endowed with persecutory, sexual, or hostile intentions toward the patient (3).

Attempts have been made to explain the Capgras syndrome from either psychodynamic or neurologic

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perspectives. Psychodynamic theory states that Capgras is a mental mechanism wherein the patient is unable to repress intense ambivalence toward a love object (2, 4). The patient expresses this ambivalence in a psychotic manner, splitting the object representation into good and bad and concretizing the bad in the form of the imposter (2). Neurologic hypotheses have usually equated Capgras with prosopagnosia, i.e., the inability to recognize faces (5–7). It is postulated that an anatomic defect in striate and parastriate areas of the cortex, which may possibly involve portions of temporal, parietal, and/or occipital lobes, causes the nonrecognition.

The case presented here is an unusual variant of Capgras syndrome. The patient maintained that there were at least eight duplicates of his wife and children, each duplicate living in a separate duplicate city with a double of the patient. A literature review (3, 8, 9) failed to uncover a similar case, although Forth and Theofilopolous (10) recently described a case wherein inanimate objects were replaced by duplicates.

Case Report

Mr. A, a 32-year-old unemployed man, was admitted with a diagnosis of schizophrenia (*DSM-III*). He was legally separated from his wife and four children and lived alone. His parents had been divorced when he was 17 years old. He had one younger sister.

At 20 years of age, he joined the service. After 7 months Mr. A was discharged with the diagnosis of schizophrenia, paranoid type. He had repeatedly experienced auditory hallucinations commenting on his activities. He believed his feelings and thoughts were controlled by the same force that governed the voices. In the decade since his service discharge Mr. A had been hospitalized approximately 20 times for periods of 1 to 3 months. Auditory hallucinations and exacerbation of paranoid ideation with accompanying delusions of control were the usual precipitants of his hospitalizations. Mr. A improved markedly after he began receiving antipsychotic medication; however, he would discontinue the medication when he was discharged from the hospital.

Despite remission of other symptoms, Mr. A had a persistent delusional misidentification that began with the onset of his psychosis. After returning home from the service he believed he had arrived at a city that appeared to be an exact replica of his hometown, but he knew it was an "imposter-city." He first thought the imposter-city was located in Russia, but later he acknowledged that some imposter-cities were in the United States. He stated that there were eight imposter-cities and that he had spent the last 8 years wandering between them without finding the real one. (His wife reported that he always returned to his hometown between hospitalizations.) Each imposter-city had duplicates of his family members. He knew they were imposters because they did not "feel" like the real people to him. He believed that in his real hometown his real family was deceived by an imposter of himself who replaced him in his absence.

Two years ago Mr. A claimed he found his way back to the real city and rediscovered his wife. He cannot explain what happened to the imposter of himself or to the other imposter-cities and families. He says that they must exist somewhere. These ideas persist despite continued exacerbations and remissions of his illness over the last 2 years.

Mr. A had no history of substance abuse, head trauma, or loss of consciousness. His medical history was negative, and his family history was negative for psychiatric or neurologic disorders. The findings from a neurologic consultation, including an EEG and CT scan, were essentially within normal limits. His IQ was 111 (verbal, 121; performance, 97). Neuropsychological testing administered after Mr. A felt that he was in his real home revealed he had difficulty with tasks requiring figure drawing and picture sequencing. He was able to retain visual images.

Discussion

The multiplicity of doubles, as well as the extension of doubles to inanimate objects (cities), makes this case an unusual variant of Capgras. However, the case is typical of Capgras because Mr. A recognized who the people and city were supposed to be but delusionally misidentified them as being clever copies of the actual objects. Therefore, despite Mr. A's perceptual difficulties on neuropsychological testing, his pathology lay not in an error of recognition but in the formation of a delusion.

This recognition of who the object is supposed to be helps to delimit Capgras from neurologic disorders. Prosopagnosia, the nonrecognition of faces, and the broader concept of agnosia in general involve loss of the ability to recognize the typography (i.e., the surface physical characteristics) of a face or object and are true *an*identifications. Similarly, patients may suffer from anomia, the loss of ability to name a familiar object, or from aphasias in general. These disorders, singly or in combination, may be accompanied by random confabulation.

The above disorders stand in clear contrast to the psychosis seen in Capgras. The Capgras patient recognizes the typography of the object and knows who or what it is supposed to be. The patient, however, denies the object's reality and then delusionally misidentifies it as an imposter or duplicate.

We suggest that the pathology of Capgras lies not in a perceptual error but in the patient's disordered response to a clearly recognized object. Limiting the diagnosis of Capgras syndrome to cases that involve the elements of 1) recognition, 2) naming, 3) denial, and 4) delusional misidentification would distinguish it as a functional disorder, clearly differentiated from the neurological problems of naming and perception.

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Tardive Dyskinesia and Body Weight

BY JAMES M. SMITH, PH.D., DANIEL D. DUNN, M.A., AND MARY P. BURKE, M.A.

Although a considerable amount of research on the manifestations and treatment of tardive dyskinesia has been conducted during the past two decades, it is only recently that systematic attention has been given to other deficits that may be associated with this disorder (1, 2). Among these deficits, several authors have noted a weight loss in some patients with tardive dyskinesia. Gardos and associates (3) reported significant negative correlations between tardive dyskinesia ratings and body weight in two patients studied longitudinally. In both patients the highest correlations with weight loss involved neck and trunk movements, suggesting that these movements led to increased energy expenditure with an almost immediate weight loss. Furthermore, it was their visual impression that there was a preponderance of thin tardive dyskinetic patients. Sovner (4) also indicated that weight loss secondary to the constant movement of tardive dyskinesia may be one of the complications of the disorder. Weight loss associated with Huntington's chorea has been explained similarly (5).

Even though these authors suggest that weight loss is secondary to the constant movement, there is also the possibility that a more central mechanism may be involved. Animal studies have implicated brain catecholamines in the regulation of food and water intake, and some clinical data suggest the involvement of dopaminergic systems in particular. Rosenberg and associates (6) reported that Parkinson's disease, which involves a diminution of dopaminergic activity, is occasionally associated with an abnormally increased sensation of hunger and an insatiable appetite. In addition neuroleptic drugs, which decrease dopaminergic activity, frequently result in weight gain (7). On the other hand, drugs that increase dopaminergic activity, e.g., dextroamphetamine, have anorectic effects with subsequent weight loss without appreciable increases in movement. One hypothesis is that tardive dyskinesia results from a neuroleptic-induced dopaminergic hyperactivity in the nigrostriatal tract, therefore, it is conceivable that a parallel hyperactivity may be evidenced in other dopamine tracts in the brain as well. One indication of this would be the development of anorexia and subsequent weight loss. This weight loss would be typical of tardive dyskinesia in its myriad forms and not merely secondary to severe gross body movements.

To our knowledge there has been no large-scale study of this topic. In addition to being of direct clinical interest, the demonstration of dopaminergic parallelism would lend indirect support to the notion that there may be a similar parallelism leading to a supersensitivity psychosis (2). In order to study the relationship between weight loss and tardive dyskinesia, we examined patient data from a previous study on tardive dyskinesia (8).

Method and Results

In a study to determine the prevalence of tardive dyskinesia among inpatients at the Harlem Valley Psychiatric Center, Smith and associates (8) obtained informed consent from 377 of the center's inpatients. Two examiners rated these patients on the Abnormal Involuntary Movement Scale (AIMS), which was designed by the National Institute of Mental Health to measure the presence and severity of abnormal movements in seven body areas. Of the 377 patients rated, 293 had a primary or secondary diagnosis of schizophrenia at some time during their hospitalization; 30% of these 293 patients had at least moderately severe

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TABLE 1
Weight Differences Between Patients With Tardive Dyskinesia and Those Without

Measurement	Patients Under 60 Years of Age				Patients 60 Years of Age or Older			
	With Tardive Dyskinesia[a]		Without Tardive Dyskinesia		With Tardive Dyskinesia[a]		Without Tardive Dyskinesia	
	N	Mean Weight (kg)	N	Mean Weight (kg)	N	Mean Weight (kg)	N	Mean Weight (kg)
Initial study								
Men (N=129)	20		40		31		38	
AIMS weight[b]		72.85		74.59		63.55		66.54
Predicted weight		78.33		76.92		72.57		73.43
AIMS weight minus predicted weight		−5.48		−2.32		−9.02		−6.85
Women (N=124)	18		32		48		26	
AIMS weight[b]		67.39		74.69		64.31		64.31
Predicted weight		67.10		68.02		66.06		66.02
AIMS weight minus predicted weight		0.28		6.67		−1.74		−1.71
Follow-up at 2 years								
Men (N=87)	16		28		22		21	
Current weight		70.67		77.00		65.61		66.76
Current weight minus AIMS weight		−1.65		0.42		+0.87		+0.45
Current weight minus predicted weight		−6.50		0.25		−7.25		−6.57
Women (N=81)	12		24		33		12	
Current weight		68.61		72.14		62.26		59.50
Current weight minus AIMS weight		−4.01		−2.64		−3.31		−5.03
Current weight minus predicted weight		0.42		4.06		−3.88		−5.63

[a]Patients had an average rating on the Abnormal Involuntary Movement Scale (AIMS) of at least 2.5 on at least one of the seven body-area items.
[b]Weight data recorded at time of AIMS evaluation.

symptoms of tardive dyskinesia. Details on AIMS and the examination procedures employed were reported in that study.

Our follow-up study was conducted 2 years later. Of the 293 patients in the original sample who were diagnosed as suffering from schizophrenia, 168 patients remained as inpatients, 97 patients were discharged, 20 patients died, and 8 patients were transferred to other institutions. By means of a retrospective chart review of the inpatients and discharged patients, we obtained the weight of each patient at the time nearest the date of the patient's AIMS examination. This information was available for all of the 168 current inpatients and for 85 of the 97 discharged patients, yielding a total sample of 253 patients (129 men and 124 women). Predicted weights were computed for each of these patients based on sex, age, and height according to the regression equations presented by the National Center for Health Statistics (9). In addition, for the sample of 168 inpatients (87 men and 81 women) current weights were obtained, that is, the patient's weight nearest the time of this follow-up. Finally, weight difference scores (raw scores as well as percent difference scores) were computed. We calculated the differences between 1) weight at the time of the AIMS evaluation and predicted weight for all 253 patients, 2) current weight and weight at the time of the AIMS evaluation for the 168 inpatients, and 3) current weight and predicted weight for the 168 inpatients.

Because age and sex have been shown to be related to dyskinesia, separate two-way analyses of variance (dyskinesia by age group) were conducted for each of the sexes. The age variable was dichotomized at age 60 (<60, ≥ 60) and the presence of dyskinesia was defined as an average rating of 2.5 or higher in any one or more of the AIMS body symptom areas. The latter criterion has been used previously (10). These analyses were performed on the AIMS weight of the patients, their predicted weights, current weights, and the three weight difference scores (both as raw scores and percent difference scores). Some of these data are presented in table 1. Although the mean differences for the younger group are in the predicted direction, in none of these analyses was dyskinesia or the interaction of dyskinesia with age statistically significant.

Because the analyses of variance took account only of the presence or absence of dyskinesia, it also seemed useful to determine whether there was a relationship between severity of dyskinesia and weight. We obtained this information by computing the correlations between AIMS item scores and AIMS weight, current weight, predicted weight, and the three difference scores (both raw scores and percent difference scores). In addition to the AIMS item scores AIMS total scores (sum of the first seven items), AIMS face scores (sum of the first four items), and AIMS extremity/trunk scores (sum of items 5, 6, and 7) were included in the correlational analyses. The latter two sub-

scores (AIMS face score and AIMS extremity/trunk score) were utilized to determine whether a weight effect might be associated more with severity of gross body movements. To eliminate age and sex as confounding factors in the correlational analyses, separate correlation matrices were computed for younger and older men and younger and older women; the age groups were dichotomized at age 60 as in the previous analyses. There were few statistically significant relationships, and because the relationships presented no consistent pattern they could easily be attributed to sampling fluctuations. Of particular interest is the fact that the AIMS extremity/trunk scores did not show higher correlations with weight than did AIMS face scores.

Discussion

These results indicate that tardive dyskinesia is not associated with a general and characteristic weight loss. In interpreting this finding one should bear in mind several factors. First, this study was conducted in an inpatient setting where weight is regularly monitored. Presumably, if a patient varied appreciably from his normal weight, attempts would be made to correct this deviation and this would attenuate weight changes associated with tardive dyskinesia. Furthermore, in older patients it is easy to see how any weight change induced along with tardive dyskinesia could be obscured by the overriding effects of physical illness on weight. One would not, of course, expect this factor to apply to young patients to any great extent because young patients are not as likely to suffer from serious physical illness. Finally, this was a cross-sectional study, and no conclusions can be drawn about the weight changes that might be evident only at the time of the initial development of tardive dyskinesia and that might show some adaptation. In our sample, especially in the older patients, undoubtedly many developed tardive dyskinesia a number of years before the AIMS ratings.

These considerations notwithstanding, the results of this study demonstrate that there are no characteristic and persistent weight changes associated with tardive dyskinesia of sufficient magnitude to offset other factors that influence body weight. Although Gardos and associates (3) presented longitudinal data on two patients showing a relationship between the exacerbation of gross body movements and weight loss, the results of the present cross-sectional study indicate that weight loss is not typical, even in patients with severe movements. Several of the patients in the sample had severe gross body movements, yet they showed little or no weight loss. This finding also suggests that the weight loss in Huntington's chorea may not necessarily be secondary to the choreic movements, as is commonly assumed.

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Antimanic Effect of Clonidine

BY ROLAND JOUVENT, YVES LECRUBIER, ALAIN J. PUECH, PIERRE SIMON, AND DANIEL WIDLÖCHER

The role of norepinephrine in affective disorders remains the subject of extensive discussion and research. We have undertaken a systematic investigation of the effects of central stimulating or blocking agents of noradrenergic receptors in this context (1, 2). We hypothesized that the use of a stimulant of α-adrenergic presynaptic receptors, which reduces the release of norepinephrine (3), might be effective in the treatment of manic states. We decided to explore clonidine because its central effects have received some support. For example, clonidine has sedative effects (4), a favorable effect on the withdrawal syndrome in drug dependence (5, 6), and the side effect of inducing a depressive syndrome when prescribed for hypertension (4).

Method

We studied the antimanic properties of clonidine in eight patients. Patients 1-5 were hospitalized in our department of psychiatry for the present episode, i.e., a typical manic episode with increased mood level, flight of thoughts, increased motor and verbal activity, but not schizophrenic symptoms. Patients 7 and 8 were hypomanic when they came to the outpatient department; they had been hypomanic for at least 8 days when we prescribed clonidine. They continued to be treated as outpatients. Patients 1-5 and 7 and 8 were bipolar, i.e., had previous occurrences of depression. Patient 6, the only unipolar patient, was a long-term inpatient hospitalized for chronic mania, which had not notably improved by the use of neuroleptics.

We progressively increased each patient's dose from $1^1/_2$ to 3 tablets of clonidine per day (1 tablet = 0.15 mg) within 3 days according to tolerance and efficacy. At night all patients received diazepam (10 mg) as a hypnotic, except for patient 7, who received chlorpromazine (25 mg). Although patient 8 had relapsed under lithium treatment, this treatment was maintained (blood level, 0.5-0.6 mEq/liter). We decided to add droperidol in case of insufficient action of clonidine, and this possibility was a means of indirectly evaluating clonidine efficiency.

Inpatients were seen twice daily by a psychiatrist and were rated once daily, always at the same hour (9 a.m.-10 a.m.), by another independent psychiatrist (R.J.) who used the mania rating scale of Bech and associates (7), an 11-item scale on which each item is scored on a 5-point scale (range=0-44). The symptoms assessed concerned motor activity, verbal activity, flight of thoughts, noise level, hostility, mood level, self esteem, contact, sleep, sexual activity, and work.

Outpatients were evaluated under the same conditions by the same rater but at 3-day intervals. All patients were rated before treatment began and again at days 3 and 6 of treatment.

Results

We observed a strong global improvement in three of the eight patients (patients 1, 5, and 8) and partial improvement in three others (patient 4, who improved in all symptoms, and patients 6 and 7, who improved in mood and motor agitation only). Only two patients required administration of droperidol: patient 1 on only the first day of treatment and patient 2, who did not respond to clonidine or to clonidine plus oral droperidol and required droperidol by intramuscular injections. The fact that for 6 patients out of 8, their psychiatrists did not find it necessary to add droperidol is further but indirect argument in favor of clonidine's antimanic effect.

All patients except patients 2 and 4 registered a notable reduction in sleep disturbance. The fact that efficacy was slight until day 3 is probably due in part to the slow increase in doses. Although not indicated in table 1, better results at day 3 were obtained when tolerance permitted a rapid increase to 0.45 mg (3 tablets) of clonidine, which seems to be an effective dose. The age, sex, diagnosis, associated treatments, and scores on the Bech scale for all patients are given in table 1. Clonidine treatment was discontinued on day 2 for patient 3 because of arterial hypotension; all other patients exhibited only slight hypotension (10-20 mm Hg) and no clinical expression during the course of clonidine treatment. Patients 5 and 8 exhibited a mood inversion (depression) necessitating the discontinuation of clonidine after 1 week of treatment; patient 8's mood spontaneously returned to normal, but for patient 5 the initiation of antidepressant treatment was necessary.

Comment

It is too early to ascertain the therapeutic usefulness of clonidine as a common treatment of manic states. Our clinical impression is that clonidine has an authen-

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TABLE 1
Demographic, Diagnostic, and Treatment Data for Eight Manic Patients Taking Clonidine[a]

Patient[b]	Age (years)	Sex	Diagnosis	Associated Treatments (mg/day)	Bech Scale Scores[c] Day 0	Day 3	Day 6
1	28	M	Acute mania	Droperidol, 50 1st day only; diazepam, 10	25	12	3
2	40	F	Acute mania	Droperidol, 150; diazepam, 10	30	31	—[d]
3	67	M	Acute mania	Diazepam, 10	23	—[e]	—
4	33	M	Acute hypomania	Diazepam, 10	19	19	6
5	66	F	Acute mania	Diazepam, 10	32	28	4[f]
6	58	M	Chronic mania	Diazepam, 10	24	16	11
7	44	M	Acute hypomania	Chlorpromazine, 25	19	16	6
8	68	F	Acute hypomania	Lithium carbonate, 750; diazepam, 10	16	10	4[f]
Mean (±SD)	50.5±16.2				23.5±5.5	18.9±7.9	5.7±2.9

[a]Maximum dose=0.45 mg/day.
[b]Patients 1–6 were inpatients; patients 7 and 8 were outpatients.
[c]Range=0–44, where 0=normothymic state and 44=maximal intensity of manic state; a score of approximately 10–20 indicates hypomanic state.
[d]Clonidine discontinued on day 4 for inefficacy.
[e]Clonidine discontinued on day 2 for severe hypotension.
[f]Inversion of mood.

tic antimanic, as opposed to purely sedative, effect. It is now recognized that α-adrenergic presynaptic stimulants such as clonidine and β-adrenergic blockers such as propranolol are able to reduce noradrenergic function (3). A relationship between this property and the effectiveness of these drugs in treating mania is strongly suggested by our results and those showing the efficiency of β-blockers in mania (8). All these data indicate the need for further investigation.

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LETTERS TO THE EDITOR

This section contains reactions to Journal *articles, statements of opinion, brief research findings or ideas, short case descriptions, requests for information, etc. Letters should be submitted in duplicate to the Editor, who makes all decisions regarding publication. Letters must be typed double spaced throughout and should not contain more than 500 words and 5 pertinent references. Criticisms of published articles will automatically be sent to the author(s) for response. Letters will be edited for clarity and conformance with* Journal *style. We regret that we cannot inform writers of the disposition of letters or return those which are not printed.*

More on Medical Responsibility

SIR: I wish to respond briefly to Dr. Frederick J. Stoddard's comment (September 1980 issue) on my article "'Medical Responsibility' in Institutional Settings." It seems to me that Dr. Stoddard and I agree about the problem but disagree about the direction in which the resolution lies. His is the more optimistic view. All of his options rely on significant increases in resources. I hope he is correct, but I see little on the horizon to suggest either that the monies needed to support better care in institutions will be forthcoming or that enough doctors will go into psychiatry or enough psychiatrists will want to work in institutional settings.

The new APA guidelines about which Dr. Stoddard speaks are commendable. However, they do not define adequacy of supervisory contact. Further, the guidelines are not official policy; they may "be adapted to local customs and practice." The billing guidelines are an improvement. Unfortunately they will further erode the already collapsing funding base of many institutions.

High principles that cannot realistically be implemented are merely rhetoric. Many psychiatrists in institutional settings cannot even follow the current guidelines—indeed, the logistics of the system make it impossible. Why then should we feel that publishing new and stricter guidelines will be more than rhetoric? I am not particularly interested in writing a new code of ethics per se; I wish to use such writing to design an improved, if imperfect, delivery system.

It is not correct to characterize my approach as a retreat from responsibility. The retreat occurred long ago when the practices I mentioned in my paper became de facto standards of institutional procedure. Dr. Stoddard and I both advocate advances; he advocates a further advance than I do, but I believe mine has a greater likelihood of being attained.

BEN BURSTEN, M.D.
Memphis, Tenn.

Galactorrhea and the Factors Behind Symptom Production

SIR: Richard M. Zeitner, Ph.D., and associates' case report "Pharmacogenic and Psychogenic Aspects of Galactorrhea" (January 1980 issue) is an example of the difficult beginning attempts we make to understand the interaction of pharmacological, endocrinological, and psychological factors in symptom production. I would like to add these comments to the complex interaction that may have been involved in this case.

Peptide hormones, such as prolactin, are often involved in the regulation of several organs. These different organs may be involved in a single physiological effect with the apparent adaptive advantage that effects on the different organs may be correlated in a "foolproof way" (1).

The brain is included in these organs. Thus, a one-way model of brain-hormone interaction is no longer tenable since hormones affect the brain and behavior. Such effects are currently the subject of much intense investigation (2). An awareness of this mutual interaction may help us to understand and more appropriately treat our patients and, in turn, may help us better understand these interactions.

Prolactin, in addition to playing an important role in mammillary development, also plays an important role in regulating mammalian maternal behavior (3, 4). However, prolactin may have several different functions in different species; thus the specific relevance of the above to humans is questionable. Yet, elevated prolactin levels have been associated in both men and women with decreased sexual desire and sexual dysfunction that may be relevant to maternal behavior (5).

The elevated prolactin levels of the patient described by Dr. Zeitner and associates may have led to an intensification of her maternal desires and conflicts, and added to her feelings of uselessness after the loss of her children. This might occur as a result of either a direct effect of prolactin on the brain or the indirect effect of its induced breast changes on physiological functions and the patient's perceptions of herself. Thus, by the mechanisms noted above, decreasing the patient's medication, as the authors did, would not only be directly helpful in decreasing the patient's obvious symptom of galactorrhea, but might also help to decrease her maternal conflicts.

If elevated prolactin levels directly or indirectly intensify maternal conflicts in patients, decreasing prolactin levels by the use of a dopaminergic agonist such as bromocriptine might be helpful in less complicated cases.

The authors noted that since Ms. A's first psychotic episode she had had three exacerbations, each characterized by auditory hallucinations and preoccupations with "a child being hurt" and "someone desperately needing me." Did the specific content of these hallucinations begin only after neuroleptic treatment and elevation of the patient's prolactin level? Could these be attributed in part to elevated prolactin levels and thus be considered a side effect of her medication?

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MARTIN R. COHEN, M.D.
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Dr. Zeitner Replies

SIR: Dr. Cohen's speculations are intriguing and further accentuate our contention that the most comprehensive formulations of clinical phenomena occur when investigators make attempts to translate etiology and treatment strategies from one level of understanding to another (i.e., a translation of psychological events into the language of neuroendocrinology).

In regard to Dr. Cohen's final speculation, the hallucinations involving "a child being hurt" and "someone desperately needing me" occurred when Ms. A was on no medication at all. Nonetheless, Dr. Cohen's thoughts about the interdigitation of prolactin and Ms. A's maternal conflicts provide another useful perspective in considering this case.

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Prison Mental Health Programs in North Carolina

SIR: "The Violation of Psychiatric Standards of Care in Prisons" (May 1980 issue) by Edward Kaufman, M.D., was timely and challenging. Dr. Kaufman obviously has had personal experiences worthy of noting, especially by those who are unconcerned about the psychiatric care of prisoners. Regrettably, many of these prisoners are poor and black. The miscarriage of justice for some citizens begins before they are ever arrested, and continues during imprisonment and throughout their lives.

However, I have great concerns about some of the sweeping generalizations Dr. Kaufman made about all prison mental health programs, federal and state. I am particularly troubled by the statement that "the conditions in the 3 recently examined prison systems reflect the general situation in prisons everywhere in this country." Apparently, Dr. Kaufman is unfamiliar with the mental health facility of the Federal Bureau of Prisons in Butner, N.C., that has a physical structure equalled by few private institutions and that is certainly superior to the majority of antiquated buildings found at state mental hospitals. The Butner facility provides quality treatment with the opportunity for vocational, occupational, and recreational therapies. Similarly, the mental health programs in the North Carolina State Penal System have been raised to a new and respectable level by the hard work of a nationally known pioneer of correctional psychiatry, Charles E. Smith, M.D., of the University of North Carolina at Chapel Hill, and the staff of the Department of Psychiatry, Duke University Medical Center.

We believe that training institutions should be involved in the management of mental health programs in corrections and responsible for preparing professionals to meet the special needs of incarcerated patients, thus raising the standards of care. In fact, psychiatric residents and medical students from both of these universities are afforded supervised electives in the psychiatric care of inmates. Our students have given this experience astonishingly high ratings.

It seems grossly unreasonable to have state mental hospital systems assume the added burden of caring for mentally ill inmates when most state mental hospitals cannot adequately care for their own patients. Moreover, penal mental health programs provide care for mentally disturbed inmates who are victims of the recent massive deinstitutionalization that occurred without sufficient planning. Penal institutions should develop autonomous comprehensive health care systems because the successful treatment of mental illness does not occur as an isolated phenomenon. Assuming inmates will return to prison once restored, they will require adequate follow-up care. Nothing can be more unsettling than the shuttling of chronically disturbed inmates between treatment facilities that may have discrepant treatment philosophies.

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Enuresis and Incontinence Occurring with Neuroleptics

SIR: In "Nocturnal Enuresis Caused by Psychotropic Drugs" (June 1980 issue) Ramakrishan S. Shenoy, M.D., further corroborated our clinical observation of urinary incontinence in patients receiving neuroleptics (1). Urinary incontinence as a side effect of phenothiazine and butyrophenone derivatives is more common than previously realized.

We reviewed this clinical phenomenon and its pathophysiological implications. Several additional facts need emphasis. Enuresis and/or daytime urinary incontinence occur with the following neuroleptics: chlorpromazine, thioridazine, chlorprothixene, thiothixene, fluphenazine, trifluoperazine, and haloperidol. Enuresis/urinary incontinence with neuroleptics appears to be an acute to subacute treatment-emergent symptom of drug administration occurring within hours or sporadically over the first several weeks regardless of the patient's previous genitourinary condition. This enuresis/urinary incontinence appears to diminish with continued neuroleptic administration or follows an on-off pattern when the drug is discontinued. Enuresis and urinary incontinence are not affected by anticholinergic agents used for associated extrapyramidal symptoms.

An 18-year-old man whom we treated sequentially with haloperidol, chlorpromazine, and fluphenazine had 14 episodes of enuresis or urinary incontinence in the first 65 days of treatment; yet he stabilized on 1200 mg of chlorpromazine and his incontinence did not recur.

Enuresis and urinary incontinence are common phenomena in diseases affecting the basal ganglia (2), e.g., parkinsonism, idiopathic orthostatic hypotension, and striatonigral

degeneration (1). Neuroleptics alter the balance of central biogenic neurotransmitters and cause extrapyramidal symptoms. Furthermore, dopamine agonists may induce urinary retention. Bromocryptine, an ergot alkaloid with significant dopamine-agonist activity, is used to treat parkinsonism and is reported to be beneficial in unstable bladder disturbances with symptoms of frequency, urgency, nocturia, and urinary incontinence unresponsive to other drugs or bladder manipulation (3).

We concur with Dr. Shenoy's statement that α-adrenergic blocking actions do not adequately explain this phenomenon because of the absence of other systemic adrenolytic effects. It should be clarified, however, that primary enuresis in male children is not a disturbance of slow-wave sleep, nor is there a greater than chance occurrence of enuresis in any particular sleep pattern (4).

Enuresis and urinary incontinence in patients receiving neuroleptics may be other forms of extrapyramidal toxicity. Further study is warranted to clarify the prevalence and pathophysiology of urinary disturbances in patients treated with phenothiazine and other antipsychotic compounds. Correlating drug effects with neurophysiology may further our understanding of the action of these agents.

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H. GEORGE NURNBERG, M.D.
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Time Orientation and Ghetto Patients

SIR: I was extremely disturbed after reading "Time Orientation and Psychotherapy in the Ghetto" (March 1980 issue) by Eric Lager, M.D., and Israel Zwerling, M.D., Ph.D. Although the intent of the article was good—to explain that long-term therapy can benefit ghetto patients if the therapist understands their present-time cultural value orientation—the article left a bad taste in my mouth.

There are some elements of truth in the notion "Live today before you die tomorrow." However, the idea that "appointments are never more than approximate schedules; they are kept according to 'black people's time' or 'Puerto Rican time' " is unacceptable. I base this assertion on 10 years of experience in treating ghetto patients. I currently work in a ghetto clinic and see about 125 patients, who are predominantly black. These patients keep their appointments or will call and arrange for another appointment if they are unable to come in. They know I expect them to be on time. The first time a patient arrives late I explain that if he arrives late a second time I will not see him. He will simply have to get another appointment.

I agree with the statement that the "most common cause for stormy reactions and treatment interruptions was the therapist's underestimation of the importance of the therapeutic relationship for the patient." I suggest that the therapist use the importance of the therapeutic relationship to encourage the patient to be prompt and to prepare for the future rather than simply excusing the patient's behavior as being culturally appropriate. It has been my impression that the ghetto patient who casually floats in late to his session is being "cool." It is extremely important that ghetto patients maintain some sense of power and control; to maintain this "overcompensatory grandiosity" they may initially attempt to downplay the importance of the therapeutic relationship by casually floating in late to their sessions. This behavior will continue unless the therapist makes it clear that it will not be accepted. Allowing this behavior to go unchecked only develops and reinforces a defense that undermines the necessity of being on time for work.

Dr. Lager and associates did make some good points which support my contention that their reasons for asserting that ghetto patients are never on time are incorrect. They noted that patients who tend to stay in treatment feel special. I agree with this as I have seen it in my practice. People who are "special" often have expectations placed on them which they are expected to meet. Thus, I explain to my patients that "rank has its price and privilege." As a result of the special status they derive from being involved with psychotherapy, I expect them to pay several prices, one of which is being on time. Compliance with this expectation is usually fairly high. Thus I have a hard time believing that their original lateness stemmed from an orientation toward time that was deeply rooted in their fundamental value system. This fundamental value is too easily stopped by a simple explanation of the importance of being on time and then by enforcing the consequences of arriving late. Of course, if the assessment of middle-class therapists is overloaded with misinterpretations of sexual and aggressive behavioral reports as being signs of impulsive behavior and therefore superego deficits, I would hardly expect such therapists to simply state that if the patient is late, he will not be seen.

Finally, I have not seen among my patients a tendency toward reduced self-awareness or minimal verbal communications about their feelings toward therapy. I have seen that there is an initial "cool" and, considering the high rate of institutionalization of ghetto patients, an appropriate distrust of therapists which does not allow the patients to talk about such issues. However, during the course of therapy that cool falls away rather quickly and one finds oneself with a very aware, sensitive patient. It is only then that a therapist realizes the danger of emotional life in the ghetto and the need to protect narcissistic vulnerabilities through the use of "being cool" and thus being late.

CARL C. BELL, M.D.
Chicago, Ill.

Dr. Lager Replies

SIR: The authors thank Dr. Bell for his comments. Our experience, however, has been that patients who are told firmly that they need to be on time before they have established a relationship tend to drop out in large numbers.

ERIC LAGER, M.D.
Philadelphia, Pa.

The Importance of Clinical Work to Psychiatrists

SIR: This is in response to "Rare Birds, Queer Ducks, or an Endangered Species?" by Sidney Werkman, M.D., "Factors in Medical Students' Choice of Psychiatry," by Paula F. Eagle, M.D., and Luis R. Marcos, M.D., and "Choosing Psychiatry: The Importance of Psychiatric Education in Medical School," by Arthur C. Nielsen, M.D. (April 1980 issue).

Another possible reason for the decline in medical graduates entering psychiatry is suggested by the fact that this decline began in 1965, about the same time that community mental health movement ideals began to influence psychiatry. The community mental health model, in addition to discharging into the community patients unable to care for themselves, displaces the hierarchical medical model by a team approach and a supervising or back-up model of psychiatric practice. Medical students see their psychiatric teachers supervising nonpsychiatric clinicians and administering hospital and academic departments rather than treating patients. They also see the resulting harm to patients. If students believe a doctor's job is to treat patients rather than solely to instruct others, they will be forced to look to other specialties. Academic psychiatrists might take Adolf Meyer's advice more seriously: ". . . all psychiatrists must remain active in the treatment of patients. In this way, the essential perspective of physician as clinician would not be lost, and his primary loyalty to the individual patient would not be forgotten." We might make working at least part-time as a clinician with seriously disturbed patients in an emergency room or psychiatric hospital a prerequisite for teaching medical students. If treating patients were made mandatory for administrative psychiatrists, patient care might also improve. Administrators would learn firsthand how their policies affected patients treated at their facilities. Until these changes are made, medical students may be wise to avoid a specialty that continues to move farther and farther away from providing direct patient care.

LEWIS GLICKMAN, M.D.
Brooklyn, N.Y.

Alcoholism: Mimicking Psychopathology?

SIR: In "Clinical Depression in Alcoholism" (March 1980 issue) Myrna M. Weissman, Ph.D., and Jerome K. Myers, Ph.D., suggested a high rate of psychiatric diagnoses in their alcoholic population. They stressed the need for adequate assessment of alcoholic patients and consideration of potential treatment for disorders and diseases other than alcoholism.

As the medical director of a large treatment center for alcoholics, seeing approximately 13,000 clients per year, I have found that this population commonly has psychiatric diagnoses of manic-depressive illness, varying types of schizophrenia, and major depressive illnesses. We have treated this population for these diagnoses and in approximately 95% of these patients the treatment has been ineffective when the primary disease of alcoholism has not been treated or has been treated ineffectively.

We conducted follow-up studies of patients maintaining sobriety who also carried major psychiatric diagnoses. In 98% of these cases, we found no evidence of abnormal mental states approximately 6 months to 1 year after onset of sobriety. It is our assessment, as well as that of others in the field of alcoholism, that the alcoholic disease process can mimic severe psychopathology during intervals between drinking episodes or during alcohol toxicity itself. We strongly recommend that no alcoholic patient be treated for psychiatric disorders until the primary alcoholism process has been arrested.

FREDERICK A. MONTGOMERY, M.D.
Yakima, Wash.

Dr. Weissman Replies

SIR: We thank Dr. Montgomery for his thoughtful letter and would certainly agree that acute alcohol toxicity can mimic a variety of other psychiatric disorders. However, this does not detract from our finding that some, but certainly not all, alcoholics do have other psychiatric disorders that are evident before the alcoholism and that seem to persist afterward.

A follow-up study using precise research diagnostic criteria of alcoholics who are maintaining sobriety 6 months to 1 year after the onset of sobriety would be quite useful. Such a follow-up might document Dr. Montgomery's clinical observation that a large percentage of these patients show no evidence of abnormal states even though they did so while untreated. It would also identify that subgroup who do have other psychiatric disorders which could be effectively treated. I am unaware of any published studies of this kind using systematic diagnostic procedures.

MYRNA M. WEISSMAN, PH.D.
New Haven, Conn.

Withdrawal States and Alcoholic Hallucinosis

SIR: In "Psychosis with Withdrawal from Ethchlorvynol" (February 1980 issue) Leonard L. Heston, M.D., and Donald Hastings, M.D., reported an unusually prolonged withdrawal state with psychotic symptoms occurring after abrupt termination of ethchlorvynol. Important features of this state included onset of extensive tactile, visual, and auditory hallucinations and the presence of a clear sensorium 8 days after the drug has been discontinued. Drs. Heston and Hastings did not comment on the status of the patient's vital signs during the initial period of his disturbance. A schizophrenic-like state, with continual auditory hallucinations, Schneiderian features, and affective blunting persisted for 18 months until the patient apparently ended it by self-administration of alcohol. Eventually the patient recovered completely.

Although Drs. Heston and Hastings speculated on the relationship between alcohol, ethchlorvynol, and the patient's psychosis, they overlooked the striking similarity between this syndrome and alcoholic hallucinosis (1). In the latter, during or after alcohol withdrawal the patient exhibits symptoms that are difficult to distinguish from schizophrenia, but does not exhibit the confusion commonly associated with withdrawal delirium. Although clinicians have frequently suspected an underlying schizophrenia and often attribute the patient's alcoholism to an attempt to "self-medicate" for

this condition, there seems to be no genetic background of schizophrenia associated with these cases (2, 3). In fact, alcoholism and schizophrenia are apparently infrequently associated (4). The symptoms often become chronic, although shorter courses are more common (1).

To my knowledge, a similar syndrome has not been associated with withdrawal from sedative drugs other than alcohol; perhaps this case is the first. The disappearance of hallucinations after alcohol intake may indicate that withdrawal of both alcohol and sedative-hypnotics produce hallucinosis by similar mechanisms and can substitute for each other effectively in its "treatment." Other questions remain unanswered, however, such as the possibility that withdrawal from alcohol itself caused the prolonged hallucinosis and why symptoms that were so resistant to pentobarbital during hospitalization were so dramatically affected by alcohol months later.

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EDWARD L. MERRIN, M.D.
San Francisco, Calif.

Dr. Heston Replies

SIR: We thank Dr. Merrin for emphasizing different aspects of our case report, but we do not think there is a substantial argument between us. Of course alcohol must be considered in the total clinical picture; we suggested an additive or synergistic effect with ethchlorvynol. However, we think that alcoholic hallucinosis alone is an unlikely explanation of the clinical picture. Our patient's alcohol intake had not been excessive before the first hospitalization but he had been taking large amounts of ethchlorvynol over the years. (We regarded our medical history as unusually reliable.) We saw three possible explanations for the clinical phenomena observed. 1) Alcohol withdrawal alone. We considered this most unlikely because we know of no report of such prolonged withdrawal among the many thousands of patients who have been observed. 2) Ethchlorvynol withdrawal. This seems a better bet because the dose was so high, the period of use was so long, and the few reports in the literature are consistent. 3) Ethchlorvynol plus alcohol. This is our choice because the patient used both drugs, which are pharmacologically similar, so that substitution (in the pharmacologic sense) and interactions could reasonably be expected.

Our only possible disagreement with Dr. Merrin is a mild disclaimer. We think that comparable withdrawal syndromes occur involving glutethimide.

LEONARD L. HESTON, M.D.
Minneapolis, Minn.

On Reading Lacan: Fundamental Concepts

SIR: In his review of Jacques Lacan's *The Four Fundamental Concepts of Psychoanalysis* (July 1979 issue) Garfield Tourney, M.D., stated that "to read and attempt to comprehend Lacan proves a near impossibility."

It is true that approaching Lacan's essays expecting to gain access to delineated concepts conveyed through the operation of logical, propositional reasoning is an exercise in futility. However, we should not read Lacan this way any more than a therapist should listen to a patient's expression with only that critical faculty which judges the veracity of propositions according to Aristotelian categories. Lacan's writings are examples of free associative cognitions and are essentially rich in nuance, metaphor, symbol, and evocation—in that ambiguous structure which is the fabric of dreams and which Freud designated collectively as the dream work in chapter six of the *Interpretation of Dreams*. Lacan's discourse requires an act of comprehension adequate to the human experience of ambiguous expressive meaning, an act of comprehension which is no less obscure when it is raised to the level of a "fetish" (literally, a disavowal), as Dr. Tourney suggested.

The density and overdetermination of Lacan's writing is disclosed through that special organ of sense Freud called evenly poised attention (*gelichschwebende Aufmerksamkeit*) (1), with its implication of bracketing the operations of judgment, cognition, and perception—characteristics of everyday understanding. Lacan should be read like the text of a dream, a good joke, or the fluid interplay of image found in such inspired poets as Rimbaud. The reader must preserve his or her capacity for surprise.

Lacan does not suffer from a "tendency to ambiguity" as a symptom. Before any judgment of obscurity or observation of symptomotology, human expression is essentially ambiguous and provisional; this fact enlivens Lacan's writings and is never lost sight of even in its explication. When reading Lacan we should not lose sight of the observation of his colleague and friend, Maurice Merleau-Ponty, that "the will to expression itself is ambiguous and contains a ferment that works at modifying it" (2). Lacan referred to this as the Discourse of the Other. Nurturing and conveying the ambiguity of experience is not the same as being lost in a web of words or defective reasoning.

This may also be seen from the perspective of reading Lacan in the tradition of the "increasing medical orientation of American psychiatry." A positive, productive approach to Lacan may also be found in the recent work on hemispheric lateralization of cognitive functioning. Following the inspiration of Bogen (3), we should read Lacan with the appositional logic of the right hemisphere that provides, as McLaughlin (4) pointed out, a neurobiologic foundation for the image-enriched, affective, and ambiguous Gestalten of primary process type cognitions.

Dr. Tourney criticized Lacan's writings for not being more propositional—that is, not being more consistent with the expressive intention of the left hemisphere. But can the left hemisphere criticize the right for seeing the world from a different angle? Isn't it amazing that both hemispheres could find a proper symphonic balance and compromise to produce a work as suggestive of novelty and as rich in expression as Lacan's? Lacan's work should be read as a melody, comparable to the great compositions of Mahler and Ravel. Ravel, when rendered mute with expressive aphasia, still pre-

served his melodic, rhythmic, and stylistic sense. If Lacan suffered from a propositional aphasia, it is then equally true that the preserved melodic sense will yield a productive reading of Lacan.

In closing, let us follow the suggestion of Dr. Tourney and take a phenomenologic reading of Lacan. Let us be guided by Lacan's own words in finding our way through his labyrinthine texts: "Without first racking our brains to understand this meaning, how can one translate what is not itself language? Led in this way to appeal to the subject, since it is to his account which we must transfer this understanding, we shall implicate him with us in a wager, a wager that we understand him and then wait until a return makes us both winners. As a result of continuing to perform this shuttling back and forth, he will learn quite simply to set the pace . . ." (5).

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EDWARD J. EMERY
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Correction

In "Psychological Manifestations of Nonbacterial Thrombotic Endocarditis" by Thomas B. Mackenzie, M.D., and Michael K. Popkin, M.D., in the August 1980 issue, the last sentence of the first paragraph on page 972 should be, "It is generally considered to be a hypercoagulable state." The staff regrets this error.

BOOK FORUM

NANCY C. ANDREASEN, M.D., EDITOR

The Powers of Psychiatry, *by Jonas Robitscher, J.D., M.D. Boston, Mass., Houghton Mifflin Co., 1980, 538 pp., $17.95.*

Dr. Robitscher was a lawyer before he was a doctor. Through medical school and psychiatric training, "some legal part of myself," he writes, "saw the psychiatrist-patient relationship through the eyes of a lawyer." Some legal part still does, if his book is an indication.

In a 538-page brief (it would have been longer, he says, without his able editor), he accuses psychiatry of many crimes. Psychiatrists, he says, give too many pills, push the ECT button too often, and recommend unjustified lobotomies. They meddle where they shouldn't, tarring politicians with armchair diagnoses, pontificating about world affairs, and handing down Olympian judgments about social problems. Judges and juries actually *listen* to them (this will surprise many psychiatrists) and base decisions about who gets custody of children, who goes to jail or goes free, who gets a job or does not, on dubious psychiatric advice. Psychiatrists open the door for draft-dodging and abortions because they dislike war or favor abortions, or simply for money.

Psychiatrists are not scientific. They are not really physicians. They pretend to be both, and society buys it. As a result, psychiatrists have become enormously powerful. They have replaced priests as the preeminent authority on what is "right and wrong, moral and immoral, expedient or inexpedient."

No one will question the abuses listed by Dr. Robitscher. Some will question how widespread they are. Few will disagree that psychiatrists dislike hearing about abuses and prefer to look the other way.

Many will question how powerful psychiatrists have become. Dr. Robitscher's book is not a Szaszian polemic—he thinks psychiatrists often do good, maybe more often than not—and he occasionally is caught in contradictions. Here is a charming example:

> During several years when I was a consulting psychiatrist for a prison, I learned the excitement and pleasure of wielding great power—the thrill that came with the ability to confer liberty, the thrill that came from knowing I could deny liberty by not recommending parole. . . . I learned that there were institutional rules that took precedence over clinical judgment. For instance, a patient should not be recommended for parole too early, no matter how I saw his psychiatric status, since the parole board would not favorably consider such a request. . . . I learned to tailor my psychiatric evaluations to the facts of prison life.

This, perhaps, better reflects the perception of most psychiatrists of their professional life than does the untrammeled exercise of naked power described in this book. At a time when fewer medical students are entering psychiatry, when psychiatrists are lampooned in fiction and by the press, when restrictive legislation makes the practice of psychiatry almost impossible, psychiatrists should read this book just to raise their spirits. They may not be as powerful as Robitscher says, but in bad moments they might like to think they are.

On abuses this is a serious, carefully documented book by one of our leading doctor-lawyer scholars. It should be taken as seriously by readers inside the profession as it undoubtedly will be taken by those outside.

DONALD W. GOODWIN, M D.
Kansas City, Kan.

BRIEF MENTION

Adjustment: The Development and Organization of Human Behavior, *by R. Douglas Whitman. New York, N.Y., Oxford University Press, 1980, 275 pp., $15.95.*

This book was designed as a text for use in courses dealing with adaptation and adjustment. Like many texts designed for use in undergraduate psychology courses, it could also serve a useful educational purpose for residents or even for senior clinicians who wish to expand or update their knowledge of psychological research and theory in the area of stress disorder.

They should not buy this book for that purpose, however. It is nicely designed and printed, bringing to mind the old aphorism that you can't judge a book by its cover. The author has tried to cover more material than he knows well, and as a consequence the treatment is intolerably superficial. He attempts to discuss such diverse and unrelated areas as the scientific method, basic genetics, social psychology, psychodynamics, the nosology and classification of disorders, the etiology of various psychiatric disorders, and treatment. His references are often outdated and sometimes wrong, and the contents tend to be excessively sketchy and glib. Stress and stress disorders receive very little attention.

Biological Studies of Mental Processes, *edited by David Caplan. Cambridge, Mass., MIT Press, 1980, 322 pp., $24.95.*

Psychiatry has always enjoyed a broad frontier with neurology. Recently, psychiatrists have become more interested in the biology of brain function and its relationship to the disease process, while neurologists, neuropsychologists, and psycholinguists have been increasingly successful in mapping out areas of brain function and abnormality. This book provides a useful introduction or overview of recent developments in cognition and language. It condenses a great deal of useful information in a single volume.

It contains 15 chapters that fall naturally into three sections. The first section contains studies of the maturational

component of cognitive development. The second section examines aspects of language development. The third section focuses on neural mechanisms underlying language in the adult. Topics such as cerebral dominance, the neurology of the aphasias, and current theories of psycholinguistics are interlaced through these chapters. The authors represent a blend of talented young investigators and distinguished senior researchers, such as editor David Caplan, Noam Chomsky, Thomas G. Bever, Mary-Louise Kean, Merrill F. Garrett, Edgar B. Zurif, Jason W. Brown, and Norman Geschwind.

After this enthusiastic recommendation, however, a caveat should be added. People who are experts in language are not necessarily experts in communication, and some of the concepts conveyed in this book are likely to be difficult or unfamiliar. It is an excellent book for those who wish to venture into the new frontiers of neuropsychology and psycholinguistics, but the journey will require time and intellectual effort for most readers who pick up this book.

Migraine: The Facts, *by E. Clifford Rose and M. Gawel. New York, N.Y., Oxford University Press, 1979, 104 pp., $10.95.*

This is a clearly written and well-designed volume directed to lay readers who wish to learn more about migraine. It is the sort of book that a psychiatrist may prescribe for his or her patients to relieve their aching curiosity, if not their aching heads. It discusses the definition of various types of headache, causes of headache, predisposing factors, pathophysiology, and treatment. The presentation is simple and clear, and an abundance of excellent drawings supplements the discussion in the text.

Fridericus, *by Frederic F. Flach, M.D. New York, N.Y., Lippincott & Crowell, 1980, 254 pp., $9.95.*

Frederic F. Flach, a New York psychiatrist whose previous work has included biological research in depression and several books on psychiatry addressed to lay audiences, has now written his first novel. Drawing on his personal experience of having found a book written about melancholia in the seventeenth century by one Fridericus Flacht, he has let his imagination consider the possibility and implications of reincarnation. The protagonist in this novel, Dr. Frederic Pleier, is a New York psychiatrist who discovers that he may be the reincarnation of a seventeenth-century physician who lived in Worms during the dreadful pestilence of the Thirty Years War. Dr. Pleier finds himself intermittently subject to periods of disappearance, during which he appears to be mysteriously drawn back into the seventeenth century to solve and work through some unfinished business. Naturally, his twentieth-century friends and colleagues are concerned. Is he crazy? Is he suffering from seizures? Is he depressed? Should he receive psychoanalysis or ECT? The novel shows many signs of being a first work, such as lack of personal distance and shallow characterization. Nevertheless, its plot line is well drawn, and suspense is well handled. Some psychiatrists should enjoy reading this first effort by one of their colleagues.

N.C.A.

Handbook of Geriatric Psychiatry, *edited by Ewald W. Busse, M.D., and Dan G. Blazer, M.D. New York, N.Y., Van Nostrand Reinhold Co., 1980, 532 pp., $34.50.*

Try as one may, it is impossible to do justice to a handbook of this high caliber in the space allotted here. It is a handbook in every sense of the word and an excellent one at that, for it is a ready reference of eclectic orientation to the theory and practice of geriatric psychiatry. Its separate essays are broad and well done, its authors are highly skilled physicians and biomedical and behavioral scientists of equal capabilities. It is a true vade mecum, and I am tempted to quote whole pages of it.

The first 10 chapters are devoted to the biological and psychological bases of geriatric psychiatry. These cover diverse aspects of the specialty—its theories and processes, the neuroanatomy and physiology of aging, the neuropharmacology, perceptual changes, psychology, and social factors, among others.

The second section is concerned with diagnosis and treatment in the light of *DSM-III* with its multi-axial approaches to diagnosis. The material is timely and includes data that will facilitate understanding of the problems at hand as well as indicate approaches to treatment.

The third section of the work has to do with future directions and presents two fine chapters, one by George Maddox on the "Continuum of Care: Movement Toward the Community" and the editors' final discussion, "The Future of Geriatric Psychiatry." Together the chapters indicate that the physician who would practice geriatric psychiatry in the future will have to broaden his or her outlook and develop a working knowledge of the total delivery of health care.

Various theories of biological aging are discussed. The authors note that for all races there are 128 women for every 100 men after the age of 55. After the age of 75, in the year 2000 there will be 191 women to 100 men.

Current thought indicates that aging per se does not independently alter personality, but the passing of time and the biological state plus events in the life cycle do influence both personality and behavior. Experiences become increasingly different rather than similar as a person ages.

Neugarten is quoted as believing that there are sex differences in certain personality traits in late life. Men are more affirmative and likely to ally themselves with groups of men; women are more individualistic.

Various psychological theories are discussed. Apparently people whose lifestyles are altered least exhibit the highest life satisfaction. To attain long life and happiness, continuity is preferred. Prejudice, minority status, and gerontophobia are considered; the health disciplines have these to deal with along with economic problems and social isolation. These plus many other complaints often make the physician feel he or she has little to offer. Social theories are discussed. One of these, which must appeal to the practicing physician, is the "activity theory" holding that the maintenance of activity is important to most individuals as a basis for maintaining satisfaction, self-esteem, and health. The doctor understands that.

Unfortunately, some elderly people have minority status thrust upon them; these individuals have little to look forward to—the gerontophobia seems to move from disliking aging things to disliking the elderly. Again in these situations the doctor feels impotent. At the White House Conference in 1971 it was estimated that four million of the twenty million

individuals over the age of 65 have suffered moderate to severe psychiatric impairment from various causes. Of this four million, 20% were said to be not legally competent.

Traditionally it has been assumed that senile dementia is due to cerebral arteriosclerosis, but more recently multiple infarctions have been held responsible for approximately a third of the dementias; the majority of these are of the Alzheimer type and unrelated to arteriosclerosis.

Evidence suggests that there is a fundamental relationship between the aging process and changes in neurotransmitter substances in the brain and in other regions of the body. Environmental and genetic influences may be interposed that will alter the rate of aging and affect the time when age-related psychology may appear. Many of the phenomena of aging may ultimately be traced to neuroendocrine factors or anatomic loci in the brain. We need to learn a great deal more to determine the importance of neurotransmitter substances to aging and the degree of control that might be developed by their manipulation.

As to the neuropharmacology of aging, it is estimated that almost a third of the people over 60 receive some kind of psychotropic medication in the course of a year. The chapter discussing this topic is excellent. It considers many of the medications used by the elderly and also considers anxiety and sleep disorders.

These are simply snippets of the many fascinating discussions that cover all aspects of the lives of the elderly. I am a bit apologetic in offering them, but I have hesitated to get into the chapters on memory, cognition, and the physical changes that occur in older years, for these are of particular interest to me and I would devote too much review space to them. Suffice it to say this is a well written, complete, and excellent reference book. At the risk of sounding trite I add that it should be in the hands of everyone who deals with elderly patients. There is information in it which will at some time come in handy, that I can guarantee.

F.J.B.

The Lust to Annihilate: A Psychoanalytic Study of Violence in Ancient Greek Culture, *by Eli Sagan. New York, N.Y., Psychohistory Press, 1979, 224 pp., $12.00.*

This book purports to be a study of violence in ancient Greek culture, a study informed by psychoanalytic principles. It is also an attempt to say something about universals in human violence, and as such it frequently draws parallels between ancient and modern political and military behavior. It is this attempt to use ancient Greece as a case study of human violence which holds interest for psychiatrists.

This slim volume contains many interesting ideas and insights about ancient Greek culture. Although not a professional student of Greek culture, Eli Sagan, a sociologist with a psychoanalytic bent, presents some fresh perspectives on the violent side of Greek culture. His speculations about conflicts of authority that permeate the *Iliad,* about the particular oedipal resolution suggested there, about how conflicts within the group are resolved by projecting hatred onto a common enemy, are of great interest. One can also learn a great deal from his observations of Greek tragedy, and he has some astute discussion of the pained and painful family relationships represented therein. His analysis of Thucydides' great history of the Peloponnesian war, Greek tragedy acted out on the stage of politics and murderous warfare, draws our attention to modern rhetoric on the justification of wars. I also think there is something to be said for the author's contention that Judaism and, later, Christianity introduced ideals of love and compassion that were certainly not emphasized (although not absent) in Greek civilization. For all these things, the book is worthwhile, and I believe many readers of this journal could read it with profit.

I have, however, a number of serious reservations about this work. First, the author is not clear on when he is talking about something uniquely violent in Greek civilization, or about violence in much or all of human civilization. I am always uncertain about what his "standard meter bar" is for measuring the Greek "lust to annihilate." Warfare, and all that went with it, was pretty grim in the ancient world (as in the modern). The book is strangely unhistorical in that regard. The Persians were not exactly nice guys either. The Romans had at least an average amount of military and political cruelty, and imperial Rome had quite a taste for popular sadism in the public forms of entertainment. The ancient Hebrews, by divine command, could be ruthless with their enemies. King Saul fell out of favor with God for not exterminating every man, woman, baby, and beast of the defeated Amalakites. War is hell, war was hell, and in the ancient world to be a loser was to be in the lowest circle of hell.

The author acknowledges, but to my mind does not take seriously enough, the facts about war and violence waged under (and sometimes in the name of) Christian civilization. I agree that ideals about peaceable resolutions and brotherly love fostered by the monotheistic religions are good and desirable and may, ultimately, make a difference in human behavior. But surely only the most naïve students of history would predict when these ideals will make enough of a difference to radically alter the story of war and destruction that has gone on in modern times.

Further, if the author is doing a psychoanalytically oriented analysis of values of Judeo-Christian civilization in comparison with Greek civilization, he must take into account the totality of the values in the former. Greek tragedies reveal a view of the gods as capricious and cruel, but what about the central image of Christianity—a deity who wants to have his own son suffer cruel torture on a cross? Surely a balanced analysis must take that into account and at least consider the relation of the crucifixion story to the question of the psychology of violence. After all, for centuries the persecution of the Jews was justified in terms of their alleged role in that story.

Another criticism comes from my bias as a Grecophile. I do not think the author has very much empathy with ancient Greek civilization. He seems on a crusade to magnify the bloody aspects of Greek civilization and gives us an unbalanced view (in the name of correcting other unbalanced views). I also see the author's solution to human violence, the universal establishment of democratic socialism, as a bit naïve and anticlimactic. It is not that I am against democratic socialism (I am more for it than for other political systems), but surely any student of human violence must take seriously a tragic view of the side of human life, and that tragic view was certainly brilliantly articulated by the Greeks. In short, I suspect that the author has not made explicit enough his own values in this inquiry, while he argues the impossibility of value-free social science.

Finally, the author is not as original (but also not as alone) as he seems to believe. With wider reading, or wider consultations within the field of ancient history and civilization, he would have discovered views relevant to his own on the combative and violent side of Greek life, e.g., Badian (1) and Havelock (2). Similarly, his discussion of the Oedipus complex is too bookish and could have been enhanced by consulting with clinicians who use the concept in their daily work.

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Psychobiology and Human Disease, *by Herbert Weiner, M.D. New York, N.Y., Elsevier, 1977, 651 pp., $42.50.*

Over the past 10 years developments at the frontiers of medicine have challenged traditional psychosomatic concepts and have created exciting opportunities for research on the mind-body problem. At the same time the increasing complexity of biomedical science has made it difficult for psychiatrists to stay abreast of medical knoweldge and maintain the integrated overview of disease processes that has always been the desideratum of the psychosomatic approach.

In this context Herbert Weiner's extraordinary book is a timely and important contribution. He has prepared authoritative reviews of seven familiar medical disorders that are traditionally associated with psychosomatic medicine. These include peptic ulcer, essential hypertension, bronchial asthma, Grave's disease, rheumatoid arthritis, ulcerative colitis, and regional ileitis. For each he presents a synthesis of current information on pathogenesis and natural history drawn from disciplines ranging from sociology to molecular biology. In the process, he presents each disorder as representing a physiological system and a medical subspecialty. He also reviews the history of psychosomatic approaches to the seven disorders and subjects these approaches to critical analysis in the light of the latest biomedical research.

By presenting these reviews Weiner accomplishes several aims. Taken separately, each review constitutes a lucid and authoritative status report that provides psychiatrists with a synthesis of information not readily available elsewhere. Together they give a history of psychosomatic approaches, concepts, and methodology that shows the origins of current trends. In this context Weiner's detailed critical analysis of experimental design is especially interesting. The reviews also serve to introduce the more technical aspects of biomedical science, including important developments at the molecular level in immunology and cellular biology. Finally, Weiner uses the synthesis of material from various disciplines to make creative suggestions for future research.

In the last chapter Weiner goes beyond the specific disorders to consider general issues in the psychobiological approach to human disease. He reexamines fundamental questions in psychosomatic medicine, such as the problem of the transduction of experience into changes in physiological state. Here he draws extensively from current research in neurobiology. He warns repeatedly against the dangers of oversimplification and premature closure. In the review chapters he indicates that each of the seven entities may actually represent a group of disorders brought together because of clinical similarities, and each may arise from heterogeneous factors and processes.

The book represents a prodigious accomplishment. Weiner seems to be at home with each physiological system and with each scientific discipline. He has given the book the unity of style and approach that can be found only with single authorship. At times it is difficult to read because the material is dense and complex. The theoretical material in the last chapter requires careful study. However, the effort involved in reading the book will be richly rewarded.

Trainees at all levels are likely to find valuable information and concepts in Weiner's book. Even the most skeptical medical students would appreciate Weiner's approach. Psychiatric residents, faculty members, and senior clinicians might all benefit from the scholarship that has gone into the preparation of the book. It would be an important addition to the library of any consultation/liaison service. It would be especially useful for anyone who is involved in a career of psychosomatic research.

The heuristic value of this book comes in part from Weiner's meticulous attention to detail. However, one of the themes that sounds through the details most clearly is the enduring quality of clinical observations.

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The First Year of Life, *edited by David Shaffer and Judy Dunn. New York, N.Y., John Wiley & Sons, 1979, 211 pp., $22.50.*

Drs. Shaffer and Dunn, critical clinicians themselves, have attempted in this book to bring together a group of authors whom they asked to provide some well-reasoned material on the later implications of early childhood experiences. The authors, who either present their own research or have prepared a critical review on a particular aspect of the theme of the book, are all highly regarded European researchers from the fields of psychology, neurology, and psychiatry. They make the book an up-to-date document on the topic at hand.

The book is divided into two sections: a theoretical section and a practical one presenting specific studies. The first section deals with such issues as the techniques and problems encountered in the study of social behavior during the first weeks of life (by M. Packer and D. Rosenblatt), the continuities and discontinuities of individual differences (by J. Dunn), and cross-cultural perspectives on social relationships in infancy (by N. Burton Jones, R.H. Woodson, and J.S. Chisholm). All of these chapters present a good summary of current knowledge but are at times so condensed that the uninitiated reader may be overwhelmed by the number of studies each author reviews and consequently unsure of the relevance of specific investigations.

There are, however, exceptions. A.F. Kalveboer, who is a member of Heinz Prechtl's group in Groningen, gives a succinct description of this group's work on the neurobehavior-

al sequelae of pre- and perinatal complications, and Arnon Bentovim tries with some success to integrate psychoanalytic concepts with recent research findings in child development.

The best of the book, at least for the practicing clinician, is the second section. Here A.M. and A.D.B. Clarke give a superb overview of the limited effect of early experience on later development. Their array of well-documented studies showing how later corrective experiences can ameliorate early caretaking damage is impressive and convincing. F. Hall, S.J. Pawlby, and S. Wolkind continue by presenting more follow-up data on their excellent studies correlating a woman's past social experiences with her later caretaking practices. These authors clearly document that mothers who come from disrupted backgrounds stimulate their infants significantly less both vocally and in touching than do normal controls. Olwen Jones touches on an issue frequently discussed among developmentalists today, namely, the possibility that parents try to make up for neurological or cognitive deficits of their children by providing them with extra stimulation. Thus Jones shows that mongoloid children are stimulated more by their mothers in a number of situations than are normal children.

The last chapter of the book reports on the 8-year follow-up investigation of Barbara Tizard's well-known study of institutionalized children in London. Since some of these children had been adopted or reunited with their biological mothers, Tizard was able to evaluate the effects of various caretaking regimens on the later school and behavioral functioning of these children. Her results make fascinating reading and demonstrate that the study is obviously one of the best of its kind. Her chapter, together with some of the others mentioned above, makes this book an important addition for the clinician interested in the field of infant development.

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Family Therapy of Drug and Alcohol Abuse, *edited by Edward Kaufman and Pauline Kaufmann. New York, N.Y., Gardner Press (Halsted Press, John Wiley & Sons, distributor), 1979, 272 pp., $24.95.*

Innovative and informative are two adjectives I would use to generalize about this book. The first of two major sections, titled Drug Abusers, contains 10 chapters; the second section, Alcohol Abusers, consists of 7 additional chapters. The authors are many, yet there is a surprising continuity and clarity.

A theme running through the entire volume is that people who abuse alcohol and drugs do so through learned behavior. This is not to imply that family therapy in its various forms can be considered a panacea, but it must be included in the therapeutic armamentarium and given a relatively high priority. Overall, this is a readable and useful book, highlighted by several chapters, notably chapters 1, 4, 9, and 10 in the first section and chapters 11, 12, and 15 in the second. Brief comments on these follow.

Chapter 1, "Constructing a Therapeutic Reality" by Salvador Minuchin, M.D., presents an excellent overview of the field with a theoretical basis and a concept of family relationships. The author makes some critical points: "Being correct has nothing to do with relevance. The therapist must learn to focus on that which is relevant. Truth needs to be relevant." Minuchin emphasizes the active process in therapy as dynamic and changing with the specific goals.

M. Duncan Stanton, Ph.D., and Thomas C. Todd, Ph.D., collaborated to coauthor chapter 4, titled "Structural Family Therapy with Drug Addicts." Their major points are that blame must not be affixed to the parents and that there is "no concern with insight or awareness but rather a focus on contemporary issues with a fair degree of success." They also emphasize the importance of having an active, involved therapist who must also be flexible, supportive, accessible, and enthusiastic. I found their method and descriptions apt and workable.

"Drug Abusing Families: Intrafamilial Dynamic and Brief Triphasic Treatment" by Dennis M. Reilly, M.S.W., introduces brief, time-limited, conjoint family therapy as an effective treatment modality. What was particularly refreshing in this chapter was the observation that to borrow techniques from other family therapy schools of thought constitutes a distinct positive advance. The author deals with how the unhealthy situation can be changed rather than how the situation developed, which, of course, is mandatory in brief therapy.

Section two starts out on a high note with "Family Therapy with Alcoholics," a review by Peter Steinglass, M.D. This is a particularly provocative, exciting chapter as well as the longest one in the book. Dr. Steinglass admonishes the reader that there is good supporting evidence that family therapy has value and is ardently boosted by its enthusiastic advocates. We must continue to objectively evaluate and assess this therapy, as we do any new treatment procedure.

Donald A. Cadogan, Ph.D., in "Marital Group Therapy and Alcoholism Treatment," reinforces what other authors, especially Stanton and Todd, have stated: that active involvement reaps more positive results than therapy by the withdrawn expert silent observer.

My major criticism of this book lies not so much with the content but with the too frequent typographical errors, which suggest to me an unnecessary sloppiness on the part of the proofreader and, sad to say, detract from the total enjoyment of the book—but not enough so that it cannot be recommended. The typographical errors fortunately are not reflected in the major ideas expressed by the authors. However, in any scholarly text, I believe it is particularly important that the proofreading be circumspect and accurate.

For those entering the field of family therapy, for those already involved in family therapy, and for any therapist who might be contemplating its use for alcohol and drug abusers, I heartily recommend this volume. It presents clear theoretical frameworks as well as many practical suggestions for implementing the theory and has enough introspection on the part of the various authors to provide further exploration and examination of this interesting modality.

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Principles of Clinical Psychiatry, *by Arnold M. Ludwig. New York, N.Y., Free Press (Macmillan Publishing Co.), 1980, 242 pp., $22.95.*

Selling textbooks of psychiatry must be a tough business. Medical students sometimes buy one but rarely read it. Psy-

chiatric practitioners usually possess an antique version, while academic purists disdain them. Examples that survive this hostile environment tend to focus narrowly on the "quick and dirty" approach for students or an increasingly mammoth, encyclopedic format with a thousand authors and stultifying prose, all priced for the OPEC market.

Arnold Ludwig's *Principles of Clinical Psychiatry* is not just a new entry; it's a very different animal. Organized around principles of clinical logic, it uses the "pearls of wisdom" approach favored by a few dimly recalled professors of clinical medicine from one's undergraduate days. The book describes major psychiatric illnesses crisply and systematically in terms of cognitive, emotional, perceptual, physiological, and behavioral disturbances. There are many tables of specific clinical features of particular illnesses, "clinical alert" charts consisting of historical and psychopathological features to clarify differential diagnosis, and many diagrams to explain real or putative mechanisms whereby psychiatric conditions develop. Ludwig's focus is assertively medical in the best sense. He likes to observe and examine, diagnose and treat. He eschews the fuzzy and emphasizes the soundly proven treatments, which usually means somatic ones. All of this has a no-nonsense aura, but Ludwig is never strident or sarcastic toward the time-honored, if scientifically unproven, therapies, which are, of course, primarily psychosocial.

What then are the flecks in the varnish? For one thing, the book predates *DSM-III*, while evidently anticipating it by studiously avoiding the moribund *DSM-II*. For a diagnosis-first clinician, this entails some obvious problems in organization and definition. All big-league depressions are classed within a rubric (and chapter) entitled "The Psychoses," whether or not psychosis is actually present. This is misleading. Grandiosity is cited elsewhere as weighing toward a diagnosis of schizophrenia, even though "grandiose themes are also common in manics." Agoraphobia seems confused with panic disorder *(DSM-III)*. Ludwig dismisses the diagnosis of borderline personality as a "wastebasket" somewhat too peremptorily. Lots of clinicians may have foundered on this one, but there are some clinical data and clarity is beginning to emerge. The concept of masked depressions is approved despite the general consensus, at least among academics, that this topic holds more dangers than rewards.

On the whole, problems of psychiatric taxonomy are handled gracefully enough, since the focus of the text is on how to think and judge. Of course, this cannot be separated completely from how to label.

At the treatment level, more problems crop up. The optimum lithium level for manic-depressive illness is cited as 1.2–1.8 mEq/liter, high by current standards. Phenelzine dosage is recommended to top out at 60 mg per day on one page and 75 mg per day on another, even though the *Physician's Desk Reference* sanctions 90 mg per day. The treatment of panic disorder whether or not associated with agoraphobia is not really clear at all. Ludwig suggests that monoamine oxidase inhibitors may be useful but does not clarify one of psychiatry's cleanest and most effective drug treatments—tricyclics or monoamine oxidase inhibitors for panic disorder.

The layout of the book is readable even though double-column in format, and the text is not spaced too tightly. The many diagrams that compress and codify some information, such as differential diagnosis, confuse other areas. A mandala-like, print-laden "personality wheel" may make some sense but seems more astrological than clear and medical. A flow chart of the metabolism of alcohol tries too hard, needs color for clarity, and looks like a bowl of spaghetti.

Topically, the emphasis on adult psychiatry legitimately excludes child psychiatry. Nevertheless, Ludwig's straightforward approach could have been usefully applied to at least a few conditions in child psychiatry, such as attention deficit disorder and separation anxiety disorder, which have adult sequelae. The chapters on psychosexual disorders and psychological factors affecting physical illness are skimpy to say the least, while organic brain syndrome receives perhaps too extensive coverage. Not surprisingly, Ludwig's chapter on drug habit disorders is excellent despite my reservations about its graphics.

The index is a real disaster. It is much too short for precise reference. For example, the controversial condition "hysteroid dysphoria" appears in bold face type on page 137 but is totally absent from the index. Textbooks need obsessionality in the index as much as they need to avoid it in their prose.

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The Psychobiology of Sex Differences and Sex Roles, *edited by Jacquelynne E. Parsons. Washington, D.C., Hemisphere Publishing Corp. (New York, N.Y., McGraw-Hill Book Co., distributor), 1980, 304 pp., $18.95.*

One will be gravely disappointed if one expects to find in this book an answer to the perennial question; "How do psychobiological, psychological, and social forces interact to shape sex role behavior?" However, if one expects to find a review of the current, relevant literature in this area with appropriate critique, one will be unusually pleased.

This volume grew out of a conference sponsored jointly by Smith College, the Five College Women's Study Committee, and each of the affiliated institutions—Mt. Holyoke, Hampshire College, Amherst College, and the University of Massachusetts. The conference was designed to meet what was perceived to be a growing need in the Five College Women's Study Program; that is, a need to integrate what was new in the psychological and social sciences in regard to the development and maintenance of sex roles. Experts who could integrate and evaluate biological and psychological viewpoints on important topics were brought together and asked to present their critiques of the various areas.

The book is divided into four sections: Psychosexual Neutrality, Sexuality, Women's Reproductive Systems and Life Cycles, and Cyclicity and Menstruation.

The chapters in the first two sections focus on gender role dimorphism, those in the last two sections on patterns of fluctuation in female behavior that have been linked theoretically or empirically to biological process. Dr. Parsons asks in the first chapter, "Psychosexual Neutrality," Is anatomy destiny? She concludes that cross-cultural and cross-species evidence has not yielded many unequivocal answers. It is her belief that there are some near universals—spatial skills and aggressiveness—that reflect a major component of biological differentiation, but even these behaviors differ cross-culturally and are highly malleable. It is her impression that other roles, like child care or defense, are consistently assigned to one gender or another but biological processes are

not the differentiating factors. She also concludes that the degree of gender dimorphism varies markedly across cultures; however, proscriptions and penalties for deviance from cultural norms have not been adequately investigated. She feels that gender dimorphism reflects the influence of biological, historical, psychological, and sociological forces.

The following chapters address in more detail the biological, psychological, and sociological bases of sex-related differences. In particular, the chapter by Broverman, Klaiber, and Vogel looks at gonadal hormones and cognitive functioning. One of their conclusions is that gonadal hormones are psychoactive. Estrogen and testosterone appear to have similar effects on the CNS; that is, both appear to act as central adrenergic stimulants, possibly through their regulatory effect on enzyme monoamine oxidase. These authors also feel (on the basis of their research) that high dosages of sex-appropriate gonadal hormones appear to alleviate depressive symptoms.

In the next chapter, Alexandra Kaplan looks at human sex hormone abnormalities viewed from an androgynous perspective—a reconsideration of the work of John Money. Of particular interest in this chapter is Kaplan's defense of the concept of androgyny, which in its simplest form means an equal balance between masculine and feminine characteristics. From a research point of view, the concept of androgyny increases the number of categories of human sexuality and sex role behavior and allows the researcher to look at psychological sex in a more sophisticated way.

Section two looks at human sexuality, heterosexuality, and homosexuality. The first chapter, by Dr. Rigby, reviews the biochemical and neurophysiological influences on human sexual behavior. The second chapter, by Dr. Bahlburg, reviews current thinking in regard to the biological and hormonal bases of homosexuality and concludes that homosexuality in general cannot be explained by endocrine abnormalities in adulthood. There is, however, some evidence from animal research that there may be prenatal sex hormone differences. These findings are not uniform, and their applicability and interpretation are still open. It may be possible that prenatal hormone abnormalities contribute to the development of sexual orientation in a minority of homosexual subjects, but this has not been sufficiently documented. The following chapter, by Dr. Falbo, looks at a psychological model of human sexuality.

Section three, Women's Reproductive Systems and Life Cycles, is perhaps the most interesting section. In particular, the first chapter, "American Birth Practices: A Critical Review," looks at the relationships between women and the social and technical practices associated with childbirth in American society from a historical point of view. It provides a critical insight into the changing patterns of delivery in the United States today and their impact on mothers and children, an important area of considerable research interest today. The authors conclude that the major hazards of childbearing have not been reduced by modern technology and efficient hospital procedure and that class differences in the safety of childbearing persist. They agree that adequate pre- and postnatal care as well as the health benefits of standard and natural delivery procedures would enhance both the physical and emotional experiences of delivery, which, in their opinion, are enjoyed primarily by middle-class participants in natural childbirth.

Finally, the last section of the book looks at cyclicity and menstruation. The lament of the first author, Carolyn Wood Sherif, that the research literature on the relationship of the menstrual cycle to mood, affect, and behavior is fraught with research error and valueless is echoed in the succeeding chapter by Rossi and Rossi. The Rossis describe their research into body time and social time in the menstrual cycle in great detail.

This book is for teachers involved in courses having to do with sex differences in sex roles. It is unusual in that it is an edited volume in which all of the pieces seem to belong and to flow naturally from one another. The biological data flow into the psychological and social data easily, and earlier chapters make reference to later chapters and vice versa. If one wishes to answer to the nature-nurture conflict one must look elsewhere, for this volume does not give pat answers. It only poses new questions. It is not easy reading, but it is a valuable "state of the art" book that experts on sex role will use and reuse.

ELISSA P. BENEDEK, M.D.
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The Many Faces of Suicide: Indirect Self-Destructive Behavior, *edited by Norman L. Farberow, Ph.D. New York, N.Y., McGraw-Hill Book Co., 1980, 427 pp., $18.95.*

If we label certain behaviors as self-destructive we suggest that they resemble suicide and that recognizing their common motives and psychodynamics can increase our ability to understand and control them. We may also suggest that they are, like suicide, psychopathological and thus the rightful concern of psychiatrists. Most readers of *The Many Faces of Suicide* will wonder whether such labeling is justified or useful and will look to this volume for an answer.

Editor Farberow appropriately dedicates his book to Karl Menninger, who, in the 1940s, developed Freud's concept of the death instinct and identified its expression in a variety of self-destructive behaviors. The book deals with behaviors that Menninger and others regarded as partial or indirect forms of suicide, including drug and alcohol abuse, noncompliance with medical treatment, obesity, cigarette smoking, delinquency and criminal activity, and high-risk sports. Although the contributors are experts in their respective areas, most had not previously considered how the concept of self-destructive behavior might apply to their subject. Consequently the treatment they give this aspect varies. Some make it the central theme of their contribution, others include it among the issues they discuss, and still others mention it only in passing.

For the most part the behaviors considered in this book are long-term patterns associated with an increased risk of death but without acknowledged self-destructive intent. The concept of self-destructiveness proves difficult to define, however, and a host of adjectives, including self-defeating, self-injurious, and risk-taking, are used interchangeably by various authors. Is it, for example, self-destructive when a patient who has had myocardial infarct, using denial, resumes normal physical activity against medical advice? Is it self-destructive when, to maximize the pleasure of a jump, a skydiver opens his parachute at the last moment? Would we call self-destructive the action of a person who cuts himself to restore a feeling of aliveness? Calling such diverse behaviors self-destructive implies that they have something in common beyond an increased risk of physical injury or

death. But that something remains elusive. Many authors approach the concept of self-destructive behavior in an uncritical fashion with the result that it becomes overinclusive and unclear.

The book is not without merit, however. It contains a number of stimulating and useful chapters on various psychopathologic and nonpathologic entities. In a chapter on alcoholism, Connelly thoughtfully examines the dynamics of this disorder and its relationship to suicide. Lichtenstein and Bernstein do not regard cigarette smoking as self-destructive, but their critical examination of that possibility and close examination of the determinants of smoking make a valuable contribution. Simpson's review of self-mutilative behavior is both scholarly and thorough and will be welcomed by those who have to deal with patients who repeatedly cut or otherwise mutilate themselves. An excellent review of the problem of drunken driving and how it might be better controlled is provided by Selzer. Finally, chapters concerning prostitutes and people who intervene in violent crimes offer understanding of behaviors that seem foreign to most of us.

The Many Faces of Suicide is about a host of behaviors that are both troubling and difficult to understand. In examining them closely the authors often recognize adaptive efforts to have gone astray and attempts to cope with life even at the risk of death. Beyond this, they identify dynamic patterns that are as diverse and complex as those involved in suicide itself. Perhaps we should recognize the limitations of our knowledge and remain cautious about intervening in behaviors that we may wish to control because they pose a threat to our values.

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Biofeedback and the Modification of Behavior, *by Aubrey J. Yates. New York, N.Y., Plenum Press, 1980, 504 pp., $24.50.*

The development of biofeedback had a revolutionary effect on psychology, psychiatry, and medicine in the 1960s and 1970s. Biofeedback offered the promise of better health through self-regulation using operant conditioning techniques. Early writings on biofeedback promised drug-free treatment for "psychosomatic" disorders. These predictions also forecast that biofeedback would open new frontiers in the treatment of many stress-related diseases. The response of the medical establishment to the enormous popularity of biofeedback ranged from excitement to skepticism. Research studies on clinical efficacy at first supported claims that biofeedback would replace medicines in the treatment of many psychosomatic, behavioral, and stress-related disorders. Popular publications were often too uncritical in touting the usefulness of this exciting new treatment. Now, however, with the publication of Aubrey Yates's *Biofeedback and the Modification of Behavior,* there is finally a comprehensive textbook on biofeedback that is a scholarly effort to review and organize the vast body of data on this treatment modality.

Yates's book is published by Plenum Press, which has shown a special interest in psychosomatic and behavioral medicine. Plenum's medicine and psychology editorial staff, headed by Leonard Pace, has demonstrated skill in the selection and support of their behavioral medicine series. This book represents a thoughtful and considered approach in attempting to unravel the controversies surrounding the efficacy and clinical applications of biofeedback treatments. Yates states in the preface his intention to review the large literature on biofeedback that has accumulated over the last 15 years. He accomplishes this task with great skill, citing more than 1,300 references. More importantly, he conveys a critical balance in his content and style that lends credibility to his conclusions and helps the reader to form his or her own.

The organization of the book is sensible. An introductory chapter on the antecedents and history of biofeedback is followed logically by a brief yet sophisticated review of instrumentation and methodology. Some readers who are new to biofeedback methods may find this review over their heads and might prefer a more elementary and practical text such as George Fuller's (1). Each of the three major divisions of biofeedback research is covered in separate chapters: "Voluntary Control of Muscle Activity," "Voluntary Control of Autonomic Functions," and "Voluntary Control of Electrical Activity of the Brain." A comprehensive chapter on other applications of biofeedback follows. These include alcoholism, asthma, bruxism, diabetes, eczema, hyperactivity, and insomnia. Each area is thoroughly reviewed, and references are current through 1978. This concludes the clinical portion of the book.

The next to last chapter on theoretical aspects of biofeedback is likely to be of interest primarily to the sophisticated reader. Throughout the book Yates carefully adheres to scientific rigor in his criticism of the research foundation on which clinical practice is based. In the concluding chapter, "The Promise of Biofeedback," he takes off his gloves and skillfully identifies those areas of biofeedback poorly supported by hard data. With tact and fairness he circumscribes those areas in which excessive claims have been made and those needing further research. Yates's criticism is balanced and optimistic. His willingness to openly criticize his colleagues' work is refreshing. I wondered if the geographic distance between Yates's homeland (Australia) and North America enabled him to be so incisive and direct in his dissection of the work of others.

A major strength of this book is the author's attempt to address the major controversies plaguing contemporary biofeedback research. These controversies include the following: 1) Does biofeedback add any significant benefit over relaxation training without feedback in producing relaxation? 2) Does biofeedback training permit generalization of relaxation from one area of the body to others? 3) Do different methods of inducing relaxation differ psychophysiologically or subjectively? 4) Do psychophysiological changes vary with subjective reports according to expectation? 5) How much can the favorable outcome reported in many studies be accounted for by placebo effect? 6) Are the machines really necessary?

Yates tackles these issues head on, supporting his arguments with critical reviews of relevant research. Clinicians will no doubt find fault with his obvious preference for rigorously controlled, laboratory-based studies over the nonspecific but powerful treatment effects often obtainable in the clinical setting.

The book has several features that might be considered weaknesses. The first is that Yates's organizational scheme for presenting his coverage of the three categories of biofeedback research (muscle activity, autonomic function, and

brain-wave activity) appears logical from the viewpoint of the functional organization of the nervous system but not necessarily from the clinical viewpoint. Many clinicians might prefer a "disease-oriented" organization to Yates's "functional" organization. Since the table of contents is not organized by disease, the index, while adequate, lacks sufficient cross-references. For example, eczema is listed only under that heading. A reader looking for sections on the clinical applications of biofeedback in the treatment of psychophysiological skin disorders, atopic dermatitis, dermatitis, itching, or pruritis would not find any page references in the index under those headings. This deficiency could be easily corrected in future editions and would be a useful feature for the clinician. Some clinicians might also object to the heavy emphasis on controlled group outcome studies and the paucity of case reports or longitudinal single case studies. However, my preference is to see individual case data presented in specialty journals such as *Biofeedback and Self-Regulation*, not in reference texts. Finally, the book is in no way useful as a practical guide for the clinician in the art and practice of biofeedback or behavioral techniques. For that the reader must look elsewhere.

This book is the best volume currently available for a thorough overview of the scientific basis of biofeedback practice. It is an essential reference text for anyone using biofeedback techniques in clinical practice and a valuable, comprehensive guide for the interested student, resident, or practitioner.

REFERENCE

1. Fuller GD: Biofeedback: Methods and Procedures in Clinical Practice. San Francisco, Biofeedback Press, 1980

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Basic Handbook of Child Psychiatry, Vol. 1: Development, *edited by Justin D. Call, Joseph D. Noshpitz, Richard L. Cohen, and Irving N. Berlin; Joseph D. Noshpitz, editor-in-chief. New York, N.Y., Basic Books, 1974, 691 pp., $35.50.*

Basic Handbook of Child Psychiatry, Vol. 2: Disturbances in Development, *edited by Joseph D. Noshpitz; Joseph D. Noshpitz, editor-in-chief. New York, N.Y., Basic Books, 1979, 722 pp., $35.50.*

Basic Handbook of Child Psychiatry, Vol. 3: Therapeutic Interventions, *edited by Saul I. Harrison; Joseph D. Noshpitz, editor-in-chief. New York, N.Y., Basic Books, 1979, 673 pp., $35.50.*

Basic Handbook of Child Psychiatry, Vol. 4: Prevention and Current Issues, *edited by Irving N. Berlin and Lawrence A. Stone; Joseph D. Noshpitz, editor-in-chief. New York, N.Y., Basic Books, 1979, 584 pp., $35.50 (4-volume set: $138.00).*

The field of child psychiatry has occasionally been taken to task for its presumed lack of cohesion and structure. It is not surprising, then, that there has been no single volume or set of books until now attempting to cover the ever-widening array of disciplines that make up the field. The recent publication of the *Basic Handbook of Child Psychiatry* belies this notion. This four-volume set is a truly magnificent piece of work. Enormously important in its own right, it will in time become a unifying milestone to child psychiatry in general. Unlike so many recent compendia, this is not a collection of previously published articles but a thoroughly integrated and coordinated effort on the part of Joseph Noshpitz and a group of hard-working co-editors and colleagues who spent more than six years soliciting, collecting, editing, and organizing some 250 separate chapters.

The authors are for the most part well known and distinguished. They represent no one school of thought or point of view; in fact, within these volumes can be found a balanced discussion of virtually every important developmental theory, method of child treatment, and aspect of childhood psychopathology.

The set of books is held together—as is the field of child psychiatry itself—by a developmental approach. Volume 1, perhaps the most ambitious of the four, is really three distinct units. The first 300 pages is a stepwise sequence of reviews of normal biological, psychodynamic, and psychosexual development from conception to late adolescence. The middle section of this volume contains roughly 40 separate chapters describing developmental variations, such as "The Adopted Child," "The Fatherless Child," "The Child Raised Overseas," "The Street Child," and another 10 chapters on children with a variety of major medical handicaps. Many of these submissions are extremely informative essays. The chapter on "Children of Different Ordinal Positions" by Edington and Wilson is particularly outstanding.

The final third of volume 1 is devoted to concepts and techniques of assessment. It is enriched by a number of unusual chapters on automated data systems, computer-assisted diagnostic techniques, and standardized data collection, which a less thorough and farsighted editor might not have thought to include.

The emphasis of volume 2 is on nosology, diagnosis, and descriptive pathology. After a fascinating summary of various diagnostic schemata not in use, the bulk of the volume proceeds to more than 30 separate reviews of the major child psychiatric syndromes, including school phobia, enuresis, habit disorders, minimal brain dysfunction, and childhood psychoses. Many of these are the equivalent of truly first-rate journal articles. The overview of the anorexia nervosa syndrome by Sours is one of the best on this subject I have encountered.

Volume 3 is constructed around theoretical and practical considerations of treatment. Since therapeutics remains perhaps the least agreed on area for all of psychiatry, it is not surprising that this volume contains some of the more controversial offerings. Although one obviously would not purchase every item in the store, the psychopharmacologists, family therapists, educational psychologists, child analysts, behaviorists, and counselors have clearly been given a fine opportunity to display their wares.

The fourth and final volume is addressed to prevention and to current issues in child psychiatry. The latter half of the book is a bit of a potpourri; the chapters tend to be short and choppy, the topics are a little arbitrary and perhaps less "basic" to the field than those of the other volumes, but by and large they make interesting reading.

One danger in volumes of this sort is the tendency to overharsh editing, which reduces the contributions to dry, homogeneous synopses. This set of books, although tightly edited, is encyclopedic in scope but varied in style. For example, witness the freedom (and charm) allowed Robert Stoller in his chapter in volume 2 on gender disorders:

I shall approach my topic in an autobiographical tone; that should bring greater accuracy than an orderly, scholarly presentation. . . . With the optimism that probably sustains other researchers who persist on one subject, I always feel that today's concepts, theories, data, and conclusions are solid and pretty accurate—a pleasant, persistent illusion that keeps one going.

In sum, if a handbook is, as Webster defined it, "a compact reference on some subject," then Dr. Noshpitz' most alarming weakness is in the naming of this work. It is not a single book, and it is not compact. It is in fact approximately 15 pounds of books, clearly to be carried more by shelf than by hand. The point to emphasize, though, is that however misleading the name, the *Basic Handbook of Child Psychiatry* should stand as a basic reference on the shelf of any serious inquirer into the world of children.

DANIEL W. ROSENN, M.D.
Boston, Mass.

Disorders of the Schizophrenic Syndrome, *edited by Leopold Bellak, M.D. New York, N.Y., Basic Books, 1979, 629 pp., $27.50.*

The title of this massive tome is a misnomer and may mislead those unfamiliar with the three preceding volumes in Dr. Bellak's series of 10-year reviews of schizophrenia. His first review, *Dementia Praecox,* appeared in 1948; the second, *Schizophrenia: A Review of the Syndrome,* in 1958; the third, *The Schizophrenic Syndrome,* with Loeb as coeditor, in 1968. The four volumes cover nearly 50 years of developments in research and therapy in a field where breakthroughs always appear to be just over the horizon. Many of the contributors to the present work convey a feeling of muted optimism; although the answers are not quite at hand, they are expected in the next volume, or perhaps in the following one, which will fittingly appear in the year 2000, the start of the new millennium.

Manfred Bleuler contributes a foreword on his 60 years with schizophrenic patients, outlining very briefly his views on etiology and treatment. Dr. Bellak provides "An Idiosyncratic Overview" as an introduction and an epilogue on "What the Clinician Can Do Until the Scientist Comes." Bleuler and Bellak together represent 100 years of clinical experience with schizophrenic patients; their chapters should be read carefully because there are few others who have studied this illness so long or spoken so well about it.

Seven chapters deal with research. Torrey reviews the area of epidemiology, which, as he says, has so far contributed remarkably little but may be beginning to live up to its promise. Meltzer's chapter on biochemical studies is much more substantial and has nearly 700 references. This area has expanded tremendously in the past 10 years. The previous volume in the series did not mention dopamine, platelet monoamine oxidase, endorphins, viruses, Dr. Meltzer's own important work on neuromuscular dysfunction, or the neuroendocrine strategy. Dr. Meltzer concludes with the expectation that the next 10 or 20 years will lead to identification of the majority of the biochemical and neurophysiologic deficits, "providing research support is available at adequate rates." The other chapters on research are as excellent as one might expect from such experts as Cancro on genetic studies, Buchsbaum on neurophysiologic aspects (a chapter that shows the probable heterogeneity of the schizophrenic syndrome), Rabin on psychological functions, Rieder on children at risk, and Lyman Wynne on family studies.

The other major section has 10 chapters on clinical aspects. These are somewhat variable in scope, but for the most part the authors cover their assigned territory well. Carpenter and Strauss discuss diagnostic issues and also deal well with prognosis. Lipton and Burnett survey psychopharmacological treatment, which sadly has advanced little in the past decade. Max Fink reviews ECT and other somatic treatments, finding ECT to be the only one of value. Gunderson individually provides an excellent chapter on individual psychotherapy and joins Loren Mosher to review group, family, milieu, and community support systems. Zwerling writes on community psychiatric treatment, only slightly overlapping Mosher and Gunderson's chapter.

E. James Anthony contributes a short chapter on prevention, an area in which, unfortunately, there is not much to review. Paulina Kernbers provides a selective review on childhood schizophrenia and autism, concluding with the expectation that in the next decade diagnostic subgroups will be further clarified, leading to more effective early intervention and treatment.

Alan Stone contributes the final chapter on legal and ethical developments. Important legal cases have reduced the risk of involuntary confinement, shortened the length of hospitalization, and raised the questions of the right to treatment and the right to refuse treatment. Dr. Stone notes that these developments have made many state hospitals more dangerous and oppressive and led to schizophrenic patients wandering the city streets or living in welfare hotels, "the new back wards of this decade of legal progress."

Schizophrenia will be with us at least until Dr. Bellak's next volume, which we may fervently hope will contain more positive findings. In the meantime we must do what we can "until the scientist comes," although we may seem to be waiting for Godot (or the millennium).

A. ARTHUR SUGERMAN, M.D.
Trenton, N.J.

The Diagnosis and Treatment of Alcoholism, *edited by Jack H. Mendelson, M.D., and Nancy K. Mello, Ph.D. New York, N.Y., McGraw-Hill Book Co., 1979, 385 pp., $17.50.*

The flyleaf of this book promises that it is a definitive but readable "shot in the arm" for all who deal in the area of alcoholism. It is, indeed, comprehensive, but it challenges the reader to some initial aversive conditioning by beginning with a concoction of ill-mixed and confusing definitions and then moving on to an erudite but not necessarily easily swallowed section on anatomy, physiology, and biochemistry. The reader must start absorbing his alcohol almost immediately, with such intervening variables as microsomal ethanol oxidizing systems (MEOS). Once the reader survives these initial onslaughts, he or she is treated to a much "mellower" concoction of research and philosophy.

The best section of the book deals with the social settings and historical perspectives of alcoholism in the United States. This would have been a marvelous introduction to what is clearly a multifaceted approach to a multidimensional subject.

Several themes arise out of all this research. The first is that alcoholism is not a solitary or unidimensional problem and that any approach to alcohol as a problem, or the alcoholic as an individual, must be multifactorial in nature. All of the authors stress these philosophical approaches, whether they are discussing cross-cultural, genetic, or behavioristic elements of alcohol and the alcoholic.

The reader should not be lulled into thinking that this book is the "mixology" manual for the understanding or treatment of alcohol. What the reader will find, however, is a very sobering appraisal of alcoholism as a genetic, medical, biochemical, and social problem. The reader will also come to understand that there are a number of adequate but not definitive treatment methods—disulfiram, inpatient clinics, aversion and other behavior modification techniques—all of which have some specific effectiveness and also some specific limitations and drawbacks. All in all, this is a "fine brew" that with a slightly different mix in the blender could have been a "great brew."

ARON S. WOLF, M.D.
Anchorage, Alaska

Between Life and Death, *edited by Robert Kastenbaum, Ph.D. New York, N.Y., Springer Publishing Co., 1979, 184 pp., $15.50; $9.95 (paper).*

As a person who has spent a considerable amount of professional time working with dying patients, I have given a great deal of thought to the question of whether or not there is life after death. Having admired Elisabeth Kübler-Ross' work with dying patients and watching her evolving interest in and acceptance of the existence of life after death, I wondered whether my own prejudiced point of view would be challenged, supported, or left untouched by the data reported here. It also occurred to me that some of my colleagues working with dying patients seem to have become more philosophical, and I have to admit that working with dying patients has also affected me and my approach to life. It has caused me to think more seriously about establishing priorities.

Does everyone who works with dying patients change? Is the experience of approaching death or apparently dying and being revived so overwhelming that it leads one to reach for a way of expressing that incredible experience and to think of an existence beyond our present knowledge of life? Must we be involved in the world of parapsychology as soon as we approach this area because of the impossibility of using our customary vocabulary and imagery when we talk of death?

These and many other profound questions affected my attitude to reading Kastenbaum's book and certainly do not make me an objective reporter. I hold immense skepticism about a world beyond, but I also have a profound admiration for Dr. Kastenbaum's previous work, and I can report that this book is a sensible, balanced, and very interesting work. It sets the stage for much needed research in the field of investigating the experience of dying.

For the most part the book presents a range of thinking about the world of life beyond. It subjects the data to a sensible analysis leading to further research and possible hypotheses. It explores the phenomena reported by a variety of observers and patients who have approached clinical death and have been revived. The common denominator for all of these reports seems to be a healthy skepticism of anecdotal material, a call for much more critical analysis of experiences of death and the world beyond. The intriguing question of whether or not there is an afterlife has been answered by the response, "not yet proven." "Not yet" obviously implies "could be."

It appears to me that all of the reports of people hovering over their bodies during resuscitation or coming back to life after being pronounced dead can be explained by a human being's need to express this incredible experience in a vocabulary that is not equal to the experience. I feel that an analogous situation could be one of trying to find a way of verbally expressing love, orgasm, or panic. If one were to invite people who have had these experiences to express the feelings involved, they would all come up with a reasonably uniform set of phrases, images, and expressions. Chances are that if the person were French he would use a vocabulary or a set of images reflecting that cultural influence. If he were Mexican he would do the same but use a different set of images and analogies expressing that culture. The reports studied for this book show such cultural differences. Differences are also noted according to degree of education, sophistication, and age. I think, therefore, that extra-body experiences are explainable on the basis of expressions of unusual experiences.

Kastenbaum has put together a reasonably interesting book and one well worth reading for the serious student of emotions. The book is well written, well organized, valuable, and provocative. It is also eminently fair to the material and appeals to a sense of critical and rational thinking rather than to emotions, beliefs, or wishful thinking. I still do not believe in the afterlife, but I enjoy very much being intrigued by the possibility. I certainly would support further exploration of the subject in a serious fashion. Even if we do not find an afterlife, we will most certainly learn about our need for hope and for belief beyond ourselves.

SAMUEL C. KLAGSBRUN, M.D.
Katonah, N.Y.

Sexual Dysfunction: A Behavioural Approach to Causation, Assessment, and Treatment, *by Derek Jehu. New York, N.Y., John Wiley & Sons, 1979, 287 pp., $17.50 (paper).*

This short book is well organized and informative. It is easy to follow because each section and chapter is neatly divided with bold-faced headings and the paragraphs have subheadings. There are many tables, lists, and numbered sets of instructions.

This is the ideal book if one were preparing for an examination. It poses such questions as, What is the most common sexual dysfunction? What can cause sexual dysfunctions? What are the organic factors? What is the influence of previous learning experience? What may be accomplished during therapeutic interviews? What are the specific behavioral procedures in treating sexual dysfunction? It also provides the answers.

The book's value to the practitioner is more introductory than immediately useful. There is a clear, detailed bibliography with 81 specific references to Masters and Johnson and 33 to Helene Kaplan, neither of whom is considered a behaviorist by the author. I expect that anyone reading Derek Jehu's book would turn quickly to Masters and Johnson and

Kaplan if he or she had not already done so. Jehu's book is an excellent advertisement for others.

I found this book quite disturbing, not because of the author, who does a thoroughly competent review of his subject, organizes his material excellently, and writes well within the limits he sets for himself. It is the "behaviorist" approach to sex as he portrays it that is disturbing. It is too much like reading a book on how to repair a bicycle. There is the chapter on the 9 steps in changing a tire and the one on the 14 ways to adjust the gears. Where is mystery, joy, intimacy? There is more emotion in some books on the bicycle.

HARVEY BLUESTONE, M.D.
Bronx, N.Y.

Towards Understanding Relationships: European Monographs in Social Psychology 18, *by Robert A. Hinde; series editor, Henri Tajfel. London, Academic Press (Harcourt Brace Jovanovich), 1979, 359 pp., $36.50; $18.00 (paper).*

Relationships between people are universal, complex, integral, and intricate threads of life. Hinde's newest book meshes candor and a lively prose style as he tackles the study of relationships. He asserts that the book is not an attempt at systematic theory building. He painstakingly analyzes the pivotal concerns of issues and concepts in the quest for a comprehensive basis for describing and classifying dyadic relationships.

Part one, which contains three chapters, not only discusses the obstacles to a science of interpersonal relationships but critically highlights distinctions between the terms "personality" and "relationship." The author scrutinizes the dialectics involved in relationships, the vicissitudes of selective perception, cultural influences, the role of judgment, and the inherent nature, purpose, and limitations of the descriptive process.

Chapters 4–11 make up part two, which extrapolates principles underlying dissonance and balance theories and learning, exchange, and interdependence theories in relationship to content, diversity, qualities, frequency, and patterning of interactions. Validation of relationships in terms of similarity versus difference or reciprocity versus complementarity is made explicit through the use of family examples and extensive research findings. The distinction between deep and casual relationships is punctuated by a very thorough explication of intimacy, interpersonal perception, and commitment. Before concluding this section Hinde raises and answers in the negative a question crucial to the entire mission of the book, namely, "Are the categories of dimensions we have discussed concerned with phenomena at a level of analysis appropriate to the problems that those interested in the dynamics of relationships wish to solve?" (p. 139). He suggests, "Only future research can tell" (p. 140).

The dynamics of interpersonal relationships are the central focus of part three, chapters 12–20. The mutual influences of partners receives protracted attention with almost repetitive illustrations focusing on mother-child interactions. It would seem that this entire section could have been enhanced by reference to cross-cultural dyad relationships, particularly when the author goes to great length to articulate concepts of exchange, interdependence, reward, conflict, and power. The functioning and dysfunctioning brought about by change and stability, boundaries, systems, and subsystems evolves with clarity and valuable insights for personality theorists, social psychologists, and family therapists.

The concluding section of the book weaves factors of time, social pressure, personal constructs, and personality constructs into a terse consideration of development. Issues of initiating and responding carry a decisive message—relationships create identity and socialization patterns. Finally, Hinde debunks the notion that he has complete answers for understanding relationships. The limitations of his quest and the building blocks achieved in knowledge integration are underscored along with the reminder that "formidable obstacles remain to be overcome" (p. 326).

The extensive references, the well-placed diagrams and tables, the concise chapter summaries, and the author's finely attuned sensitivity, timing, and honesty make this a first-rate scholarly work. Although the book is intended primarily for advanced undergraduates, graduates, and research workers, it should appeal to helping professionals, personnel managers, and international agencies concerned with human development.

MAUREEN MCCARTHY, PH.D.
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Books Received

This listing acknowledges the receipt of recent books. Books of particular interest to the readers of this journal will be reviewed as space permits, and copies will be sent to the publishers. Books cannot be returned to the publishers.

The Patient Speaks of Her Mother, 2nd ed., *by Harold A. Abramson, M.D. New York, N.Y., Vantage Press, 1980, 240 pp., $10.00.*

Psychoanalytic Treatment of Schizophrenic, Borderline, and Characterological Disorders, 2nd ed., *by L. Bryce Boyer, M.D., and Peter L. Giovacchini, M.D. New York, N.Y., Jason Aronson, 1980, 415 pp., $30.00.*

The Twenty-Year Phenomenon: Men and Women Talk About the Breakup of Their Long-Term Marriages, *by Jean Brody and Gail Beswick Osborne. New York, N.Y., Simon and Schuster, 1980, 283 pp., $11.95.*

Training of the Developmentally Handicapped Adult: A Practical Guide to Habilitation, *by Roy I. Brown, Ph.D., and E. Anne Hughson, M.Sc. Springfield, Ill., Charles C Thomas, 1980, 191 pp., $14.75.*

Treatment of Psychotic and Neurologically Impaired Children: A Systems Approach, *by David M. Brubakken, Ph.D., Judy A. Derouin, A.C.S.W., and Helen L. Morrison, M.D. New York, N.Y., Van Nostrand Reinhold Co., 1980, 260 pp., $17.50.*

Sexual Variance in Society and History, *by Vern L. Bullough. Chicago, Ill., University of Chicago Press, 1980, 692 pp., $9.95 (paper).*

Feeling Good: The New Mood Therapy, *by David D. Burns, M.D. New York, N.Y., William Morrow and Co., 1980, 385 pp., $12.95.*

Ethology and Nonverbal Communication in Mental Health: An Interdisciplinary Biopsychosocial Exploration, *edited by Samuel A. Corson and Elizabeth O'Leary Corson, with Joyce A. Alexander. Elmsford, N.Y., Pergamon Press, 1980, 268 pp., $41.00.*

Symbolism in Greek Mythology: Human Desire and Its Transformations, *by Paul Diel. Boulder, Colo., Shambhala, 1980, 218 pp., $12.50.*

Gay Is Not Good, *by Frank M. du Mas, Ph.D. Nashville, Tenn., Thomas Nelson, 1980, 320 pp., $11.95.*

Drug Abuse from the Family Perspective: Coping Is a Family Affair, *edited by Barbara Gray Ellis, M.S.S., M.P.H. Rockville, Md., National Institute on Drug Abuse, 1980, 140 pp., no price listed (paper).*

Toward a Definition of Clinical Social Work: Papers from the NASW Invitational Forum on Clinical Social Work, June 7–9, 1979, Denver, Colorado, *edited by Patricia L. Ewalt. Washington, D.C., National Association of Social Workers, 1980, 104 pp., $5.00 (paper).*

The Long Journey Home, *by Carol Ferland. New York, N.Y., Alfred A. Knopf, 1980, 294 pp., $10.95.*

National Drug/Alcohol Collaborative Project: Issues in Multiple Substance Abuse, *edited by Stephen E. Gardner, D.S.W. Rockville, Md., National Institute on Drug Abuse, 1980, 126 pp., no price listed (paper).*

Preventing Mental Illness: Efforts and Attitudes, *by Raymond M. Glasscote, M.A., and Eleanor Kohn, M.S.W., Allan Beigel, M.D., Merrill F. Raber, M.S.W., Nancy Roeske, M.D., Bonnie A. Cox, M.S.W., James B. Raybin, M.D., and Bernard L. Bloom, Ph.D. Washington, D.C., Joint Information Service of the American Psychiatric Association and the Mental Health Association, 1980, 138 pp., $10.00.*

Homosexual Desire, *by Guy Hocquenghem; translated by Daniella Dangoor. London, England, Allison & Busby (New York, N.Y., Schocken Books, distributor), 1980, 140 pp., $9.95; $5.95 (paper).*

Living with Schizophrenia: A Guide for Patients and Their Families, *by Alexander P. Hyde, M.D. Chicago, Ill., Contemporary Books, 1980, 128 pp., $8.95.*

Coping with the Crises in Your Life, *by Edgar N. Jackson, New York, N.Y., Jason Aronson, 1980, 210 pp., $15.00.*

How to Find a Job: A Woman's Handbook, *by Susan J. Jeffers, Ph.D., and Ellen F. Carr, M.B.A. New York, N.Y., Jeffers/Carr Associates, 1980, 62 pp., $2.95 (paper).*

New Perspectives in Abnormal Psychology, *edited by Alan E. Kazdin, Alan S. Bellack, and Michel Hersen. New York, N.Y., Oxford University Press, 1980, 563 pp., $18.95 (includes instructor's manual).*

Internal World and External Reality: Object Relations Theory Applied, *by Otto F. Kernberg, M.D. New York, N.Y., Jason Aronson, 1980, 348 pp., $27.50.*

How to Enjoy Your Life in Spite of It All, *by Ken Keyes, Jr. St. Mary, Ky., Living Love Publications, 1980, 220 pp., $4.95 (paper).*

Becoming a Family Therapist: Developing an Integrated Approach to Working with Families, *by Charles H. Kramer, M.D. New York, N.Y., Human Sciences Press, 1980, 312 pp., $19.95.*

To Live Until We Say Good-Bye, *by Elisabeth Kübler-Ross; photographs by Mal Warshaw. Englewood Cliffs, N.J., Prentice-Hall, 1980, 157 pp., $5.95 (paper).*

Rapprochement: The Critical Subphase of Separation-Individuation, *edited by Ruth F. Lax, Sheldon Bach, and J. Alexis Burland. New York, N.Y., Jason Aronson, 1980, 504 pp., $15.00.*

The Criminal Justice and Mental Health Systems: Conflict and Collusion, *by Kent S. Miller. Cambridge, Mass., Oelgeschlager, Gunn & Hain, 1980, 126 pp., $17.50.*

Practicing Psychotherapy: Basic Techniques and Practical Issues, *by Demund C. Neuhaus, Ph.D., and William Astwood, Ph.D. New York, N.Y., Human Sciences Press, 1980, 203 pp., $16.95.*

Biofeedback: Clinical Applications in Behavioral Medicine, *by David S. Olton and Aaron R. Noonberg. Englewood Cliffs, N.J., Prentice-Hall, 1980, 416 pp., $21.00.*

The Social Consequences of Psychiatric Illness, *edited by Lee N. Robins, Ph.D., Paula J. Clayton, M.D., and John K. Wing, M.D., Ph.D. New York, N.Y., Brunner/Mazel, 1980, 263 pp., $20.00.*

Reconciliations: Inner Peace in an Age of Anxiety, *by Theodore Issac Rubin, M.D. New York, N.Y., Viking Press, 1980, 260 pp., $11.95.*

Abnormal Psychology: The Problem of Maladaptive Behavior, 3rd ed., *by Irwin G. Sarason and Barbara R. Sarason. Englewood Cliffs, N.J., Prentice-Hall, 1980, 555 pp., $19.95.*

The Story of Ruth, *by Morton Schatzman, M.D. New York, N.Y., G.P. Putnam's Sons, 1980, 306 pp., $11.95.*

Child Psychiatry and the Law, *edited by Diane H. Schetky, M.D., and Elissa P. Benedek, M.D. New York, N.Y., Brunner/Mazel, 1980, 286 pp., $22.50.*

Bereavement Counseling: A Multidisciplinary Handbook, *edited by B. Mark Schoenberg. Westport, Conn., Greenwood Press, 1980, 249 pp., $25.00.*

Are You Listening, Doctor? *by Albert Schrut, M.D. Chicago, Ill., Nelson-Hall, 1980, 231 pp., $15.95.*

Sexual Counseling for Ostomates: A Resource Book for Health Care Professionals, *by Ellen A. Shipes, R.N., M.N., and Sally T. Lehr, R.N., M.N.; illustrated by Martha Tarrant. Springfield, Ill., Charles C Thomas, 1980, 96 pp., $7.75 (paper).*

Medicine and Religion: Strategies of Care, *edited by Donald W. Shriver, Jr. Pittsburgh, Pa., University of Pittsburgh Press, 1980, 168 pp., $10.95.*

Psychophysiological Recording, *by Robert M. Stern, William J. Ray, and Christopher M. Davis. New York, N.Y., Oxford University Press, 1980, 234 pp., $14.95; $8.95 (paper).*

The Art of Psychotherapy, *by Anthony Storr. New York, N.Y., Methuen, 1980, 186 pp., $13.95.*

The Extramarital Affair, *by Herbert S. Strean. New York, N.Y., Free Press, 1980, 214 pp., $12.95.*

The Hyperactive Child and the Family: The Complete What-To-Do Handbook, *by John F. Taylor, Ph.D. New York, N.Y., Everest House, 1980, 244 pp., $12.95.*

Schizophrenia and Civilization, *by E. Fuller Torrey, M.D. New York, N.Y., Jason Aronson, 1980, 221 pp., $20.00.*

The Psychosurgery Debate: Scientific, Legal, and Ethical Perspectives, *edited by Elliot S. Valenstein. San Francisco, Calif., W.H. Freeman & Co., 1980, 582 pp., $25.00; $14.00 (paper).*

Parents and Peers in Social Development, *by James Youniss. Chicago, Ill., University of Chicago Press, 1980, 295 pp., $23.00.*

Aging and Mental Disorders: Psychological Approaches to Assessment and Treatment, *by Steven H. Zarit. New York, N.Y., Free Press, 1980, 444 pp., $19.95.*

OFFICIAL ACTIONS

Report of the Secretary: Summary of Meetings of Board of Trustees and Executive Committee May 1979–April 1980

IT IS MY personal and Constitutional privilege as Secretary to report to the membership the actions taken by the Board over the past year. The report is arranged in alphabetical order according to topics; the items presented here do not include many matters that have been referred to appropriate components for further study and recommendation. As prescribed by our Constitution, especially important issues will be clarified at our Annual Meeting and a full report published in the *American Journal of Psychiatry* for permanent reference.

Beginning in June 1979, meetings originally scheduled as Executive Committee meetings were expanded to include the full Board. Thus, Area Trustees were present at each meeting throughout the year. They had greater continuity in their knowledge of issues being considered and are able to inform and be guided by their constituencies.

Many issues discussed by the Board over the past year related to capital and operating expenses and ensuring fiscal stability for the Association. An extraordinary meeting of the Board was held in October 1979 to discuss these issues and to prepare information and options which were presented to the Assembly at subsequent meetings. As recommended by the Assembly, the Board then acted to assess the membership for capital funds needed for the purchase of land for a new headquarters building, and also approved a 25% dues increase as well as a registration fee at the Annual Meeting to offset some of its costs. (A larger dues increase would have been necessary if this registration fee had not been established.) Assembly officers and members, as well as district branch presidents, assisted with an intensive educational campaign to inform the membership about these actions. The Board is extremely gratified with the initial response of the membership: approximately 52% of the members have paid more than $2.2 million toward the assessment. This has eliminated for the present a need to borrow money at high interest rates in order to finalize agreements with the developer. The Board has established an ad hoc committee to review individual requests to waive or reduce the assessment; the President, the Trustees, the Medical Director, and others are responding personally to a number of requests for additional information or to complaints. Your Treasurer's and other reports will contain more specific information about these fiscal issues.

This is an edited version of the report presented by the Secretary to the annual business meeting in San Francisco, Calif., May 5, 1980.

A consulting firm has been hired to assist in developing a long-range plan for the Association, and functional budgeting is being implemented this year. It is expected that these actions will help the Board to make sound fiscal decisions for the Association and will allow us to maintain and initiate programs of benefit to the membership.

The Board has acted on several matters of benefit to individual members, including a reduction in the rates for malpractice insurance for many members and improvements in the major medical plan for members and their families.

In the coming year there will be a focus on stimulating recruitment into the field of psychiatry and to membership in the Association.

I would remind you that every APA member is privileged to attend any session of components of the Association, except for meetings of the Ethics Committee or when a component is in executive session. Your strong support is appreciated; your recommendations for consideration by the Board or other components are most welcome.

The Board took the following actions.

ANNUAL MEETING

1. Voted to continue to present awards at the Convocation and to hold the business meeting and forum immediately after the opening session, beginning with the 1980 Annual Meeting (May '79).

2. Approved guidelines for holding meetings outside the continental United States (post-annual meetings) (June '79). In general, the guidelines noted the usefulness of promoting international exchange; suggested that post-annual meetings be held every other year, with some flexibility to accommodate to special locations or invitations; encouraged planning as far in advance as possible; noted the need for enough psychiatrists in the host country to assist in organizing the meeting; noted that cost factors should be considered, that such trips should have explicit professional and educational objectives, and that there is a direct line of staff responsibility regarding costs when joint meetings are held.

3. Approved charging a registration fee for members and guests attending the Annual Meeting to support much of the cost of these meetings; in turn, this would offset the amount needed for an increase in member dues (Sept. '79).

4. Approved a three-year trial of providing day care at Annual Meetings with the expectation that the program would become self-sustaining. Authorized $5,000 in the 1980 budget to support the program (Dec. '79).

5. Voted to schedule an extraordinary session at the 1980 Annual Meeting for a discussion of ERA and the relationship between mental health and equal rights; and approved the following statement to be read at the beginning of the opening session on Monday, May 5, and to be further publicized: "The American Psychiatric Association is on record as being in favor of 1) equal rights for women and 2) the Equal Rights Amendment. In an attempt to further these goals, the Board and the Assembly have endorsed the boycotting of states which have not ratified the ERA (by not holding Annual Meetings in these states). The recent membership referendum overturned the boycott policy. To address these issues, the American Psychiatric Association will hold an extraordinary meeting of the membership. . . . The Board urges your attendance" (May '80).

BUDGET/FISCAL

1. Voted to adopt a functional budgeting format, utilizing decision packages, effective 1981, to replace the existing exclusive departmental budgetary approach; authorized pilot projects in certain departments to be determined by the Medical Director, or on certain major decision issues, to utilize functional budgeting format during the remainder of the 1980 budget year; encouraged departments and components to include at least minimal decision packages in submitting budgets for 1980 (June '79).

2. Approved the 1980 budget of $8.8 million, which included income from a registration fee for members and guests attending the Annual Meeting to offset some of its cost, as well as a 25% dues increase (Dec. '79).

3. Approved a one-time assessment of the membership for capital funds to support purchase of land on which to build a new headquarters for the Association. Voted to proceed with plans for a Voluntary Fund Drive for additional capital funds for the building project (Oct. '79).

4. Established an Ad Hoc Committee to Review Requests to Reduce or Waive the Assessment and clarified policies regarding assessment and approved guidelines for waiver of the assessment (Feb. '80).

5. Established a Resource Development Committee to broaden the scope of present efforts to seek outside funding and to anticipate potential new programs and additional sources of funds.

6. Authorized the Joint Commission on Public Affairs to seek competitive bids from major publishers to produce a series of books on psychiatric topics of wide public interest. The publisher would produce and market the books and pay appropriate royalties to APA (Feb. '80).

7. Approved the following timetable for the annual budget process: 1) In April the Board shall establish a maximum ceiling on total expenditures, a maximum revenue to be realized by dues collection, and revenue requirements other than dues collection. 2) In September the Board shall approve major additions or deletions of programs. 3) In September the Board shall be provided with a report on progress and on any problems in meeting established limits. 4) At any regular meeting of the Board or its Executive Committee, the Medical Director or the Budget Committee may request a change in one or more of the established limits. 5) In December the Board shall review the annual budget to determine whether the limits established by the Board have been met.

CENTRAL OFFICE

1. In June, September, October, and December the Board heard reports from the Building Committee about the progress of plans to purchase land and enter into contractual arrangements with a developer to build a new headquarters building for the Association.

Authorized legal counsel to review and approve all documents related to this joint effort with the developer and to empower the Medical Director to act as Attorney-in-Fact to execute these documents on recommendation from legal counsel (Feb. '80).

2. Authorized construction of office space in the Museum Building and up to $7,000 from the Reserve for Future Expansion and Development to cover the cost; this new space will be used for staff support for the Annual Meetings.

3. Authorized disbursements from the Reserve for Future Expansion and Development account for capital improvements to the present Central Office buildings: $4,000 for purchase and installation of carpeting; $3,000 to install automatic temperature controls for night setback as required by law; and $6,175 to convert the furnace to gas rather than oil, which will result in significant savings in fuel costs (Sept. '79).

4. Approved in principle hiring a part-time, nonstaff APA member to serve as Editor of the *Hospital & Community Psychiatry* journal and adding six members to its Editorial Board (the Editor and the Board would then operate in a manner similar to the *American Journal of Psychiatry*) (Dec. '79).

5. Approved funding for a Deputy Editor for the *American Journal of Psychiatry* (Dec. '79) and appointed Dr. Morris A. Lipton to this position (Feb. '80).

COMPONENTS

1. A number of components were established, terminated, or transferred throughout the year: a) On May 17, 1979, the Ad Hoc Committee to Assist in Formulating Contingency Plans for the Results of the 1979 Election, the Ad Hoc Committee for the President's Commission on Mental Health, and the Ad Hoc Committee to Prepare the Board's Statement on Proposed (JCC) Amendments to the Constitution and By-Laws on the 1979 Ballot were terminated. The Ad Hoc Committees on Election Procedures, Implementation of the 1976 Referenda on Psychiatric Certification, Chronic Mental Patient, Civil Commitment, Long-Range Planning, and To Study the Council and Component Structure and Their Relationship to the Board and Assembly were continued. The Commission on Judicial Action (with staggered terms for members) and the Commission on Standards of Practice and Third-Party Payment were also continued for another five years. b) In June 1979, the Board established a new Council on Aging and approved charges for several of its task forces, which will seek outside funding to support much of their work. The final report of the Ad Hoc Committee on the 1976 Referenda on Certification was received at this meeting; its recommendations, together with those of the Council on Medical Education and Career Development, were transmitted to the American Board of Psychiatry and Neurology, publicized in *Psychiatric News*, and discussed by other components, including the Assembly; the ad hoc committee was discharged with thanks. A Committee for the Protection of Human Subjects in Psychiatric Research was

established within the Council on Research and Development, replacing a former task force on the topic.

Also in June the Council on Emerging Issues was terminated; a Committee on Emerging Issues was established within the Council on National Affairs. Components administratively housed within the former council were transferred to other councils. The Committee on Abuse and Misuse of Psychiatry in the United States was transferred to the Council on National Affairs; a new charge is being developed by the Council, and members have not yet been appointed. c) In September 1979, the Board established a Committee on Abuse and Misuse of Psychiatry and Psychiatrists within the Council on International Affairs and approved its charge. d) In December 1979, the Board established a Committee on Alcoholism and a Committee on State Hospitals within the Council on Mental Health Services. An Ad Hoc Committee to Review Requests to Waive or Reduce the Assessment was also established at this meeting. An Editorial Advisory Panel for *Psychiatric News* was established for one year; there will be liaison with the present Committee to Review *Psychiatric News*.

2. In September 1979, the Board asked all Councils and components to review their activities and determine if some programs or projects might be supported by outside funding.

3. The 1980 fall meetings of Councils and components were originally scheduled to be held in one hotel in Los Angeles. However, the Budget Committee found that at least $70,000 could be saved if these meetings were held on the East Coast. In December 1979, the Board voted to hold these meetings September 24–27, 1980, in Washington, D.C. The meetings will be accommodated in three hotels, and Central Office space will also be used.

4. The Ad Hoc Committee to Study Councils and Components recommended that the Reference Committee be reconstituted. The Assembly and the Reference Committee endorsed the concept of a new Joint Reference Committee (JRC). In December 1979, the Board approved the composition of the JRC to include the President-Elect as Chair, the Speaker-Elect as Vice Chair, two members from the Board and two members from the Assembly, and the Medical Director as voting members. Council chairs and appropriate commission chairs will report to the JRC and be ex officio members without vote. In February 1980, the Board approved an amendment to Chapter 6, Section 7 of the By-Laws (as prepared by the Committee on Constitution and By-Laws) to be on the 1981 ballot.

5. In June 1979, the Board authorized changing the name of a new Council to Council on Governmental Policy and Law. In December 1979, this Council stressed that additional staff (an attorney to draft legislation and regulations) was needed. In February 1980, the Board authorized funding of $17,500 from its Contingency Fund; Dr. Stone will contribute an additional $7,500 from his Presidential Fund to hire someone (perhaps a law student); the first effort will be development of a model commitment law.

6. *DSM-III*. a) In June 1979, the Board authorized publication of *DSM-III* and initially recommended that the classification system be implemented by January 1, 1980; however, when it was found in December that a longer period of time was necessary for individuals to receive the manual and be trained in its use, the Board voted to recommend that it be implemented no later than July 1, 1980. In September the Board approved guidelines for granting permission to reprint under the *DSM-III* copyright. The basic policy is restrictive; the Medical Director is authorized to grant permission to reprint segments but no major section may be reprinted, and the Diagnostic Classification may be reprinted on approval. The Board will approve any requests to translate the manual into other languages. In December 1979 the Board authorized the Council on National Affairs to develop a plan to translate the manual into Spanish. b) To help offset the cost of developing and printing *DSM-III*, the Board at several meetings considered ways to publish educational material related to the manual. An ad hoc committee of the Board was established in December to study these issues. On recommendation of this ad hoc committee, the Board voted in February 1980 to request that the President appoint an editor to review material written by several organizations, develop an introduction and outline, provide guidelines for using some of the present material or developing new chapters, discuss the problems with the representatives of the various contributing organizations, and report back to the Board.

A task force on educational programs was established within the Council on Medical Education and Career Development; conferences are planned. In September 1979, the Board authorized a working relationship between APA and Roche Laboratories, with Audio Visual Medical Marketing playing an intermediary/coordinating role to support educational conferences. The Board also authorized APA's cosponsorship with Columbia University of a course which was held in December in New York City.

7. In February 1980, the Board voted to have APA seek formal liaison with or representation on the ADAMHA/WHO committees for the development of *ICD-10* (which will relate to *DSM-IV*).

8. In December 1979, the Board authorized publishing the 7th edition of the Information Bulletin for Applicants, 1980 for accreditation in administrative psychiatry that includes a revised fee schedule: $300 is charged for the initial application, $150 to repeat one subject, and $200 to repeat two subjects.

9. In June 1979, the Board voted to hold meetings of the full Board in cases when Executive Committee meetings had been scheduled; thus all Areas are represented by their Trustees at all meetings during the pilot program of one year.

10. In December 1979, the Board approved the attendance of a representative of the Falk Fellows at meetings of the Board.

11. On recommendation of a Task Force on Directories, the Council on Internal Organization, and the Reference Committee, the Board approved publishing a single directory every two years (including some aspects of the *Biographical Directory* but reduced in size), with the understanding that the Board would review specifics of the categories to be included. This directory would be published in-house, with the expectation that it will produce more income than if published elsewhere.

12. In December 1979, the Board approved recommendations of the Task Force on the Impaired Physician which call for encouraging AMA and other organizations to establish policies ensuring psychiatric services for medical students; authorized the task force to develop a questionnaire to survey the district branches about methods of identifying and helping impaired physicians and to explore ways in which hospital and medical staffs can be encouraged to develop techniques for aiding impaired physicians. When problems and issues are clarified, the task force will develop informa-

tive resources such as a publication or video tapes. There will be collaboration with the American Hospital Association.

13. At several meetings the Board discussed issues related to projects providing peer review of psychiatric cases.

In June the Board was informed that the initial case review of CHAMPUS patients hospitalized over 180 days had begun following the endorsement of the project by the Assembly and the Board in May 1979. The Board approved the Assembly's recommendations that the current productive negotiations between APA and CHAMPUS continue to be pursued vigorously while a more acceptable balance between protection of privacy and need for accountability is achieved, and that these negotiations address themselves to concerns expressed by the district branches, committees, and individuals. It was reported at this meeting that CHAMPUS has agreed to no retroactive denial on inpatient care; there will be no retroactive review; patients and providers will be notified 14 days in advance of any action to deny benefits. Two Assembly liaisons were appointed to the Ad Hoc Committee on Health Insurance for Mental Disorders, which reviews this project and reports to the Board.

In April 1979, the Board authorized staff to explore a service that would provide Aetna with peer review of psychiatric cases covered by that company. In June the Board authorized the APA Office of Peer Review to proceed with the Aetna Psychiatric Peer Review Project for six months, at which time an analysis and report would be furnished to the appropriate components.

In September the Board received a report of the Ad Hoc Committee on Health Insurance for Mental Disorders and endorsed the Committee on Confidentiality's recommendation to request Aetna to return the mental health treatment form to the office of the attending physician after peer review has been accomplished; the Board reaffirmed its previous stance that APA will not enter into any new contracts at the national level until issues can be further clarified and voted not to extend the Aetna program beyond December 1979 without further evaluation. The Board indicated to the district branches that it will not interfere with local arrangements made with insurance carriers.

In December 1979, after hearing a detailed report from the Committee on Peer Review and recommendations from the Ad Hoc Commitee on Health Insurance for Mental Disorders, the Board authorized the APA Office of Peer Review to proceed with the APA/Aetna Project with the understanding that there will be careful monitoring by, and progress reports to, the Board and Assembly. Staff was authorized to respond to requests from other insurance companies to negotiate similar projects; these would need further action by the Board and review by the Assembly. The Board also acted on recommendations to be used in negotiations with third-party payers. The Board has continued to discuss how involved the APA should be in providing peer review and has asked staff to seek ways of providing indemnity and to explore the feasibility of establishing a separate corporation to handle peer review and related contracts.

15. The Board was advised in December 1979 that the Committee on Peer Review is revising the *Manual for Psychiatric Peer Review* and asked the committee to send first drafts to the district branches for comments by the branches' component on peer review.

16. In September 1979, the Board approved by-laws for a political action committee; the Assembly endorsed the Board's action in October; in December the Board approved a change in name to American Psychiatric Political Action Committee and approved the membership of its first board of directors, which will first meet in April 1980.

17. In December the Board approved guidelines for accepting financial support from pharmaceutical companies: APA will control the manner in which funds are spent and the content of any educational program and materials used in such a program; have responsibility for determining standards of taste and ethics for all aspects of programs receiving pharmaceutical support; and will acknowledge receipt of such support. A group of members and staff will meet with representatives of the pharmaceutical industry to discuss these guidelines during the 1980 Annual Meeting.

18. Approved entering into contractual arrangements with Mutual of New York for a re-insurance contract to extend psychiatric inpatient benefits of a lifetime maximum of $250,000; to extend the major medical plan to provide a supplement to Medicare; authorized a reduction in the life insurance premiums; and authorized a reduction in premiums for accidental death and dismemberment (Dec. '79).

19. Approved reduced rates to APA members as proposed by Chubb for professional liability insurance as of May 1, 1980; authorized the Ad Hoc Committee to Review APA's Professional Liability Insurance to continue negotiations with Chubb for even better rates and to investigate with other brokers the possibility of better plans for APA members (Dec. '79).

20. In April 1979, the Board was informed of the favorable decision in the Virginia suit, considered one of the most positive decisions for the practice of psychiatry in recent years. Because many local legal actions have a national impact, the Board voted to suggest to the district branches and Area Councils that, when anticipating litigation, the Commission on Judicial Action be asked to review the issue. (In September 1979, the Board was informed that the psychologists will appeal the Virginia case; the APA filed an amicus curiae brief in this appeal.)

21. In December 1979, the Board authorized a contribution of $8,000 to the California Psychiatric Society to assist with legal fees in Jamison v. Farabee, a right-to-refuse-treatment case in California.

22. On recommendation of the Commission on Judicial Action, the Board (June '79) authorized the Commission to participate in Project Release v. Prevost, a federal case that involves a full-scale effort to criminalize the New York commitment process; in December 1979, the Board authorized APA to file amicus briefs in Rennie v. Klein and Rogers v. Okin.

23. Authorized hiring the firm of Coopers and Lybrand as consultants to APA in preparing a long-range plan for the Association and established a component to work with the consultants (Dec. '79).

24. Authorized the Council on Medical Education and Career Development and the Office of Education to develop a plan for an audiovisual self-study program (for CME credits), derived in part from the content of the Annual Meetings, with the understanding that an outside contractor would be used for taping but that the program would be under APA control and ownership (Dec. '79).

25. In June 1979, the Board established an ad hoc committee to develop a position statement on patient package inserts; this ad hoc committee reviewed material developed by several other components. In September 1979, the Board

approved a position recommended by the ad hoc committee, contingent on approval by the Assembly. In October the Assembly revised a statement proposed by the Committee on Women to be compatible with the position approved by the Board in December and which will be published in the *American Journal of Psychiatry*.

26. In December 1979, the Board authorized publication of two task force reports: Task Force on Biofeedback and Task Force on Tardive Dyskinesia.

27. In December 1979, the Board approved a position statement on Teenage Parents and Their Children that includes suggested elements for effective programs.

28. In February 1980, the Board approved publication of the report of the Task Force on Alternative Patterns of Organization of State Governments to Provide Mental Health Services to Children and Adolescents and recommended that it be offered to the *Hospital & Community Psychiatry* journal for publication.

ELECTIONS

1. In June 1979, the Board referred the Area I action paper as approved by the Assembly to the Ad Hoc Committee on Election Procedures for revision of the section in the *Operations Manual* concerned with election procedures and campaign guidelines. In September the Board approved the new guidelines contingent on approval by the Assembly. In October the Assembly approved the election guidelines and their implementation in the 1980 election. In December 1979, the Board approved a revision in the *Operations Manual* that relates these new guidelines to operating policies.

2. Revised the *Operations Manual* to provide early appointment of the Nominating Committee, so that members could contact their Area representatives on the committee earlier in the process (Dec. '79).

3. In December 1979, the Board was notified of the receipt of a referendum on ERA and national meeting sites; signatures of more than 200 voting members had been verified; the referendum was on the 1980 ballot.

4. In December 1979, the Board approved two amendments to the Constitution and By-Laws regarding the composition of the Assembly (Chapter 7.1) and clarification of Chapter 8.6 assessments. In February 1980, the Board approved an amendment to Chapter 6.1 to change the composition of the Reference Committee. All of these amendments will be read to the membership at the Annual Meeting in 1980 and appear on the ballot in 1981.

ETHICS

1. Approved revising Section 9 of the *Principles of Medical Ethics with Annotations Especially Applicable to Psychiatry* as follows: "Ethical considerations in medical practice preclude the psychiatric evaluation of any adult charged with criminal acts, prior to access to, or availability of, legal counsel. The only exception is the rendering of care to the person for the sole purpose of medical treatment" (Sept. '79). (The Assembly approved this annotation in October 1979.)

2. Approved a revision to the *Operations Manual:* "When a member is under investigation for an ethical violation, he/she shall not be permitted to resign until all charges of unprofessional conduct have been resolved" (Sept. '79).

3. In December 1979, approved publication of a collation of the *Opinions of the Ethics Committee*.

INTERNATIONAL ACTIVITIES

1. Approved APA's joining with the Gerontological Society and the American Geriatric Society in endorsing the visit to the United States of a study group on diseases of the elderly from the People's Republic of China (Sept. '79).

2. Voted to publish the Report of the Committee to Visit South Africa in the *American Journal of Psychiatry* (June '79).

3. Sent a telegram to President Carter, the World Health Organization, and the United Nations deploring the situation in Cambodia and offering APA's assistance (Dec. '79).

4. Elected Dr. Semyon Gluzman, an imprisoned Soviet dissident, to Distinguished Fellowship (Dec. '79).

5. Throughout the year the Board heard plans for the Second Pacific Congress of Psychiatry, to be held after the Annual Meeting in May 1980, as well as for the Joint APA (Area II)/WPA Regional Symposium, to be held October 30–November 8, 1981, in New York City.

6. After the appearance of a *New York Times* article, sent a cablegram to Professor Snezhnevsky, Director of Psychiatry, Institute of Medical Sciences, U.S.S.R., asking for repudiation of an allegation that Academician Andrei D. Sakharov was threatened with "commitment to a psychiatric hospital" because of his political dissidence. The cable further stated that "we hope this report is in error since, if true, it would be taken as new evidence for the claim that there is political abuse of psychiatry in the Soviet Union."

LIAISON ACTIVITIES

1. Approved APA's becoming a participating organization of the Professional and Technical Advisory Committee for the Long-Term Care Program of the Joint Commission on Accreditation of Hospitals. In September 1979, the Board did not favor providing financial support for a new Accreditation Council on Mental Retardation since APA had accepted the JCAH invitation to serve on a Professional and Technical Advisory Committee for Services for Mentally Retarded and Other Developmentally Disabled Persons Program.

In December 1979, the Board endorsed the Assembly's recommendation that APA's representatives to the JCAH committee responsible for formulating the consolidated standards be instructed to inform the committee immediately that APA cannot accept these standards and that revised standards should be formulated.

In February 1980, the Board voted to instruct APA's representatives to the JCAH PTACs for the Hospital Accreditation Program, Psychiatric Facilities, and Long-Term Care to continue to insist that there be a provision for a medical staff in the accreditation standards for all psychiatric facilities, but that this provision would not preclude the organization of a clinical staff. It is the opinion of the APA Board that if there is no medical staff, a facility should not be considered a *psychiatric* facility; that institutions should be able to elect to have a medical staff if they wish to be accredited under the

standards for psychiatric facilities; and that if they do not wish to have a medical staff, they should be accredited under standards designed for other types of facilities. The Board also asked APA's representatives to the JCAH PTACs to report regularly to the Council on Mental Health Services.

2. Approved the establishment of formal liaison between the American Academy of Child Psychiatry and APA (May '79).

3. Authorized staff to cooperate with the United Mine Workers in a study of psychiatric disabilities following a mine accident (May '79).

4. Authorized staff to contract with Aetna and the United Auto Workers for a meeting to discuss chronic psychiatric disability.

5. Voted to submit only one nominee for each vacancy on the American Board of Psychiatry and Neurology (Dec. '79).

6. Voted to accept an invitation to organizational membership on the President's Committee on Employment of the Handicapped at no cost to APA, as recommended by the Committee on Rehabilitation (June '79).

7. Endorsed a resolution of the American Association for the Advancement of Science on science in-state programs (June '79).

8. Affirmed its policy that APA will not cosponsor meetings unless the Association is actively involved in the planning process and that such cosponsorship is usually limited to meetings of related organizations (June '79).

9. Supported the application of the American Board of Qualification in Electroencephalography to the American Board of Psychiatry and Neurology for establishment of special certification in EEG (June '79).

10. Asked the American Medical Association to reconsider its action to withdraw from the Liaison Committee on Continuing Medical Education and endorsed having one accrediting body for Continuing Medical Education (Sept. '79).

11. Approved storing the files of the National Commission on Confidentiality of Health Records at the APA Central Office and providing minimal staff support to the Commission (Sept. '79).

12. Strongly opposed HEW's approving the National Accreditation Association and American Examining Board of Psychoanalysis, Inc., as a nationally recognized accrediting organization (Sept. '79).

13. Endorsed comments on new regulations on day care (as prepared by the Council on Children, Adolescents and Their Families) and sent them to DHEW (Sept. '79).

14. Approved joining with other major organizations to honor Mr. Norman Lourie on his retirement as Secretary of Welfare for the State of Pennsylvania (Sept. '79).

15. In December 1979, the Board authorized the Committee on Medical Education to develop programs and materials related to recruitment of medical students in collaboration with the American Medical Students Association; official liaison with AMSA will be through the APA committee.

16. Approved liaison with the American Academy of Family Physicians, the American Academy of Pediatrics, and the American College of Physicians (Dec. '79).

17. Approved in principle a working document, "Psychiatry in Training for Primary Care Specialties," to be used in further discussion with the American Academy of Family Physicians and the American College of Physicians, and authorized the Committee on Psychiatry and Primary Care Education to collaborate with representatives of these organizations to develop a final document (Dec. '79).

18. Voted to suggest to the Residency Review Committee, the American Association of Directors of Residency Training Programs, and the American Association of Chairmen of Departments of Psychiatry that each residency program develop a written procedure to be followed when checking the credentials of residents requesting transfer; and further, that the RRC consider including this requirement for procedures (without defining them specifically) in the *Essentials and Guide for Psychiatry Residency Training Programs* (Dec. '79).

19. Endorsed a document prepared by the National Cancer Institute, "Coping with Cancer: A Guide for the Health Professional," at no cost to the Association (Dec. '79). (This is part of a larger NCI program to provide information on the psychosocial aspects of cancer to patients and their families, both directly and through health professionals.)

20. Approved APA's having organizational membership on the U.S. Council for the International Year of the Disabled (1981) (Dec. '79).

21. Authorized collaboration with the AMA in producing a *Mini CPT-IV* for psychiatrists (Dec. '79).

22. Approved guidelines prepared by the Section Council on Psychiatry regarding appointments to the Section Council and APA's delegates to the AMA House of Delegates (Dec. '79).

23. Approved APA's becoming a member of the Federation of Organizations for Professional Women, with dues of $200 per year. The locus for the activity and liaison will be the Committee on Women, with Dr. Spurlock attending the meetings which are held in Washington, D.C.

24. Endorsed the American Electroencephalographic Society Guidelines for EEG as recommended by the Councils on Medical Education and Research and Development (Feb. '80).

25. The Council of Medical Specialty Societies needed to increase dues to support ongoing programs and increased activities. In February 1980, the Board approved an increase in APA's dues from $1.75 to $1.85 per voting APA member. APA's representatives have organizational responsibilities on CMSS.

26. Approved APA's cosponsoring sessions at the 1981 AMA Winter Scientific Meeting at no cost to APA. Dr. Shervert Frazier, a member of the AMA Council on Continuing Physican Education, will be responsible for program development in concert with APA staff Dr. Henry Work and Ms. Carol Davis.

27. Approved APA's cosponsorship of a National Institute for Long-Term Care, which is tentatively scheduled for September 26-27, 1980, in Washington, D.C.

MEMBERSHIP

1. On recommendation of the Assembly and the Membership Committee, approved allowing a grace period of at least one year before any member who has not complied with Continuing Medical Education requirements is dropped (Dec. '79).

2. Supported having the Membership Committee hold workshops to educate the district branches about Fellowship procedures at the Annual Meetings, orientation sessions for district branch presidents-elect, and perhaps a presentation to the Assembly, rather than holding regional workshops (Dec. '79).

3. Approved criteria guidelines for consideration of potential APA member benefit programs (Dec. '79).

4. In the 1979 election, members voted to increase the requirement as General Member from 5 to 8 years to be eligible for consideration for Fellowship. In February 1980, the Board voted a one-year phase-in implementation of this amendment. For the 1980 nomination process, members with six or more years as General Member will be eligible for consideration as Fellowship nominees, including those who had already been nominated by their branch.

5. Considered routine membership matters at each meeting and took some or all of the following kinds of actions: authorized dropping from APA membership those members who had resigned from or been dropped by their district branches for nonpayment of dues; authorized administrative reinstatement of those members who paid their dues arrearages and returned to good standing in both the branch and the national Association; approved transfers and acceptances or denied them if circumstances indicated; accepted applications and resignations; granted dues waivers when warranted, or Inactive member status; approved membership status changes from one category to another; acted on recommendations for Distinguished or Honorary Fellows (May, June, Sept., Dec. '79; Feb., April '80).

H. KEITH H. BRODIE, M.D.

Report of the Treasurer

General. This report is prepared from audited figures for the fiscal year which ended December 31, 1979. Data presented also appear in the auditor's annual report. A statement of financial condition is shown in table 1, and table 2 reflects revenues and costs for the year. This report, together with the tables, will provide the membership with the information they need to assess the operation and financial condition of the Association. In the interest of complete and accurate reporting of fiscal information, all assessment receipts, as well as the investment of such receipts, have been accounted for separately. Therefore, table 1 includes a new fund designation, the Building Fund, which displays the assets and liabilities associated with the assessment and fund drive.

Fiscal status. Execution of the fiscal year 1979 budget resulted in income of $8,635,808 (1/2 percent below the budget estimate) compared with expenses of $8,585,215 (1 percent below budgeted expenditures). This resulted in a net surplus from general fund operations amounting to $50,593. The surplus of income over expenses for 1979 basically stems from teamwork among APA policy makers and staff to overcome obstacles posed by inflation and recession through bolstering income and minimizing expenses. Income was strengthened by *DSM-III* sales of $560,000, revenues from Continuing Medical Education courses offered during the annual meeting that exceeded budgetary estimates, a strong grant/contract program that produced outside support in excess of $1,300,000 for needed programs, and increases in rate schedules for the products and services provided by APA to the public. Expenses were controlled during 1979 by the successful efforts of component chairs and department heads to keep expenditures within budget allocations and specific cost containment efforts by the leadership and staff, including the elimination of three existing positions during the year.

Investments. The stated investment policy of the Association is to "employ sound investment vehicles affording the maximum return consonant with safety of capital, i.e., the type of investment a prudent individual would seek." With this in mind, safety of capital has been identified as the first objective of the investment program.

Total investment income for the year amounted to $200,943. Of this amount, $23,053 stemmed from savings and loan certificates (reflected in cash and equivalents in table 1). Earnings from U.S. Treasury bills, short-term bonds, and short-term commercial bank certificates of deposit amounted to $67,017. These investments represent the general fund working capital of the Association and are invested in accordance with requirements in the annual cash budget. Balances ranged from a high of $1,200,000 in midyear to a low of $150,000 at year end. This experience reflected an orderly execution of the cash budget and maximum return on short-term holdings within the conservative bounds established by fiscal policy.

Pursuant to the sale of the Connecticut Avenue and Hillyer Place properties for $1,120,000, APA received 9% notes receivable from the buyer in the amount of $1,070,000 on February 28, 1979. During the year the Association received payments on the balance in the amount of $200,000. The outstanding balance as of December 31, 1979, was $870,000. Interest income on these notes for the year was $69,719.

APA was fortunate during 1979 with respect to the equities portion of its portfolio. Total fund return on equities during the year amounted to 23.7% as compared with 10.4% for the Dow-Jones Industrial Average and 18.4% for the Standard & Poor "500." The total fund investment return on the overall portfolio since late 1975 (the time of the formation of the Investment Advisory Committee) has been 41.5 percent.

The quality of the equities portion of the portfolio has improved markedly since the inception of the Investment Advisory Committee. The committee inherited a stock portfolio in which the original cost of securities was greater than the corresponding market value. The committee has continually worked to achieve an appreciation of securities, with minimum risk, in order to bring the market value of securities above the historical costs. The committee's steady performance led to the achievement of this goal as of Dec. 31, 1979.

Status of reserves. The Association averted the need for an increase in member dues during 1978 and 1979 by utilizing surplus funds realized during 1976 and 1977. In February 1977 the Executive Committee of the Board of Trustees approved the establishment of the Reserve for Stabilization of Member Dues. The purpose of this action was to develop a reserve account from which sufficient funds could be transferred to operating income for each of three years (1977, 1978, and 1979) in order to meet pressures for increases in operating expenses and thus preclude the need for a dues increase until 1980. At the time this reserve account was established, APA anticipated that an extremely high level of surplus funds would be generated from operations during 1976 and possibly during 1977. This expectation was based largely on the Board's intention to change its fiscal year from April–March to a calendar-year basis, effective January 1, 1977. The surplus of income over expenses for 1976 amounted to $833,704. A portion of this surplus ($579,075) was placed in the Reserve for Stabilization of Member Dues and the remainder was allocated to the Reserve for Future Expansion and Development ($254,629).

An additional surplus of $507,302 was realized from 1977 operations. Of this amount, $279,650 was placed in the Reserve for Stabilization of Member Dues, $225,371 went to the Reserve for Future Expansion and Development, and $2,281 was placed in Unrestricted Surplus. As of December 1, 1978, the balances of these three reserve accounts were as follows: Reserve for Stabilization of Member Dues, $316,512; Reserve for Future Expansion and Development, $1,980,000; and Unrestricted Surplus, $260,009.

This is an edited version of the report presented by the Treasurer to the annual business meeting in San Francisco, Calif., May 5, 1980.

TABLE 1
Comparison of Fiscal Years 1978 and 1979

Item	1978	1979
ASSETS		
General fund		
Cash and equivalents	$ 335,000	$ 268,291
Accounts receivable	652,182	616,015
Accrued interest receivable	7,711	6,004
Deferred charges and prepaid expenses	217,342	261,933
Marketable securities		
Stocks and bonds at cost (market value $504,036)	—	501,990
Stocks and bonds at quoted market value	488,394	—
U.S. Treasury Bills at cost	311,926	133,250
Due from Building fund	—	120,467
Notes and advances receivable from APMA[a]	1,757,085	1,757,085
Less valuation reserve	(1,757,085)	(1,757,085)
Notes receivable, sale of property	—	870,000
Land and building, Central Office, at nominal value	1	1
Furniture and equipment, Central Office, at nominal value	1	1
Land and building, new headquarters, at cost	1,113,886	—
Deduct		
Cash and marketable securities allocated to restricted fund	(305,189)	(285,881)
Total general fund	2,821,254	2,492,071
Building fund		
Cash and equivalents	—	$ 31,709
Accrued interest receivable	—	5,907
Deferred charges and prepaid expenses	—	120,467
Marketable securities		
Repurchase agreements held at bank at cost	—	1,250,000
U.S. Treasury Bills at cost	—	97,775
Total building fund	—	1,505,858
Restricted fund		
Cash and marketable securities allocated from general fund	305,189	285,881
Total restricted fund	305,189	285,881
Total assets	3,126,443	4,283,810
LIABILITIES		
General fund		
Accounts payable and accrued expenses	270,965	442,920
Due to district branches	31,014	1,119
Deferred income	5,125	51,361
Reserve for Stabilization of Future Dues	607,039	—
Reserve for Future Expansion and Development	1,689,473	1,682,473
Less valuation reserve, marketable securities	(45,967)	—
Unrestricted surplus		
At beginning of year	260,009	263,605
Net change during year ended	3,596	50,593
At end of year	263,605	314,198
Total general fund	2,821,254	2,492,071
Building fund		
Due to general fund	—	120,467
Deferred credits, building fund receipts	—	—
Assessment payments received	—	1,347,775
Voluntary fund drive receipts, less expenses	—	31,709
Interest earned	—	5,907
Total building fund	—	1,505,858
Restricted funds		
Principle balances of restricted funds	305,189	285,881
Total restricted funds	305,189	285,881
Total liabilities	3,126,443	4,283,810

[a]American Psychiatric Museum Association, Inc.

Although the Reserve for Stabilization of Member Dues contained $316,512 in early December 1978, an amount of $607,039 from this source was needed for transfer into income for 1979 operations in order to balance the budget. Had this fact been anticipated in earlier years, a larger amount of the surplus funds generated from 1976 and 1977 operations could have been placed into the Reserve for Stabilization of Member Dues at that time. Therefore, the Board of Trustees, during its December 9–10, 1978, meeting, approved the transfer of $290,527 from the Reserve for Future Expansion and Development to the Reserve for Stabilization of Member Dues, which brought the total of that reserve account to $607,039. The Board further approved that this total amount of $607,039 be transferred from the Reserve for Stabilization of Member Dues into operating income for 1979, which increased projected income to the level of anticipated expenses and balanced the 1979 budget. The resulting balances of reserve accounts as of December 31, 1978, were as fol-

TABLE 2
Comparison of Functional Revenues and Costs for Fiscal Years 1978 and 1979

Item	1978 (%)	1979 (%)
Revenues		
Publications		
Advertising	29.27	25.82
Subscription and related fees	4.27	5.02
Book sales	4.22	10.72
Member services		
Dues from members	42.30	36.63
Meetings income	7.11	7.50
Other income	1.40	1.45
Other income (investments, overhead on grants, etc.)	11.43	12.86
Total dollars	$7,577,042	$8,635,808
Costs		
Governance (Board of Trustees, Councils, Assembly)	17.54	18.86
Publications (APA journals and book sales)	36.27	33.99
Public Affairs (public information and government relations)	7.72	8.04
Member services (membership services and educational programs)	28.16	28.15
General and administrative (administrative offices and other costs)	10.31	10.96
Total dollars	$7,573,446	$8,535,215

lows: Reserve for Stabilization of Member Dues, $607,039; Reserve for Future Expansion and Development, $1,689,473; and Unrestricted Surplus, $263,605.

During 1979 the balance of the Reserve for Stabilization of Member Dues ($607,039) was utilized as income to preclude the need for a dues increase during that year, in accordance with the plan developed by the Board of Trustees in February 1977. The Reserve for Future Expansion and Development was reduced by $7,000 during 1979 (from $1,689,473 to $1,682,473) due to special funding for two policy components. The Unrestricted Surplus increased by $50,593 during 1979 because of the surplus from general fund operations. The balances of the reserve accounts as of Dec. 31, 1979, were as follows: Reserve for Future Expansion and Development, $1,682,473; Unrestricted Surplus, $314,198.

Publications. The Association maintains approximately 90 book titles in inventory. Total revenues for 1979, net of discounts, were $805,370. This represents an increase of $532,175 over 1978 sales of $273,195. The increase basically stems from sales of *DSM-III* during the last quarter of 1979 amounting to $560,000. Revenues from the initial sales of *DSM-III* helped support overall expenses of APA during 1979. Publication sales usually amount to about $250,000, for a slight excess of revenue over expenses, which keeps the activity on a self-sustaining basis.

Psychiatric News generated revenues of $1,280,469, contrasted with expenses of $1,045,315, for a surplus of $235,154. The revenue amount represents an increase of $45,090 over 1978 totals. The *American Journal of Psychiatry* produced revenues of $1,192,595 and incurred expenses of $1,207,736, for a deficit of $15,141. It should be noted that the Association levies an overhead charge to its three publications at the rate established for indirect costs for federal grants and contracts. The overhead rate for the *American Journal of Psychiatry* during 1979 was 36.1% of direct costs. This publication shows a surplus of $305,135, excluding APA overhead charges. The *Hospital & Community Psychiatry* journal realized revenues amounting to $259,187 and expenses of $476,625. The resulting deficit was $177,438. After adjusting for the APA overhead charge to the journal, the deficit for 1979 is reduced to $50,015.

Grants and contracts. In February 1977, the Association began working to increase its level of support from grants and contracts. Since that time, income from these sources has increased from $294,842 per year to an annual rate of $1,374,178. These additional funds have enabled APA to provide needed products and services without placing an additional burden on other income sources. The Association is establishing a Resource Development Committee, which will endeavor to generate additional grant/contract funds to support the provision of products and services to members and their patients.

Conclusion. It is a pleasure to present to you what I believe is a very forthright and positive annual report on the fiscal activities of the APA during 1979 and the financial condition of the Association at this time. You are welcome to discuss any aspect of this report with me during the annual meeting or to review the total audited figures for income and expenses at APA headquarters.

I wish to express my gratitude to you for entrusting me with the responsibility for the fiscal affairs of the APA, and to the Assembly, to the Board of Trustees, to the officers, and to the Investment Advisory Committee for their support and their ongoing interest in the financial stability of the Association. Appreciation is also due to the Medical Director, the Deputy Director for Business Administration, the Comptroller, and the entire staff for their capable assistance whenever needed during the year.

CHARLES B. WILKINSON, M.D.

Report of the Medical Director

I AM PLEASED to present my annual report to each of you, the first in the new decade—the 1980s. Although I will follow my usual pattern of highlighting staff activities and developments, I would also like to use this occasion to make a few additional comments and observations.

One of my hopes for this new decade is a wider perception among members of APA's strengths and capacities as well as a realistic evaluation of the problems facing our patients and our profession. Whenever I have an opportunity to visit district branches throughout the country, I make a point of focusing on this theme. In this context I have attempted to deal with some of our members' episodic moods of doom and gloom about the future of psychiatry. This syndrome is characterized by the belief in certain myths which, in my judgment, can lead to errors in policies and actions. The first is the myth that the profession is under severe siege and is cut off from allies. The reality is that we have many problems and there are areas where we must struggle very hard. However, from my perspective, it is a serious error to underestimate the many friends and potential allies that psychiatry has—in the general public, the medical profession, the courts, the legislative and executive branches of government, the clergy, and mental health professionals. We must be careful to differentiate the irrational and malignant antipsychiatry groups from those who have indicated a willingness or demonstrated a potential to discuss issues with us in a serious manner, even if some of these groups do criticize us from time to time. Unless we do this, our policies can rigidify, we can alienate genuine friends, and the profession will develop a momentum toward self-fulfilling negative prophecies.

Of course there are very serious challenges facing us in the years ahead, but in my view a strongly functioning APA can and will deal with these problems—with vigor, intelligence, and persistence.

A second myth that we should lay to rest is the perception of psychiatry as mired in a perpetual identity crisis. From my vantage point, we underwent several shifts in direction during the 1960s and 1970s. My travels to many district branches, however, have convinced me that a consensus has evolved during the last few years. We have emerged from a period of flux and rapid change with a much clearer medical identity. This identity involves strong alliances with the rest of medicine and also recognizes our fundamental role as physicians. Such a model is not one of biological reductionism, but gives strong emphasis to the psychological, the social, including epidemiological, and other salient variables directly related to psychopathology and its treatment. Of course, debate continues on the precise role of psychosocial variables and we have occasional passionate differences over the psychiatric implications of major social policies. Nevertheless, my reading of the membership is that the ideological differences among us diminished significantly during the late 1970s. It is time to state forcefully that the identity crisis is over.

This is an edited version of the report presented by the Medical Director to the annual business meeting in San Francisco, Calif., May 5, 1980.

In the light of our need to be strong in order to cope with the many challenges that will face us in the '80s, I believe it would be a tragic mistake to pull up the wagons and retreat into a constricted reactive posture. We must clarify the priorities, objectives, and functions of the profession and the Association and then choose wisely among them.

My staff and I have been deeply influenced in the past six months by the variety of fiscal issues facing the Association: the levying of the assessment to finance a new headquarters building, the dues increase, and the unprecedented imposition of a registration fee for members attending the annual meeting.

In particular, the assessment and dues increase were lightning rods that brought to our attention many issues in addition to the manifest fiscal questions. The image that the Association projects to its members is extremely important and we must take a close look at what the benefits of membership really are. I have read every letter protesting the assessment. More than that, I have looked at every scrawled note on the bill itself. I didn't know that several of our members were descended from Samuel Adams and identified with those who put on a famous tea party. Of course, many of our members were traumatized by the assessment and the dues increase, as indeed were the Assembly and the Board last fall. Nevertheless, I have been delighted with the magnificent response so far to the assessment and the dues increase. I have also been pleased that the number of resignations is not unusual for this stage of our fiscal year. The opportunity to engage in a dialogue with members who have protested the fiscal decisions has been a rewarding challenge for me. Many of you have also been engaged in this process and I urge you to increase your activities. Concerned and responsive leadership will make a real difference.

During the past six months an interdepartmental staff committee, consisting of the Medical Director; Carol Davis, Special Assistant to the Medical Director; Dr. Lee Gurel, Director of Membership Services and Studies; Mary Jane Merrick, Chief of Data Processing; Marta DeLalla, Membership Associate; Ron McMillen, Assistant Director of Public Affairs; Dr. Jack White, Deputy Director for Business Administration; and Robert Milanicz, Comptroller, have met regularly to monitor the assessment and the dues billing. This has involved planning responses to the inquiries and questions that have come from our members about the assessment and dues increase; working with the Ad Hoc Committee to Consider Waivers of the Assessment so that guidelines could be established; ensuring that all appropriate data about the assessment waivers and the dues were entered into the computer; and reviewing data to determine the percentage of members who had paid the assessment and dues to consider what appropriate actions might be if the cash flow for the Association was not maintaining 1979 levels. This has indeed consumed a great deal of staff time. However, we have responded to every inquiry from a member and attempted to ensure that the members are aware of the benefits of belonging to APA. We have been successful, in several instances, in persuading a member to pay the assessment and dues and not resign from the Association.

When you return home from this meeting, an all-member

mailing will be delivered. It will contain routine items like submission forms for next year's annual meeting, and we have also included a brief newsletter, "APA Is Working For You," highlighting membership benefits and activities; a Quick Reference Telephone Directory for locating specific staff who can help with various problems; and information about public affairs activities and media resource details, among other things. The Association is active in many areas that touch every member's practice, and it is important that we communicate this clearly and concisely. Many of our members feel isolated or apart from APA and each of us has an obligation to reduce this distance.

As I have stated on a number of occasions, we must have priorities because we can no longer attempt to do a little bit of everything. Fiscal restraints must be recognized and choices among alternatives made. With the institution of long-range planning and functional budgeting, I believe that we will make these hard choices and that our objectives and functions will be clearer.

I believe each of you will enjoy this annual meeting, our 133rd. Not only is San Francisco a pleasant place to be, but the program content is exceptionally rich and exciting. There will be extensive press coverage of the meeting and of events surrounding it. I am convinced that the scientific coverage will be beneficial for psychiatry. My personal thanks goes to Dr. Allan Beigel and the Scientific Program Committee for their continued commitment to excellence.

Each of you also has my special thanks for your support of our staff. President Stone, Speaker Pasnau, and other APA leaders have been extremely positive in their support of our strong and effective staff, and I am most appreciative of their actions demonstrating their intent in this matter.

I am pleased that I have been asked to continue as your Medical Director. With full recognition that I am clearly accountable to the Board of Trustees, to the Assembly, and to the membership, I will continue as your Medical Director with the belief that I am living up to my own standards and your expectations.

The remainder of my report deals with more specific staff activities. For historical purposes, the Medical Director has asked each department and division head, as well as each deputy director, to prepare detailed reports, which are appended.

MEDICAL DIRECTOR'S OFFICE

Several additional activities have been assumed by our office during the past year to improve coordination of particular functions.

My office has assumed responsibility for monitoring all of APA's liaison activities with other organizations and preparing a special report for each meeting of the Board of Trustees on these activities. We currently have official liaison representatives to more than 30 organizations, and the APA President very often appoints ad hoc representatives to attend meetings deemed to be of interest to the Association. All of these representatives submit reports and recommendations to the Trustees. My office ensures that all suggestions and action items are transmitted to the appropriate people and that effective action is taken.

I have recently transferred to my office the monitoring of all of APA's international affairs activities, including staff support for the Council on International Affairs. Many of our members are increasingly interested in transcultural psychiatry and in relationships with colleagues in other countries. We are actively working to establish an efficient and organized data base for these activities.

One example of such activities this past year, in addition to those mentioned in the annual report of the Council on International Affairs, was helping the Embassy of the Peoples' Republic of China to coordinate the schedule of a visiting team of Chinese neurologists and internists who were here in the United States studying the diseases of the elderly. This type of activity broadens our colleagueship with physicians and specialists from other countries.

The Office of the Scientific Program and its staff have been transferred to the Office of the Medical Director, and we are now closely involved in the preparation and production of the program and syllabus for the Annual Meeting.

Dr. Donald W. Hammersley, Deputy Medical Director, Ms. Claudia Hart, his administrative assistant, and Ms. Dottie Myrick, his secretary, serve as a part of the Medical Director's Office and provide assistance in implementing policies of the Board. They also provide valuable staff support to various APA components. Dr. Hammersley has the administrative responsibility for several departments in the Central Office (the APA CHAMPUS Peer Review Project, Membership Services and Studies, Library and Museum), is Editor of *Hospital & Community Psychiatry* and Director of the Hospital and Community Psychiatry Service, and provides primary staff support to a number of organizational components, including the Commission on Standards of Practice and Third-Party Payment, the Ad Hoc Committee on Interprofessional Relations, and the Ad Hoc Committee on Health Insurance for Mental Disorders.

GENERAL COUNSEL

APA general counsel, Rogovin, Stern & Huge, under the direction of Mr. Joel Klein, has been very active this past year in a variety of areas of legal concern to the Association. As various courts and agencies seek to expand the application of the antitrust laws to medicine, APA's interest and responsibility in these matters has heightened. Last year I reported to you on work done in the case involving the Virginia Neuropsychiatric Society; clinical psychologists sued two Blue Shield programs and the district branch, alleging a conspiracy to deny direct reimbursement for psychologists. Rogovin, Stern & Huge represented the district branch and a successful result was achieved at trial. The court found no conspiracy and stated that clinical psychologists require medical supervision. The case is currently being appealed. Similar issues have been raised in a suit filed by the Ohio Attorney General, charging the Joint Commission on Accreditation of Hospitals with violating the antitrust laws by failing to grant psychologists full admitting and clinical privileges. The Ohio Psychiatric Association and the Ohio Medical Association have been charged as coconspirators with JCAH. Mr. Klein is monitoring these matters very closely as well as developments in the FTC suit against the AMA, which places serious restrictions on the right of medical societies to restrict advertising.

Another area that required a significant amount of time was developing and negotiating the contract for APA's new headquarters building in downtown Washington. This project has been brought to a successful conclusion and involved

major tax and real estate issues, as well as delicate negotiations with a major Washington developer. Dr. John McGrath, Chair of the APA Building Committee, and Dr. Jack White, Deputy Director for Business Administration, worked closely with Mr. Klein on this.

OFFICE OF BUSINESS ADMINISTRATION

Under the very capable direction of Dr. Jack W. White, Deputy Director for Business Administration, this office has completed its first five years. Highlights of the office's activities during this period have included preparation and execution of balanced budgets for each of the past five years, establishment and maintenance of a strong control mechanism for management of the Association's investment portfolio, continued reorganization and refinement of the business functions, implementation and monitoring of a new personnel classification system, and fiscal issues complicated by deterioration of the overall economy, by the proposed dues increase for 1980, the initiation of an annual meeting registration fee for members, and the financing of a new headquarters building.

I would particularly call your attention to Dr. White's report on the 1981 budgeting process and implementation of a functional budgeting system. Functional budgeting will allow the Association to achieve its objectives by assigning resources to accomplish those objectives and will ensure control of expenses as the budget is executed. Functional budgeting will lead to the development of improved fiscal policies as well as serve as a means to control costs.

Dr. White is to be particularly congratulated for helping to ensure that the Association found suitable space for a new building and that the fiscal arrangements were favorable to APA.

OFFICE OF EDUCATION

This office, under the capable direction of Dr. Carolyn B. Robinowitz, Deputy Medical Director, provides the management skills as well as the expertise in a variety of educational endeavors now under the aegis of APA. For example, this office worked with the committee that developed the fourth Psychiatric Knowledge and Skills Self-Assessment Program, which allows APA members to test their performance on an examination against a group of their peers and provides up to 40 hours of Category I CME credit. More than 5,700 APA members have chosen to participate in this important continuing education activity.

During 1979, in collaboration with a consortium of psychiatric educational service organizations, the Office of Education held several conferences to help psychiatric educators improve their teaching activities and skills. This project, through a grant from NIMH, provides specific information to assist educators in areas such as selection and use of assessment technique and methodologies, patient management problems, work habits, professional attitudes, etc.

Another major activity of the office during the year was planning the Conference on Psychiatric Manpower and Recruitment, held in March 1980 in collaboration with representatives of a consortium of psychiatric education and service organizations under an NIMH grant. More than 140 participants considered issues of psychiatric manpower, distribution, and utilization as well as recruitment. Both the quantity and the quality of the participants attested to the intense interest in this problem. The meeting will result in a report, including far-reaching recommendations to attack the recruitment problem on many levels. It is the first major step in what we know will be a long-term process.

APA also collaborated with the American Medical Student Association and the American Association of Directors of Psychiatric Residency Training in developing a brochure that describes current residency programs in psychiatry. In addition, the APA Committee on Medical Education has developed a brochure, "Choosing Psychiatry," directed toward medical students. Copies are available from the Office of Education.

The office has sponsored several symposia to assist members in the use of *DSM-III*. The first was a two-day conference developed in collaboration with Columbia University and held in New York City in December 1979. There were three other one-day symposia: one during February in Houston, one during March in San Francisco, and one during April in Boston. These symposia were clinical and practical in nature and well received by the 1,500 participants.

The important work of helping APA members, especially those who do not attend annual meetings, to meet their continuing education requirements is primarily carried out through the district branches and Area Councils and through other national organizations. APA and the Office of Education have served as educational consultants and helped to plan and review programs for Category I CME credit; this past year, APA sponsored 225 programs for a total of 1,976 hours of Category I CME credit. In addition, the office has fostered the development of self-study materials, including the "Survey of Psychiatry," a self-learning package consisting of audiotapes, background materials, syllabus, workbook, and assessment exercises.

The Office of Education also continues to work with the large number of components housed within the APA Council on Medical Education and Career Development, implementing the various policies and recommendations that emanate from the Council.

It should be noted that the office coordinates many aspects of psychiatric education in the United States and relates not only to other associations and to APA components but also to policy makers. Many of the major problems facing psychiatry during the 1980s will have a substantial educational component and I am pleased that we have become increasingly proactive in these matters.

GOVERNMENT RELATIONS

This division, under the capable guidance of Mr. Jay Cutler, provides the interface for APA policy with the legislative and executive branches of the federal government. At the same time it provides APA insight into, and analysis of, the health policy development process of Congress and the executive branch so that we can develop policy on these activities. This staff also provides support to the Joint Commission on Government Relations and to the Councils on Aging and on Governmental Policy and Law.

In an effort to respond to district branch concerns, the division is attempting to develop the capacity to identify major state legislative activities of common concern to all district branches and to provide the branches with analytic state

government relations information, serving as an informational resource on such major issues. For example, the division provided Vanderbilt University with updated information on the governance issues for community mental health centers; provided the San Diego District Branch with detailed information on the conflict between the HMO law and the HEW regulatory interpretation; and provided the Kentucky District Branch with background material on pending legislation dealing with reimbursement of other mental health professionals for the treatment of nervous, mental, or emotional disorders. The division also compiled a 50-state survey of laws dealing with patient rights provisions as a resource for this legislative issue at the local level.

Mr. Cutler's report deals in depth with APA Congressional testimony and APA legislative initiatives in the 96th Congress. He is to be congratulated on his continued attention to legislative issues of importance and concern to all of psychiatry.

OFFICE OF MINORITY AFFAIRS

A sizeable percentage of the work done in this department continues to focus on matters related to mental health and underrepresented groups, i.e., children, minorities, and women.

The APA/NIMH Fellowship Program, under Dr. Jeanne Spurlock's able direction, remains a positive force in recruiting and supporting minority residents in psychiatry. There were 59 applicants this year to the APA/NIMH Fellowship Program and 14 fellowships were awarded. The new Fellows include a native Hawaiian interested in child and community psychiatry, a Hispanic resident planning to work in community psychiatry, and an American Indian who expects to work in the area of cross-cultural psychiatry.

The staff directs a major percentage of its time and effort toward supporting the work of the Council on Children, Adolescents and Their Families and of the Council on National Affairs.

In addition to her many liaison activities with a variety of organizations, her membership on the Graduate Medical Education National Advisory Committee, and on the ABPN Committee on Certification in Child Psychiatry, Dr. Spurlock continues her teaching contributions to the child psychiatry training program at Children's Hospital, and to the medical student education program at Howard University and at the Uniformed Services Medical School.

PROFESSIONAL AFFAIRS

Dr. Henry H. Work, Deputy Medical Director and Director of Professional Affairs, provides staff assistance to the Assembly through the District Branch Office; to the Council on Research and Development; to the Scientific Program Committee (and chairs the Staff Committee on the Annual Meeting); to the Consultation and Evaluation Services Board; to the Achievement Awards Board; and to the Program Committee for the annual Institutes on Hospital & Community Psychiatry. Dr. Work also serves as member or officer of more than 9 other organizations (in particular as secretary of the Group for the Advancement of Psychiatry and secretary-general of the American College of Psychiatrists).

Professional correspondence is a very large part of the activities of this office. People from all over the country write to APA requesting help for themselves, relatives, and friends who suffer from mental illnesses. These letters demand personal attention, which is promptly offered. Our colleagues write to us for general and specific advice related to the mental health field. Young people have also learned that APA is a source of information on careers. So voluminous are these requests that Dr. Work and his staff prepared a pamphlet on how to become a psychiatrist. Requests for career information in 1979 totaled 1,433.

Some changes occurred in this office during 1979: staff support to the H&CP Service (including the Film Loan Library and Supplementary Mailings) is now provided by the *H&CP* journal staff.

Although recognizing that this is a time of fiscal stringency, Dr. Work has emphasized that there must not be any cutback on the quality of services rendered to our members and to the public, and the entire staff joins in these sentiments.

Dr. Work's report more fully discusses the 31st H&CP Institute, held in New Orleans in September 1979. However, I did want to mention that this was a very successful meeting, with over 800 persons registered. The theme, "Myths, Slogans and Bandwagons: A Realistic Appraisal," was well received and the evaluation comments stressed the high quality of the program.

PUBLIC AFFAIRS

In 1979 Mr. Robert L. Robinson retired as Director of Public Affairs. A search committee, chaired by Dr. Shervert Frazier, is currently reviewing resumes of applicants for this job. During this period Mr. Ron McMillen, Acting Director, has kept the division functioning. Media relations were dominant during 1979, following up on the Snowmass conference for national media writers held in September 1978. Efforts were made to present a more positive view of psychiatry in the public press, radio, and television. During 1979 *Time* and *Newsweek* both featured psychiatry in cover articles which emphasized newer directions in psychiatry. Following the telecast of "One Flew Over the Cuckoo's Nest," NBC aired a special segment of "Prime Time Sunday" that presented a positive view of ECT. Also, many newspapers featured positive articles on ECT. Mr. McMillen's report highlights media coverage in 1979.

Another major activity during 1979 was responsibility for the production of *DSM-III* and of a completely revised 5th edition of *A Psychiatric Glossary*. Promotional activity by this division accounted for advance sales of 30,000 copies of *DSM-III* and 6,000 of *A Psychiatric Glossary*. Staff also supervised the publication of three new Task Force reports.

New projects initiated during the year included a series of books for lay audiences on psychiatry, a new project that will allow reporters to prepare advance articles on the annual meeting, and planning for the 1980 national media conference for editors and publishers.

CONCLUSION

These are just a few highlights of Central Office staff activities. I urge each of you to take this report home and care-

Report of the Speaker

I AM PLEASED to report that the Assembly of District Branches is alive and well, that its officers are optimistic and enthusiastic about its accomplishments and its future, and that its members, your elected Representatives and Deputy Representatives, have worked hard and well on your behalf during the past year. This year the relations between the Board of Trustees and the Assembly have matured to a level of mutual respect. I have the sense of a team, working together to confront and solve some of the most complex and perplexing problems ever to face our profession, our organization, and our patients' lives. I feel indebted to our President, Alan Stone, who took great effort to consult with me and with the Assembly at every turn. Although Dr. Stone did not always agree with me or with the Assembly, his fairness and openness to diverse opinions and policies set a very high example for those who follow him. I should like to acknowledge as well the crucial roles the APA staff have played in the development of the Assembly-Board relations. At the helm is our trusted colleague and friend, Mel Sabshin, whose support of the Assembly and its officers is ever felt and rarely commended. Henry Work, who has been my friend, professor, and mentor for more than 20 years, has been unselfish with his time, advice, and occasionally his criticism. Ms. Ann Whinnie brings to her job 25 years of experience with APA and the Assembly and reminds us that our "new" ideas were already old when she started working here. The innovation has been Ms. Jeanne Robb in her role of Board-Assembly affairs coordinator. She has visited the Area Councils, developed agenda for meetings, and played a central role in the writing of a procedures handbook for the Assembly. APA members have every reason to be proud of the dedicated, professional personnel who work so hard and care so much about our organization and its objectives.

Einstein once remarked that he liked to state things as simply as possible, "but not simpler." This has been a very complex year; stating it simply would minimize the difficulty of the problems and the agonizing nature of some of the decision making. Nonetheless, I must be brief and can only highlight some of the year's significant issues and accomplishments. I will leave the report of good news to the President and the Medical Director and limit my report to seven major problem areas—Money, Education, Politics, Bureaucracy, Law, Internal Organization, and Future Planning. The Assembly and the District Branches have been most concerned with these problems during the past year.

MONEY

Reflecting the inflationary aspects of the national economy, the costs of operating the Association increased significantly during the past several years. Because of careful fiscal planning and budgeting, a dues increase was not necessary until this year. How unfortunate that it should come just at the culmination of more than five years of planning and debate over a new headquarters for our national office, and during the sharpest rise in inflation since World War II. The Assembly and Board carefully weighed the alternatives and decided to levy an assessment on each dues-paying member and ask for additional contributions from them and from the non-dues-paying members as well. They also decided to institute a modest registration fee at the Annual Meeting. The process through which these difficult decisions were made as well as the timeliness of the final actions are examples of our organization working at the highest level of cooperation and efficiency. This course of action has received support from large constituencies in the organization, and almost every District Branch president sent a letter to the members in support of these fiscal decisions.

National and state fiscal cutbacks were also of vital concern to the Assembly. The threatened termination of research and training funds could not have come at a worse time for our psychiatric residency education programs. During a period when we are facing a decline in the number of psychiatric residents, when psychiatry is a specialty of low interest to medical students, and when one-third to one-half of these medical students cannot distinguish between the roles of psychiatrists, psychologists, and social workers, we must place a high priority on strengthening our teaching centers, not in dismantling them. Some states, notably California, are facing severe cutbacks that threaten public education, particularly state hospital-community mental health programs. We must develop new alliances to challenge these issues, as well as look to new relationships with industry and private funding sources.

EDUCATION

The conflicting attitudes toward Continuing Medical Education also concern the Assembly. The compliance of members in reporting their required hours of CME activities, although very high, nonetheless reveals that many members are having trouble with this requirement. The cost to APA in monitoring is high, and the problem of dealing with noncompliant members is perplexing. The Assembly has been presented with an action paper calling for the discontinuation of the mandatory CME reporting now required by our Consitution and By-Laws. There is also confusion at the national level over the accreditation of CME activities After the AMA resigned from the Liaison Committee for CME and appointed a new Committee for Accreditation of CME, APA found itself with a foot in each committee. The Assembly played an important role in developing the original CME guidelines for APA. It will continue to keep a close watch on national developments and the district branches' proposals for future policies.

POLITICS

For several years the Assembly has urged the development of guidelines for psychiatrists in their professional rela-

This is an edited version of the report presented by the Speaker of the Assembly to the annual business meeting in San Francisco, Calif., May 5, 1980.

tionships with nonpsychiatrist mental health professionals. The need for guidelines became even more urgent with the lawsuits in Virginia and Ohio. The delay involved in adopting these guidelines reflects more on the difficulty of developing them than on the efficiency of our organization. The Assembly feels that some sort of guidelines are needed as soon as possible. With the passage of time, they can be modified and augmented as may be required.

The Equal Rights Amendment has also presented the Assembly with a dilemma. The support of the economic boycott has been one of the most divisive issues in the recent history of APA. The Assembly has supported ERA and has urged the Board on several occasions to back its support by not meeting in non-ERA states. Referenda on three occasions have indicated that the members do not support the Assembly in this strategy. Many members of the Assembly viewed the recent referendum as a less than ideal way to resolve this issue. Most Assembly members felt that presentation to a forum of elected representatives, providing a full discussion of all points of view, followed by a vote of the Board of Trustees, would have been far better than a last minute referendum presented to the members without prior notification and with little public discussion in *Psychiatric News*. In fact, in the fall the Assembly urged the Board not to submit a referendum on ERA this year. However, the Constitution and By-Laws permit individual members to submit referenda in this manner without review by the Assembly. Perhaps this points to a deficiency in our procedures that should be revised in the future. On an issue of this magnitude, hasty action is of little benefit to ourselves, our patients, or our organization.

On the national political level, the Assembly supported the creation of the American Psychiatric Political Action Committee. This important structure will make it possible for individual psychiatrists to become involved in influencing the creation and modification of legislation in ways which were not possible before. The Assembly will watch with great interest how this independent body functions and what implications it might have for similar developments on the district branch and Area levels.

BUREAUCRACY

With the promulgation of the new Consolidated Standards for Psychiatric Facilities by the Joint Commission on the Accreditation of Hospitals, it became clear that psychiatric facilities were facing an almost overwhelming task of understanding and meeting the thousands of criteria. Assembly members found several sections to be unacceptable to psychiatry, particularly the section dealing with the Medical Staff. In the new standards the Medical Staff gives way to the Clinical Staff. Other areas of treatment planning, patients' rights, medical audits, and review activities made dramatic changes in hospitals' conceptualization of patient care. Administrators were expected to spend thousands of hours of professional time attempting to meet the criteria. Often standards relating to similar activities were dispersed throughout the document, making compliance frustrating and difficult. The Assembly alerted the Board to the problems and the Board, through its Council on Mental Health Services and with the help of the Medical Director, moved quickly to take some remedial action. The Assembly cannot help but wonder whether a voluntary organization such as JCAH really represents a great advantage over the federal government. The Assembly will continue to play a role in watching and advising as the year progresses.

Filing forms for third-party payers is a tedious, taxing business for all of us. When confronted with the arbitrary and often discriminatory treatment insurance companies give our patients, it is small wonder that most of us feel angry and frustrated. Peer review is often seen as a screen for denial of insurance benefits to those who need them most and an insult to our integrity and sense of professional responsibility. It has been difficult for the Assembly to support the efforts of APA's CHAMPUS project and the work with the Aetna Insurance Company in developing a new program of psychiatric peer review tied to insurance benefits under these programs. Legitimate concerns about confidentiality notwithstanding, the Assembly has become convinced that without the peer review provisions contained in these programs, third-party coverage for all psychiatric treatment will be jeopardized. The Assembly has urged caution and careful review before expanding the program to include other carriers; efforts of the Assembly members of the ad hoc committee studying these projects have been of considerable help in changing some of the questionable practices that could have threatened confidentiality and at the same time have facilitated the research and evaluation efforts that may pave the way to greater coverage for our patients. The Assembly will continue to participate in these important studies and in the final decisions about the future of these activities.

LAW

There is a Mexican curse: "May there be a lawyer in your life." Since I assumed the office of Speaker there has been a lawyer in my life every day. Fortunately, Joel Klein has been on our side, advising us and helping us to understand and to act. Currently cases of great importance are being tried in courts all over the country. Right to refuse treatment and right to treatment cases sometimes confound those of us who believe that scientific, rational medical judgment is superior to jurisprudence in determining treatment issues. The most recent action of APA in the abortion case before the U.S. Supreme Court exemplifies the timely participation by our legal staff in issues important to our profession. We badly need a position statement on the civil commitment of adults. As is the case with the guidelines for working with nonpsychiatrist mental health professionals, the document need not be considered a final product. The Assembly hopes that a policy will be adopted soon for use in the courtrooms and with the legislatures.

INTERNAL ORGANIZATION

No report of the Speaker in recent years would be considered complete without an update on the progress made following the Key Conference. This year Assembly members participated in an Ad Hoc Committee on Reorganization, paying particular attention to the Council and component structures. As a result of their study, the committee recommended that the Reference Committee be restructured and the timing of the meetings altered to permit the new Joint Reference Committee to serve both the Board and the Assembly. Proposals for these changes will be presented during

this meeting, and the proposed changes in the Constitution and By-Laws will be on the ballot in 1981. The Assembly endorses these changes and believes that they will lead to greater efficiency and cost effectiveness.

FUTURE PLANNING

One of the keys to effective budgeting lies in prioritizing activities and eliminating those activities with the lowest priorities. In order to establish priorities, it is necessary to have long-range planning that determines which activities will be needed in the future and which activities may be phased out. Under the current system of line-item budgeting, it is not possible to differentiate such activities; hence, cost containment falls like a meat axe across the board. The Assembly listened to an eloquent description of long-range planning by Milton Greenblatt, chairperson of the Ad Hoc Committee, and endorsed his recommendations that APA join with a professional planning group to develop long-range plans for the Association. Participation by Assembly members in the process will allow for full discussion by most of the constituencies in APA and provide a more rational approach to cost containment and effective use of APA resources.

VALEDICTION

The year has not been simple, and the problems that have arisen have been taxing, yet I have grown and learned much as a result of my experience as Speaker. In retrospect I enjoyed it immensely. My sadness in leaving the office is alleviated by the confidence that I have in the new Speaker, Mel Lipsett, and by my conviction that his wisdom and experience will provide him with the skill needed to guide the Assembly and APA through these troubled times.

ROBERT O. PASNAU, M.D.

Report of the Speaker-Elect

SHAKESPEARE APTLY described the seven ages of man. We can loosely compare them with the development of the Assembly in its birth and growth over the past 27 or so years. We have come from infancy to adulthood. Here we part a little with the Bard and have to synthesize some of the stages. Combining the stages of soldier and statesman, we must fight to maintain our principles of effective treatment for our patients and must espouse and sustain our objectives with a variety of groups. We must help educate those groups, such as legislators, about the value of our objective of effective, professional treatment. We also need to exhibit the generativity and creativity Erikson defined along with our collective selves in the trichotomy of members as self, and Assembly as family existing in the third part of the trichotomy—a rapidly changing society.

With this meeting we enter a new decade, already filled with less than happy portents for the future in regard to psychiatric care and marked by a decline in interest in public mental health care. This is not a phenomenon that has suddenly erupted; it has been looming ever larger over the past decade. However, it is a phenomenon that we have been more effectively rising to meet, challenge, and battle. We are aware of the discussions about budgets at the national, state, and local levels. The philosophy is "guns before butter," which translates into a reduction of human services, including mental health care. Here in California we are faced with a referendum called Proposition 9, to be voted on in June. If it passes, it will further reduce income to the state. The State Director of Mental Health has suggested closing the few remaining state hospitals, returning the patients to their communities, but without appropriate funding for their care. It is not surprising that some patients are beginning to show up in the criminal justice system.

In preparation for this report, I did some research into the reports of past Speakers-Elect. My findings proved interesting, stimulating, encouraging, and at times disconcerting. One universal theme that prevailed was that of increasing membership participation. Currently this theme, which fits in well with our thrust toward greater democratization, is essential for our continued growth and development. I believe we need to aid the increased development of the Area Councils and the more active participation of the District Branch Councils and membership in the procedures and processes of our organization. One possibility which I raise for your consideration is that the district branch representative who is liaison to a task force or committee develop a mini-task force within his district branch to assist him. The participation of the Assembly has been invaluable in the development of *DSM-III* and Council reports. Our members in the district branches are a reservoir of much needed talent.

We are embarking on a new process in our organization—the process of functional budgeting. It is an exciting concept that will enable us to more sharply define objectives, goals, and priorities. We have grown in undertaking new activities vital to us in today's world. We have initiated many excellent programs, encompassing many activities. Unfortunately, there is a limited budget, restricted even more by inflationary pressures. There is also the specter of diminishing public and government support for human services. In our Association we have to establish our own process for what we consider to be our most important ways of achieving the objectives outlined in our Constitution. At the March 22 meeting of the Assembly Executive Committee Dr. Jack White told us that approximately 150 functions had been defined. Obviously some will have a higher priority than others, but how do we decide which these should be? It is my belief that the Assembly, via the Area Councils and the district branches, should have an important voice in the delineation and selection of the priorities.

This can only be done by an involved and informed membership. Three issues that I feel deserve a high priority are proactive involvement in legislation, research, and public information. We have developed a voice at the federal level; we now need to assist the development of a potent voice at the state level. Many district branches and some Area Councils have already done this. I feel it is essential that we assist those district branches and Area Councils which do not have such a system. We also need to push for more attention to research in psychiatry by encouraging closer liaison between the district branches and the departments of psychiatry within their jurisdictions. We need to let people know of our concerns about the care of the mentally ill and to enlist community support for good professional care.

It is my contention that we have to take a searching look at the process of referendum. The concept is an excellent one. However, I fear we are drifting toward governance by referenda. I do not feel that this is in the best interests of APA and recommend careful thiking about the process and its ramifications.

I am mindful of and pleased by the honor you have bestowed on me. The Speaker is the moderator in the Assembly and represents your voice at the Board of Trustees. I shall try to carry on the worthy tradition of my predecessors.

MELVIN M. LIPSETT, M.D.

This is an edited version of the report presented by the Speaker-Elect of the Assembly to the annual business meeting in San Francisco, Calif., May 5, 1980.

Report of the Membership Committee

THE MEMBERSHIP COMMITTEE met October 16, 17, 18, and 19 (1979) at APA headquarters in Washington, D.C. During this period it discussed Fellowship criteria and appraised its own operations and functions, met with Dr. Carolyn Robinowitz to discuss Continuing Medical Education requirements, met with the Assembly Membership Committee to consider policy and action matters common to both committees, and acted on Fellowship nominations and membership issues.

The Membership Committee, both alone and in conjunction with the Assembly Membership Committee, reviewed the report of the Board of Trustees' Ad Hoc Committee on Fellowship Criteria. The committee felt that its conduct of the Fellowship reviews basically agreed with the report's spirit and recommendations. Indeed, many of the suggestions are already being practiced and in a careful, thoughtful, and judicious fashion. The committee has acted on the assumption that it must take into account the total person and his or her breadth of experience and skills.

Fellowship is an honor and the committee endorsed the concept of Fellowship by invitation rather than by individual application; it enthusiastically supported the suggestion that district branches take full responsibility for presentation of the most complete information possible for their nominees. Specifically, the committee feels that 1) letters of recommendation could be written by nonpsychiatrists as well as psychiatrists, 2) letters of recommendation should address the specific criteria and requirements for Fellowship, 3) there could be more definitive feedback about deferrals, with emphasis on the district branches' responsibility, 4) district branches might counsel certain Fellowship aspirants not to apply, and 5) psychiatrists should be aware of the different criteria, the different areas, and the point system used in judging applicants, while remembering that the committee's deliberations and processes are flexible and geared toward an assessment of the total person.

A basic understanding of, and agreement on, national standards and criteria for Fellowship are more important than mechanisms (such as the point system) for assessing nominees. We must increase communication among district branches, the Central Office, and this committee in order to improve the quality of information submitted on behalf of the nominees. Both committees strongly support better education of, and collaboration with, the district branches concerning Fellowship matters. The committee recommended conducting a workshop for Fellowship/Membership Chairpersons, as well as offering workshops to Area Councils. Proposals included a one-day workshop for district branch representatives, possibly at APA headquarters, followed by a series of smaller regional workshops, plus a voluntary workshop at the Annual Meeting.

Clearly, there is legitimate concern over defining the vari-

This is an edited version of the report presented by the Membership Committee to the annual business meeting in San Francisco, Calif., May 5, 1980.

TABLE 1
Membership Transactions

Status	Number in 1979
New	1,155
Provisional Members	19
Members-in-Training	499
General Members	610
Associate Members	11
Corresponding Members	11
Corresponding Fellows	4
General Members-at-Large	1
Advancements	457
Provisional Member to Member-in-Training	11
Member-in-Training to General Member	417
Associate Member to General Member	27
At-Large Associate Member to At-Large General Member	2
Reinstatements	49
General Members	48
Associate Members	1
Transfers in member status	101
General Member to Fellow	79
General Member to Member-in-Training	16
Inactive Member to General Member	1
Corresponding Member to General Member	1
At-Large General Member to Fellow	1
Corresponding Member to Corresponding Fellow	3
Resignations and drops	351
Total resignations from APA	96
Total drops for nonpayment of APA dues	190
Total drops/resignations from district branches and consequently dropped from APA	65
Life Fellows and Members and 50-Year Life Fellows/Life Members	360
Life Fellows, 1979	167
Life Members, 1979	177
50-Year Life Fellows/Members	5
Life Associate Members	11
Inactive status	44
Inactive Members	32
Inactive Fellows	5
Temporary Inactive status	7
Waiver actions	79
Requests granted for waiver of dues	79
Honorary and Distinguished Fellows	2
New Honorary Fellows	1
New Distinguished Fellows	1
Membership Committee Deferral/Denial Actions	164
Deferred, transfer General Member to Fellow	78
Deferred, applications for Corresponding Member	1
Requests for waiver of 2-year waiting period, transfer General Member to Fellow, denied	1
Requests for waiver of 8-years as a General Member, transfer General Member to Fellow, denied	63
Requests for transfer to Inactive Member/Fellow, denied	2
Requests for transfer to Temporary Inactive Member/Fellow, denied	6
Requests for waiver of dues denied	13
Deceased	236

TABLE 2
APA Membership[a]

Status	Number as of Jan. 1, 1980	Number as of Jan. 1, 1979
Provisional Members	17	9
Members-in-Training	1,526	1,441
Associate Members	511	576
General Members	15,559	15,079
Fellows	3,909	4,041
Life Members	690	535
Life Fellows	2,001	1,909
Life Associate Members	10	—[b]
Corresponding Members	198	175
Corresponding Fellows	210	202
Distinguished Fellows	29	28
Honorary Fellows	36	33
Inactive Members	513	508
Inactive Fellows	136	139
Total	25,345	24,676

[a]Net gain of 669.
[b]Life Associate Member created by Constitutional amendment, 1979.

ous characteristics that "create" a Fellow. What do we mean by "outstanding," "with distinction," "depth and scope of knowledge," or "breadth of skills and interests"? "Excellence" requires not only better calibration but better documentation as well. Most importantly, this committee cannot be adversarial in nature and function; rather, it must be supportive and respond to the needs of the members. We welcome a dialogue on these matters.

In a somewhat related matter, concern has been expressed over defining the term "professional suitability" as currently noted in the APA By-Laws. This term appears as a criterion for APA membership. The Membership Committee along with the Assembly Membership Committee, struggled with a definition and interpretation; we developed a statement that the Board of Trustees at its December meeting referred back to us for further study. The Board suggested that the code of medical ethics may help clarify this term.

The inescapable fact of a decline in membership also confronts us. The 5.3% growth rate that occurred between 1975 and 1976 dropped to a 3% growth rate between 1978 and the present. There has also been a greater increase in the non-dues-paying membership than in the dues-paying membership. In 1975 the non-dues-paying membership was 11.9% of the total; in 1979 it was 13.7% of the total. This means that new members must be recruited, perhaps largely through local community efforts, if we are to maintain and enhance a dynamic APA.

Requests for exemption from CME requirements based on disability, illness, limited practice, and early retirement are now being, and will continue to be, submitted to the Membership Committee. In the process of reviewing the exemption requests, the committee has developed several guidelines concerning exemption from CME requirements which it will recommend to the Board of Trustees. The committee will be flexible and considerate in its actions, and, as in other matters, will need the district branches' help to justify and support the exemption requests.

In view of the large number of members—approximately 8,000—who have not yet requested CME certification, the committee recommended that a letter be sent immediately by the Office of Education reminding these members of the requirements and informing the noncomplying members that their membership may be in jeopardy. The committee also recommended that a grace period of at least one year be allowed before any member who has not complied with CME requirements is dropped. In fact, even a two-year grace period—up to July 1981—may be considered.

The committee reviewed its position on the current eligibility requirement for Fellowship that extends the number of years needed as a General Member from five to eight. The referendum changing the requirement occurred during March and April, and notification was sent to the district branches on May 7.

The committee agreed that it could not review nominations that did not meet the eight-year requirement, and felt that this action was in accord with the wishes of the electorate and the Board of Trustees (who could have, but did not, specify a later implementation date). Subsequently, a number of district branches expressed considerable dismay and chagrin that the committee did not review the nominations for which a waiver of the eight-year requirement had been requested. Although the committee felt it had acted fairly and in good faith, the expectations of the district branches had to be respected. The Board of Trustees, with the active support of the chairman of the Membership Committee and the Membership Office staff, fashioned a suitable compromise plan whereby anyone who has been a General Member for six years or more is eligible to apply for Fellowship this summer.

A summary of membership actions during calendar year 1979 is presented in table 1. The status of APA membership in January 1980 compared with January 1979 is presented in table 2.

PHILIP M. MARGOLIS, M.D.

Report of the Committee of Tellers

THE COMMITTEE OF TELLERS met on March 20, 1980, at APA headquarters to certify the results of the 1980 election. Ballots had been mailed on February 4 to 23,682 eligible voting members. Ninety undeliverable ballots were deducted from that number; the final number of eligible voting members was 23,592; 10,800 ballots were returned and included in the final vote tally (which represents 45.8% of the eligible voting members).

The Tellers acted on questionable ballots that had been held for their decisions. The Tellers also verified that all candidates for the 1980 election had certified the accuracy of their submitted biographical information and that all candidates had submitted the required statements of compliance with election guidelines.

The Committee of Tellers certified that the following individuals were elected to office: Daniel X. Freedman, M.D., President-Elect (70.2% of votes cast); Robert J. Campbell III, M.D., Vice-President (51.9% of votes cast); George H. Pollock, M.D., Treasurer (52.8% of votes cast); Elissa P. Benedek, M.D., Trustee-at-Large (55.4% of votes cast); John J. McGrath, M.D., Area III Trustee (64.7% of votes cast); and Paul F. Slawson, M.D., Area VI Trustee (59.1% of votes cast).

In order to have a valid election on constitutional changes, 40% of the eligible voting members must cast votes. An abstention is considered to be a vote cast and counts toward determining whether 40% has been reached. Of that number, two-thirds must approve amendments to the Constitution; a majority must approve amendments to the By-Laws. The results of the voting were: Article III (creates Elections Committee)—41.7% of eligible voters cast votes, amendment passes, with 83.6% in favor; Chapter 9.2 (defines functions of Tellers and Elections Committee—41.1% of eligible voters cast votes, amendment passes, with 80.8% in favor; Chapter 9.8 (composition of Elections Committee)—41.4% of eligible voters cast votes, amendment passes with 81.5% in favor; and Chapter 9.9 (institutes recall mechanism for officers)—37.9% of eligible voters cast votes; vote on this amendment is invalid as it failed to achieve required 40% of voters casting votes.

Finally, the Committee of Tellers certified the vote count on the referendum concerned with holding APA meetings in states that have not ratified the Equal Rights Amendment: 5,679 members voted in favor of the referendum, which represented 54.6% of votes cast; 4,461 members voted against the referendum, or 42.9% of votes cast; and 268 members abstained, or 2.6% of votes cast. The total number of votes cast was 10,408, representing 44.1% of eligible voting members.

On April 11, 1980, the Committee of Tellers recommended that the Board of Trustees accept the above results of the 1980 election, and the Board accepted them.

EDWARD C. KIRBY, M.D.

This is an edited version of the report presented by the Committee of Tellers to the annual business meeting in San Francisco, Calif., May 5, 1980.

Report of the Committee on Constitution and By-Laws

THE COMMITTEE ON CONSTITUTION AND BY-LAWS (CCBL) has appreciated the cooperation of the components and members of the Association who have shared issues with us or made recommendations to us that have helped us to carry out our functions this year. As chairperson, I wish to thank the members of the committee for their good work and thoughtful contributions, and APA's counsel, Joel Klein, and staff members, Lee Gurel and Carol Lehmann, for their patience, good sense, and helpfulness.

The APA membership recently approved amendments recommended by the CCBL and others to create an Elections Committee that would establish and oversee election policies. We hope this move will increase efficiency in elections, and lead to more orderly campaigning. The CCBL also recommended, and the Board accepted, changes in the *Operations Manual* that would shift some functions from the Tellers Committee to the Elections Committee.

At the Board's request, the CCBL proposed some brief, clear wording to the Board to be used if the Board chose to have another ERA referendum. (Since another ERA referendum subsequently came to the Board and the APA membership by petition, the Board chose not to add one of their own, no matter how clear.)

In a measure to improve what many APA members consider a troublesome lack of clarity in the wording of recent referenda, the CCBL proposed and the Board accepted a new mechanism for the *Operations Manual* that will review the wording of referenda for clarity.

The CCBL recommended to the Board retention of the words "professional suitability" in the By-Laws, Chapter 2, along with three clarifying paragraphs.

The CCBL obtained an opinion from Joel Klein on votes being taken by the Assembly at the annual business meeting. It is Mr. Klein's opinion that votes can be taken on any matter fairly raised by an Assembly member who questions an item in one of the reports presented, asks for a vote, and has his request seconded by another Assembly member. (Since this is the only meeting of the Assembly also regularly attended by APA members, who customarily sit with their representatives, this interpretation may provide greater attention and responsiveness to these reports.)

Still under review by the CCBL are matters regarding ethical issues, a possible limit to the size of the Assembly, a By-Law stating that resignations will not routinely be accepted if dues or ethical complaints are awaiting resolution, the status of By-Laws 4.2 and 6.3 vis-à-vis current APA appointment procedure, and an invitation to the Assembly to propose some words for the By-Laws defining Assembly powers and responsibilities.

The CCBL, in keeping with the requirements of the Constitution and By-Laws, is presenting to the membership the following proposed amendments, which have been approved by the Board of Trustees (see appendix 1). The proposed amendments are summarized as follows:

1. Chapter 1, Section 6: The change will bring the Constitution and By-Laws of the APA in line with ABPN requirements for residency training established in 1977.
2. Chapter 1, Section 7: The additional phrase in this section makes it consistent with requirements for other member categories and is in keeping with the policy of the Association.
3. Chapter 2, Section 1: The addition to this section clarifies that certification of membership applications is done by the Membership Committee.
4. Chapter 6, Section 7: The amendment changes the composition of the Reference Committee, adds two members of the Board and two of the Assembly—an interesting new point of influence for the Assembly—and removes voting power from the Council chairpersons. This was recommended by the Board's Ad Hoc Committee on Council Structure and has been approved by the Board.
5. Chapter 7, Section 1: We have proposed that current Chapter 7.1 be deleted and a new Chapter 7.1 be enacted both to bring the By-Laws in line with current Assembly procedure and to allow the Assembly some flexibility in choosing among several plans, currently being considered by the Assembly's Committee on Procedures, designed to improve Assembly representativeness.
6. Chapter 8, Section 6: The change clarifies exemptions from paying both dues and assessments.

After six years on the CCBL, I have reached the constitutional time for retirement. I am thankful to the former members of the committee with whom I served and to the current members—Drs. Scott Nelson, Elissa Benedek, Hugh Smith, Kathleen Mogul, and Arthur Zitrin—for their good work and willingness to devote their time to the business of the committee.

LAWRENCE HARTMANN, M.D.

This is an edited version of the report presented by the Committee on Constitution and By-Laws to the annual business meeting in San Francisco, Calif., May 5, 1980.

APPENDIX 1
Proposed Amendments to the APA Constitution and By-Laws

The proposals that follow were received within the 60-day deadline before the annual meeting and were presented at the annual business session. They will appear on the ballot in March 1981.

Note: The following changes in type style indicate the kind of change proposed in the Constitution and By-Laws: 1) italic=proposed additions to the Constitution and By-Laws (e.g., *"by resolution of the Assembly"*), and 2) brackets=proposed deletions from the Constitution and By-Laws (e.g., "may originate [either] . . ."). Articles and sections of the Constitution and chapters and sections of the By-Laws that are not listed here have not been subject to any proposed changes.

THE BY-LAWS

CHAPTER ONE. MEMBERS

6. General Members shall be physicians who have [had at least three years of] *completed* acceptable training and who have either a valid license to practice medicine or hold an academic, research, or governmental position that does not require licensure.

7. Fellows shall have been General Members for at least eight years and shall have made a significant contribution to the field of psychiatry. *Fellows must have either a valid license to practice medicine or hold an academic, research, or governmental position that does not require licensure.* At its discretion the Board, upon recommendation of the Membership Committee, may waive the requirement for eight years as a General Member. The criteria and procedures for selection and nomination of General Members for Fellowship shall be established by the Board and the Membership Committee and shall apply uniformly for all District Branches.

CHAPTER TWO. MEMBERSHIP PROCESSING

1. Admission to membership shall require valid election by the appropriate District Branch approved to process membership applications, with certification *by the Membership Committee of the Association* that constitutional requirements for membership have been met.

CHAPTER SIX. COUNCILS, COMMITTEES, BOARDS, AND OTHER ORGANIZATIONAL ENTITIES

7. A Reference Committee shall be established to act upon the concerns of the several councils *and commissions* and to refer matters [to and from the Board.] *from the councils and commissions to the Board and/or the Assembly and from the Board or the Assembly to them.* It shall be composed of the President-Elect, who shall be the chairperson; [the chairperson of each council] the Speaker-Elect of the Assembly, *who shall be vice chairperson; two members of the Board of Trustees; two members of the Assembly;* and the Medical Director. *Ex-officio non-voting members shall be the chairpersons of the councils and commissions.*

CHAPTER SEVEN. THE ASSEMBLY AND THE DISTRICT BRANCHES

[1. The Assembly shall be comprised of an elected representative and alternate representative from each District Branch.]

1. There shall be an Assembly of District Branches whose voting members shall be elected and shall include at least a) one representative from each District Branch, and b) an Executive Committee including Area Representatives and Assembly Officers.

CHAPTER EIGHT. PRIVILEGES AND RESPONSIBILITIES

6. Every Fellow, General Member, Associate Member, and Member-in-Training shall pay both dues and assessments as determined by the Board and the District Branches. All other categories of membership shall be exempt from paying dues *and assessments* to both the Association and its District Branches. A dues-paying member shall be exempt from paying such dues *and assessments* when the sum of that member's years of active membership in the Association plus the member's age at the start of the fiscal year shall equal 95.

1979 Annual Report of the American Board of Psychiatry and Neurology, Inc.

OFFICE AND MEMBERS OF THE BOARD

At its Policy Meeting on November 15, 1979, the Board elected the following officers: Dr. Marc H. Hollender, President, Dr. Melvin D. Yahr, Vice President, Dr. Dewey K. Ziegler, Secretary, Dr. Richard M. Steinhilber, Treasurer, and Dr. James N. Sussex, Executive Committee Member at Large.

Dr. John A. Talbott was elected to succeed Dr. Donald G. Langsley, who completed his term of four years in December 1979. Drs. Patrick F. Bray and S. Mouchly Small were elected to serve second terms of four years.

The 1979 Directors of the American Board of Psychiatry and Neurology, Inc., are as follows:

Nominated by the American Medical Association: Dr. Robert J. Joynt* (term expires December 1980), Dr. Richard M. Steinhilber* (term expires December 1981), Dr. T.R. Johns II* (term expires December 1982), Dr. James N. Sussex* (term expires December 1982), and Dr. S. Mouchly Small* (term expires December 1983).

Nominated by the American Neurological Association: Dr. Leonard Berg (term expires December 1981), Dr. Melvin D. Yahr* (term expires December 1982), Dr. Patrick F. Bray* (term expires December 1983), and Dr. John R. Calverley (term expires December 1980).

Nominated by the American Psychiatric Association: Dr. Marc H. Hollender* (term expires December 1981), Dr. Carolyn B. Robinowitz (term expires December 1982), Dr. Alfred M. Freedman* (term expires December 1982), Dr. Richard I. Shader (term expires December 1980), Dr. John A. Talbott (term expires December 1983).

Nominated by the American Academy of Neurology: Dr. Audrey S. Penn* (term expires December 1982) and Dr. Dewey K. Ziegler* (term expires December 1982).

EXAMINATIONS

Part I

At the Part I (written) examination on April 16, 1979, 1,672 psychiatrists were examined; 1,337 (80%) passed, 335 (20%) failed. A total of 423 neurologists were examined; 337 (80%) passed, 86 (20%) failed.

For the 1980 Part I examination, 1,797 psychiatrists and 530 neurologists have accepted assignment. (See table 1 for the number of candidates declared eligible and notified for Part I in 1980 and table 2 for a comparison of these figures for other years.)

TABLE 1
Number Declared Eligible and Notified for Part I Written Examination April 22, 1980

Item	Psychiatry	Neurology	Total
Number declared eligible and notified	1,952	546	2,498
Number who declined or withdrew	155	16	171
Number remaining for written examination as of March 1980	1,797	530	2,327

TABLE 2
Comparison of Number Declared Eligible and Notified for Part I Examination

Item	1974	1975	1976	1977	1978	1979
Total eligible	2,098	3,146	3,113	2,419	2,581	2,521
Total who accepted examination	1,821	2,408	2,391	2,087	2,361	2,367
Total who actually appeared	1,627	2,240	2,191	1,860	2,159	2,095

Part II

There were 437 candidates—352 psychiatrists and 85 neurologists—present at the Part II examination given in San Francisco, Calif., January 8–9, 1979 (see table 3). A total of 744 candidates—579 psychiatrists and 165 neurologists—were examined in New Orleans, La., April 9–10, 1979 (see table 4). There were 516 candidates—396 psychiatrists and 120 neurologists—present at the Part II examination given in Denver, Colo., June 9–10, 1979 (see table 5). The Fall 1979 Part II examination was administered to 1,027 candidates in Chicago, Ill., October 29–30, 1979 (see table 6). Of these candidates, 818 were psychiatrists and 209 were neurologists.

Future examination sites for 1980 are Los Angeles, Calif. (April 14–16), Milwaukee, Wis., (June 30–July 1), and New York, N.Y. (October 27–28).

CHILD PSYCHIATRY

Members of the current Committee on Certification in Child Psychiatry are Dr. Jeanne Spurlock, chairperson; Dr. Joseph Green, vice-chairperson, Dr. Irving Philips, secretary, Dr. Norman Bernstein, Dr. Norbert Enzer, Dr. Elva Poznanski, and Dr. Michael Cohen, liaison from the American Board of Pediatrics.

At its February 26–27, 1979, examination in Pittsburgh, Pa., the committee examined a total of 172 candidates. The number of passing candidates was 121 or 70%. Of the remaining 51 candidates, 74 (8%) conditioned the examination, while 37 (22%) failed.

At its September 27–29, 1979, meeting the committee agreed to move the deadline for filing applications for the

This is an edited version of the report of the American Board of Psychiatry and Neurology, Inc., to the American Psychiatric Association in Washington, D.C., April 11, 1980.

*These Directors are serving their second term of four years and are not eligible for reappointment.

TABLE 3
Analysis of Performance of Group Who Took Part II Examination in San Francisco, Calif., Jan. 8–9, 1979, by Specialty and Geograph c Area

	Total		Psychiatry		Neurology		Child Neurology		U.S. Graduates		Foreign Graduates	
Item	N	%	N	%	N	%	N	%	N	%	N	%
Candidates	437	100	352	80.5	79	18.1	6	1.4	298	68.2	139	31.8
Results												
Pass	243	55.6	195	55.4	44	55.7	4	66.7	177	59.4	66	47.5
Fail	189	45.3	154	43.7	33	41.8	2	33.3	118	39.6	71	51.1
Fail (must reapply)	5	1.1	3	0.9	2	2.5	0		3	1.0	2	1.4

TABLE 4
Analysis of Performance of Group Who Took Part II Examination in New Orleans, La., April 9–10, 1979, by Specialty and Geographic Area

	Total		Psychiatry		Neurology		Child Neurology		U.S. Graduates		Foreign Graduates	
Item	N	%	N	%	N	%	N	%	N	%	N	%
Candidates	744	100	579	77.8	151	20.3	14	1.9	398	53.5	346	46.5
Results												
Pass	483	64.9	381	65.8	92	60.9	10	71.4	287	72.1	196	56.6
Fail	153	20.6	114	19.7	37	24.5	2	14.3	76	19.1	77	22.3
Fail (must reapply)	108	14.5	84	14.5	22	14.6	2	14.3	35	8.8	73	21.1

TABLE 5
Analysis of Performance of Group Who Took Part II Examination in Denver, Colo., June 9–10, 1979, by Specialty and Geographic Area

	Total		Psychiatry		Neurology		Child Neurology		U.S. Graduates		Foreign Graduates	
Item	N	%	N	%	N	%	N	%	N	%	N	%
Candidates	516	100	396	76.7	112	21.7	8	1.6	304	58.9	212	41.1
Results												
Pass	291	56.4	216	54.5	71	63.4	4	50.0	222	73.0	69	32.5
Fail	154	29.8	121	30.6	29	25.9	4	50.0	50	16.5	104	49.1
Fail (must reapply)	71	13.8	59	14.9	12	10.7	0		32	10.5	39	18.4

TABLE 6
Analysis of Performance of Group Who Took Part II Examination in Chicago, Ill., Oct. 29–30, 1979, by Specialty and Geographic Area

	Total		Psychiatry		Neurology		Child Neurology		U.S. Graduates		Foreign Graduates	
Item	N	%	N	%	N	%	N	%	N	%	N	%
Candidates	1,027	100	818	79.7	184	17.9	25	2.4	611	59.5	416	40.5
Results												
Pass	595	57.9	468	57.2	111	60.3	16	64	423	69.2	172	41.4
Fail	352	34.3	288	35.2	56	30.4	8	32	148	24.2	204	49
Fail (must reapply)	80	7.8	62	7.6	17	9.3	1	4	40	6.6	40	9.6

examination in child psychiatry to October 1. Those candidates successfully completing the adult psychiatry Boards in the fall would be eligible to make application after this deadline.

The committee is currently engaged in development of an operations manual.

BOARD DECISION AND ITEMS OF INTEREST

1. The ABPN hosted a meeting of the Trans-National Certification group in Chicago on May 18–19, 1979. This group consists of representatives from Canada, the United Kingdom; Australia, and New Zealand and has been set up to study equivalent patterns of training and examinations. This was the first Trans-National Conference of its type and the reaction was extremely favorable. Future conferences are being planned and the ABPN intends to continue its representation in this group.

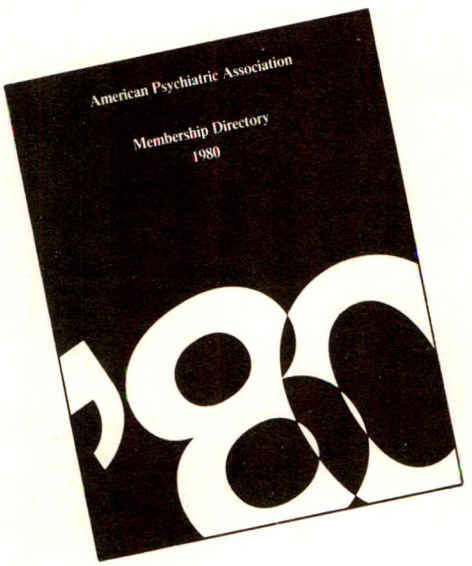
2. At its January 1979, Part II examination in San Francisco, the American Board of Psychiatry and Neurology developed a supplemental training session for new psychiatry examiners. This was highly successful and is being repeated at each examination.

At its policy meeting on November 15–17, 1979, the American Board of Psychiatry and Neurology took the following significant actions: 1) affirmed its requirement of an unlimited license to practice medicine in the U.S. or Canada in or-

der to be admitted to examination and expressed its concern regarding the altered requirement of the state of New Hampshire in issuing licenses limited to a particular specialty; 2) moved the deadline for receipt of applications for the Part I examination to September 1, effective for the 1981 examination; 3) agreed to submit recertification plans for psychiatry and for neurology to the ABMS; 4) agreed to continue to work with the AADPRT, AADCP, and APA to develop means whereby program heads and department chairpersons participate more actively in the certification process; and 5) agreed to adopt the following guidelines for ABPN examiners involved in Board review courses: The American Board of Psychiatry and Neurology, Inc., wishes to call to the attention of the profession that it is questionable ethics for a physician conducting, or participating in, any educational enterprise, particularly continuing education, to notify in any fashion those taking such courses that he/she is or has been a Director of, or examiner on, the ABPN. Such notification may be taken to mean that the material presented by that physician is in some way authorized or sponsored by the ABPN or that the course content and/or examination is in some way identical with that used in ABPN examinations.

LESTER H. RUDY, M.D.

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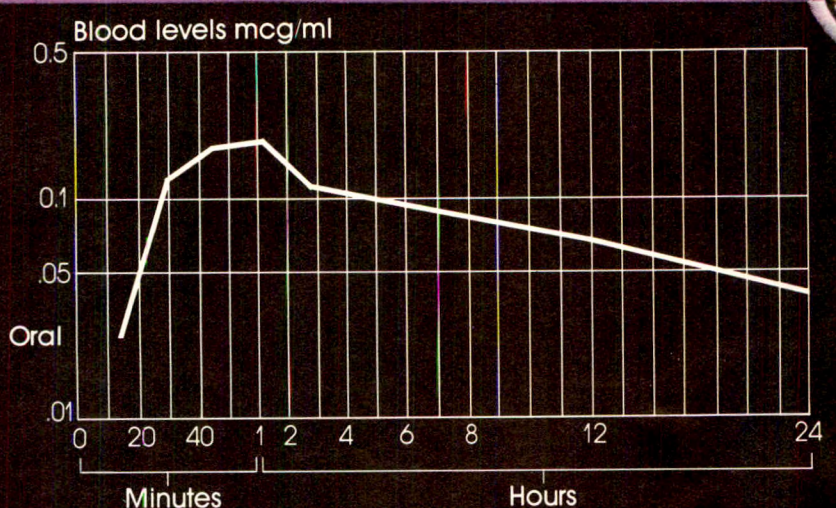
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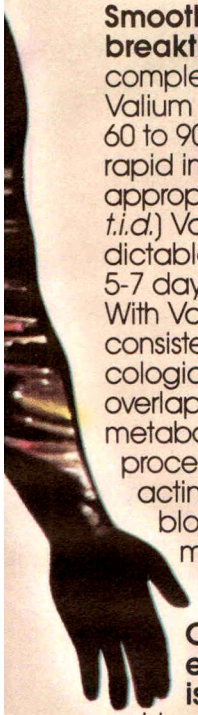
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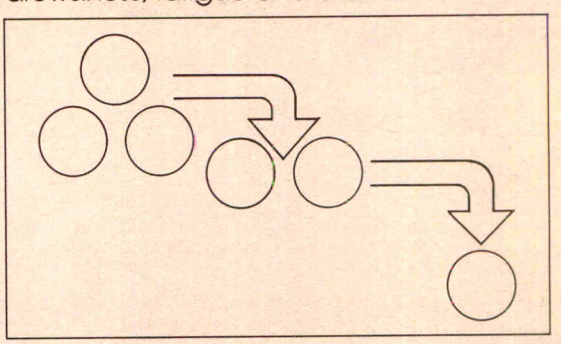
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Indications: Management of anxiety disorders, or short-term relief of symptoms of anxiety; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).
The effectiveness of Valium in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Anxiety disorders, symptoms of anxiety, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

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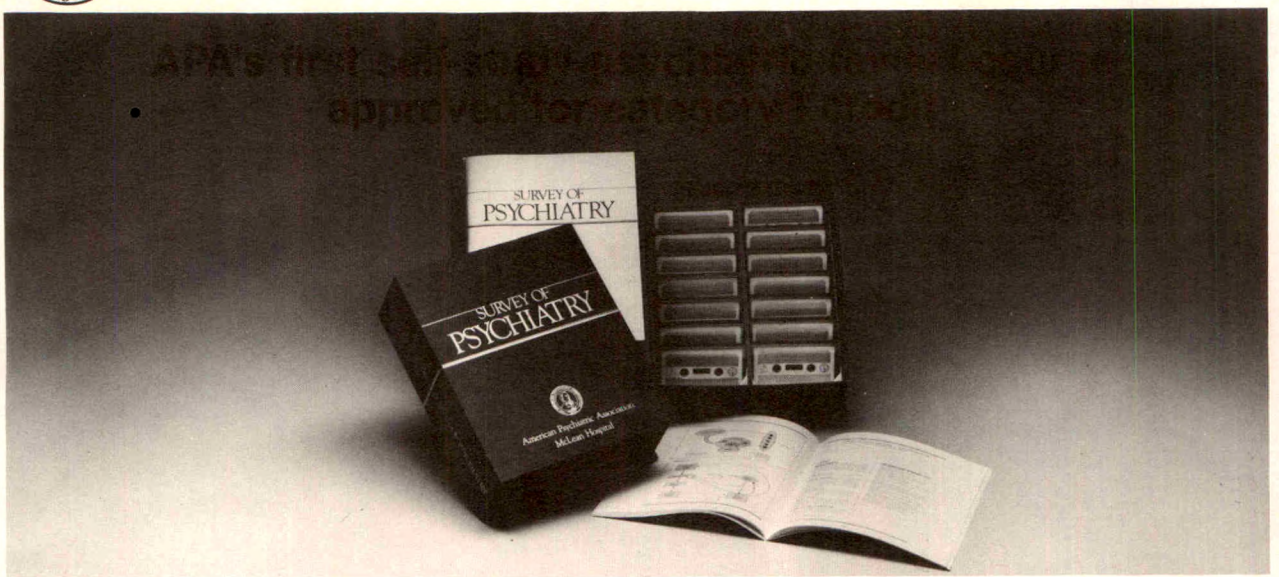
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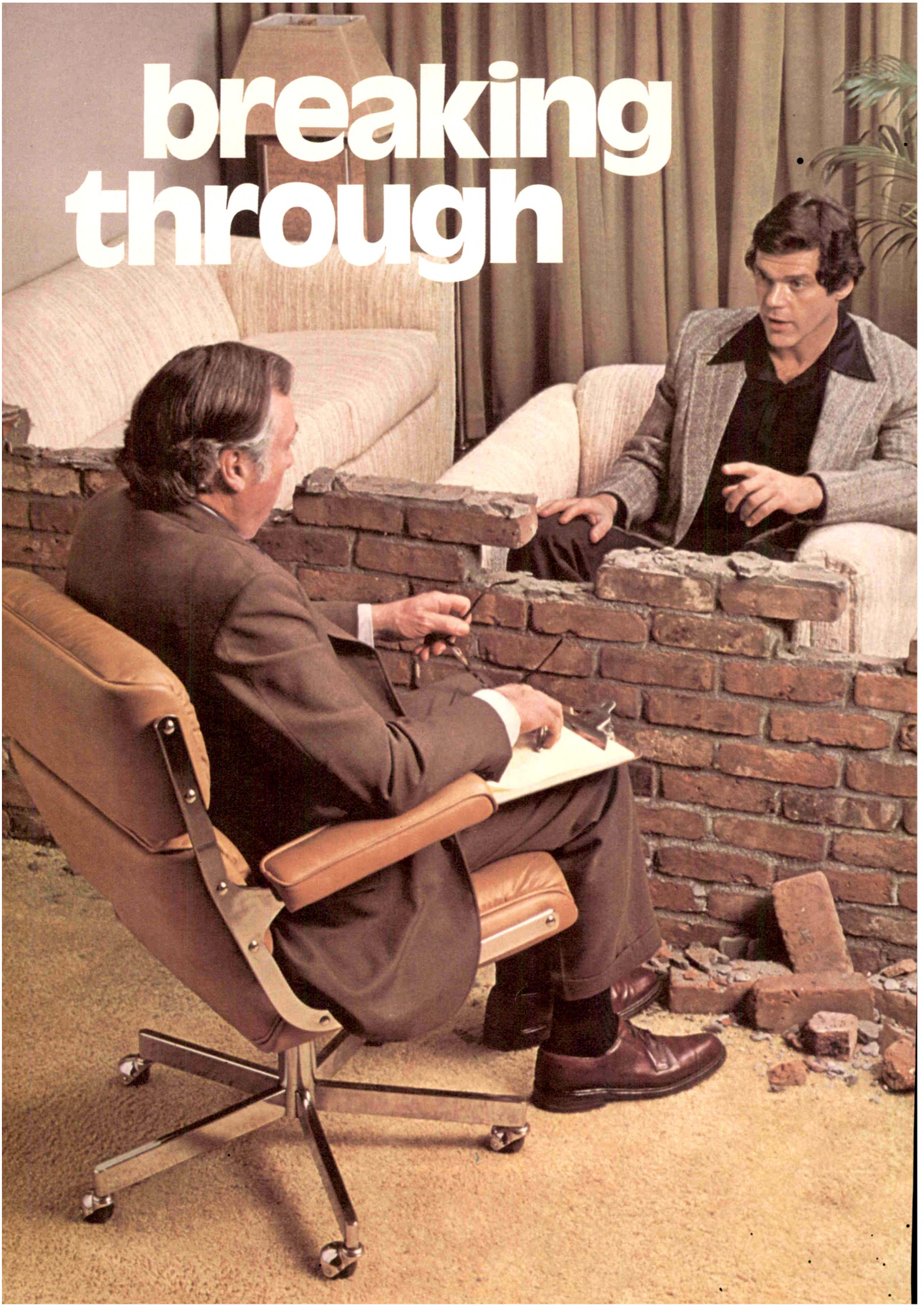
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CONTRAINDICATIONS: Central nervous system depression from drugs (barbiturates, alcohol, narcotics, analgesics, antihistamines); evidence of bone marrow depression; known hypersensitivity to phenothiazines or amitriptyline. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred from such combinations. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with TRIAVIL. Therapy should then be initiated cautiously with gradual increase in dosage until optimum response is achieved. Not recommended for use during acute recovery phase following myocardial infarction.

WARNINGS: TRIAVIL should not be given concomitantly with guanethidine or similarly acting compounds since TRIAVIL may block the antihypertensive action of such compounds. Use cautiously in patients with history of urinary retention, angle-closure glaucoma, increased intraocular pressure, or convulsive disorders. Dosage of anticonvulsive agents may have to be increased. In patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely. Tricyclic antidepressants, including amitriptyline HCl, have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time, particularly in high doses. Myocardial infarction and stroke have been reported with tricyclic antidepressant drugs. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Not recommended in children or during pregnancy.

PRECAUTIONS: Suicide is a possibility in depressed patients and may remain until significant remission occurs. Such patients should not have access to large quantities of this drug.

Perphenazine: Should not be used indiscriminately. Use with caution in patients who have previously exhibited severe adverse reactions to other phenothiazines. Likelihood of some untoward actions is greater with high doses. Closely supervise with any dosage. The antiemetic effect of perphenazine may obscure signs of toxicity due to overdosage of other drugs or make more difficult the diagnosis of disorders such as brain tumor or intestinal obstruction. A significant, not otherwise explained, rise in body temperature may suggest individual intolerance to perphenazine, in which case discontinue.

If hypotension develops, epinephrine should not be employed, as its action is blocked and partially reversed by perphenazine. Phenothiazines may potentiate the action of central nervous system depressants (opiates, analgesics, antihistamines, barbiturates, alcohol) and atropine. In concurrent therapy with any of these, TRIAVIL should be given in reduced dosage. May also potentiate the action of heat and phosphorous insecticides. There is sufficient experimental evidence to conclude that chronic administration of antipsychotic drugs which increase prolactin secretion has the potential to induce mammary neoplasms in rodents under the appropriate conditions. There are recognized differences in the physiological role of prolactin between rodents and humans. Since there are, at present, no adequate epidemiological studies, the relevance to human mammary cancer risk from prolonged exposure to perphenazine and other antipsychotic drugs is not known.

Amitriptyline: In manic-depressive psychosis, depressed patients may experience a shift toward the manic phase if they are treated with an antidepressant. Patients with paranoid symptomatology may have an exaggeration of such symptoms. The tranquilizing effect of TRIAVIL seems to reduce the likelihood of this effect. When amitriptyline HCl is given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. Paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs.

Caution is advised if patients receive large doses of ethchlorvynol concurrently. Transient delirium has been reported in patients who were treated with 1 g of ethchlorvynol and 75-150 mg of amitriptyline HCl.

Amitriptyline HCl may enhance the response to alcohol and the effects of barbiturates and other CNS depressants.

Concurrent administration of amitriptyline HCl and electroshock therapy may increase the hazards associated with such therapy. Such treatment should be limited to patients for whom it is essential. Discontinue several days before elective surgery if possible. Elevation and lowering of blood sugar levels have both been reported. Use with caution in patients with impaired liver function.

ADVERSE REACTIONS: Similar to those reported with either constituent alone.
Perphenazine: Extrapyramidal symptoms (opisthotonus, oculogyric crisis, hyperreflexia, dystonia, akathisia, acute dyskinesia, ataxia, parkinsonism) have been reported and can usually be controlled by the concomitant use of effective antiparkinsonian drugs and/or by reduction in dosage, but sometimes persist after discontinuation of the phenothiazine.

Tardive dyskinesia may appear in some patients on long-term therapy or may occur after drug therapy with phenothiazines and related agents has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw. Involuntary movements of the extremities sometimes occur. There is no known treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms. It is advised that all antipsychotic agents be discontinued if the above symptoms appear. If treatment is reinstituted, or dosage of the particular drug increased, or another drug substituted, the syndrome may be masked. Fine vermicular movements of the tongue may be an early sign of the syndrome. The full-blown syndrome may not develop if medication is stopped when lingual vermiculation appears.

Other side effects are skin disorders (photosensitivity, itching, erythema, urticaria, eczema, up to exfoliative dermatitis); other allergic reactions (asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions); peripheral edema; reversed epinephrine effect; hyperglycemia; endocrine disturbances (lactation, galactorrhea, gynecomastia, disturbances of menstrual cycle); altered cerebrospinal fluid proteins; paradoxical excitement; hypertension, hypotension, tachycardia, and ECG abnormalities (quinidine-like effect); reactivation of psychotic processes; catatonic-like states; autonomic reactions, such as dry mouth or salivation, headache, anorexia, nausea, vomiting, constipation, obstipation, urinary frequency or incontinence, blurred vision, nasal congestion, and a change in pulse rate; other adverse reactions reported with various phenothiazine compounds, but not with perphenazine, include grand mal convulsions, cerebral edema, polyphagia, pigmentary retinopathy, photophobia, skin pigmentation, and failure of ejaculation.

The phenothiazine compounds have produced blood dyscrasias (pancytopenia, thrombocytopenic purpura, leukopenia, agranulocytosis, eosinophilia); and liver damage (jaundice, biliary stasis).

Pigmentation of the cornea and lens has been reported to occur after long-term administration of some phenothiazines. Although it has not been reported in patients receiving TRIAVIL, the possibility that it might occur should be considered.

Hypnotic effects, lassitude, muscle weakness, and mild insomnia have also been reported.

Amitriptyline: Note: Listing includes a few reactions not reported for this drug, but which have occurred with other pharmacologically similar tricyclic antidepressant drugs and must be considered when amitriptyline is administered. *Cardiovascular:* Hypotension; hypertension; tachycardia; palpitation; myocardial infarction; arrhythmias; heart block; stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth; blurred vision; disturbance of accommodation; increased intraocular pressure; constipation; paralytic ileus; urinary retention; dilatation of urinary tract. *Allergic:* Skin rash; urticaria; photosensitization; edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis; leukopenia; eosinophilia; purpura; thrombocytopenia. *Gastrointestinal:* Nausea; epigastric distress; vomiting; anorexia; stomatitis; peculiar taste; diarrhea; parotid swelling; black tongue. Rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido; elevated or lowered blood sugar levels. *Other:* Dizziness, weakness; fatigue; headache; weight gain or loss; increased perspiration; urinary frequency; mydriasis; drowsiness; alopecia. *Withdrawal Symptoms:* Abrupt cessation after prolonged administration may produce nausea, headache, and malaise. These are not indicative of addiction.

OVERDOSAGE: All patients suspected of having taken an overdosage should be admitted to a hospital as soon as possible. Treatment is symptomatic and supportive. However, the intravenous administration of 1–3 mg of physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage of physostigmine should be repeated as required particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine. On this basis, in severe overdosage with perphenazine-amitriptyline combinations, symptomatic treatment of central anticholinergic effects with physostigmine salicylate should be considered.

J8TR32 (DC6613215)

For more detailed information, consult your MSD Representative or see full Prescribing Information. Merck Sharp & Dohme, Division of Merck & Co., INC., West Point, Pa. 19486.

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Lithobid

Slow-Release Lithium Carbonate 300 mg.

Before prescribing, see complete prescribing information in Rowell literature. The following is a brief summary.

WARNING
Lithium toxicity is closely related to serum lithium levels, and can occur at doses close to therapeutic levels. Facilities for prompt and accurate serum lithium determinations should be available before initiating therapy.

Indications: Treatment of manic episodes of manic-depressive illness. Maintenance therapy prevents or diminishes the intensity of subsequent episodes in manic-depressive patients with a history of mania.

Warnings: Lithium should generally not be given to patients with significant renal or cardiovascular disease, severe debilitation or dehydration, sodium depletion, or to patients receiving diuretics.

Lithium therapy has been reported in some cases to be associated with morphologic changes in the kidneys.

Caution patient and family to watch for diarrhea, vomiting, tremor, mild ataxia, drowsiness, or muscular weakness as signs of lithium toxicity, and to discontinue therapy and contact a physician should they occur. Patients receiving combined therapy with lithium and an antipsychotic should be monitored closely for early evidence of neurologic toxicity and treatment discontinued promptly if such signs appear. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery).

Lithium should not be used in pregnancy, especially during the first trimester, unless potential benefits outweigh possible hazards.

Not recommended for children under 12.

Precautions: Lithium tolerance is greater during the acute manic phase and decreases when manic symptoms subside.

Lithium therapy may lead to sodium depletion. Normal diet (including salt) and adequate fluid intake (2500-3000 ml) must be maintained, at least during initial stabilization period. Protracted sweating or diarrhea can decrease tolerance; in such cases, administer supplemental fluid and salt.

Each tablet contains 40 mg of sodium chloride, equivalent to 15.7 mg of sodium.

Sweating, diarrhea, and concomitant infection with elevated temperatures may require temporary reduction or cessation of dosage.

Adverse Reactions: Mild to moderate toxic reactions may occur at serum lithium levels from 1.5 to 2.5 mEq/L, and moderate to severe reactions at levels from 2.0 to 2.5 mEq/L. Fine hand tremor, polyuria, and mild thirst may occur during initial therapy and persist. Transient and mild nausea and general discomfort also appear during initial therapy. These effects usually subside with continued treatment or temporary reduction or cessation of dosage. If persistent, discontinue dosage.

Diarrhea, vomiting, drowsiness, muscular weakness, and lack of coordination may be early signs of toxicity and may occur at levels below 2.0 mEq/L. At higher levels, ataxia, giddiness, tinnitus, blurred vision, and a large output of dilute urine may be seen. Serum levels above 3.0 mEq/L may produce a complex clinical picture, involving multiple organs and systems. Serum levels should not exceed 2.0 mEq/L during acute phase.

The following reactions appear to be related to serum lithium levels, including levels within the therapeutic range: Neuromuscular—tremor, muscle hyperirritability (fasciculations, twitching, clonic movements of whole limbs), ataxia, choreo-athetotic movements, hyperactive deep tendon reflex; Central Nervous System—blackout spells, epileptiform seizures, slurred speech, dizziness, vertigo, incontinence of urine or feces, somnolence, psychomotor retardation, restlessness, confusion, stupor, coma; Cardiovascular—cardiac arrhythmia, hypotension, peripheral circulatory collapse; Gastrointestinal—anorexia, nausea, vomiting, diarrhea; Genitourinary—albuminuria, oliguria, polyuria, glycosuria; Dermatologic—drying and thinning of hair, alopecia, anesthesia of skin, chronic folliculitis, exacerbation of psoriasis, xerosis cutis; Autonomic—blurred vision, dry mouth; Thyroid Abnormalities—euthyroid goiter and/or hypothyroidism (including myxedema) with lower T_3 and T_4. I^{131} uptake may be elevated; EEG Changes—diffuse slowing, widening of the frequency spectrum, potentiation and disorganization of background rhythm; EKG Changes—reversible flattening, isoelectricity or inversion of T-waves; Miscellaneous—fatigue, lethargy, transient scotomata, dehydration, weight loss, tendency to sleep.

Reactions unrelated to dosage include: transient EEG and EKG changes, leukocytosis, headache, diffuse nontoxic goiter with or without hypothyroidism, transient hyperglycemia, generalized pruritus with or without rash, cutaneous ulcers, albuminuria, worsening of organic brain syndromes, excessive weight gain, edematous swelling of ankles or wrists, thirst or polyuria, sometimes resembling diabetes insipidus, and metallic taste. A single case of a syndrome resembling Raynaud's has been reported.

Dosage and Administration: *Acute Mania*—900 mg b.i.d. or 600 mg t.i.d. (1800 mg per day) usually will provide serum lithium levels ranging between 1.0 and 1.5 mEq/L. Serum levels should be determined twice per week until serum level and clinical condition have been stabilized.

Long-Term Control—900 mg to 1200 mg per day in two or three divided doses usually will maintain serum lithium levels at 0.6 to 1.2 mEq/L. Serum lithium levels should be monitored at least every two months.

How Supplied: 300 mg peach-colored tablets, imprinted "ROWELL 7514" in red, are supplied in bottles of 100 and 1000.

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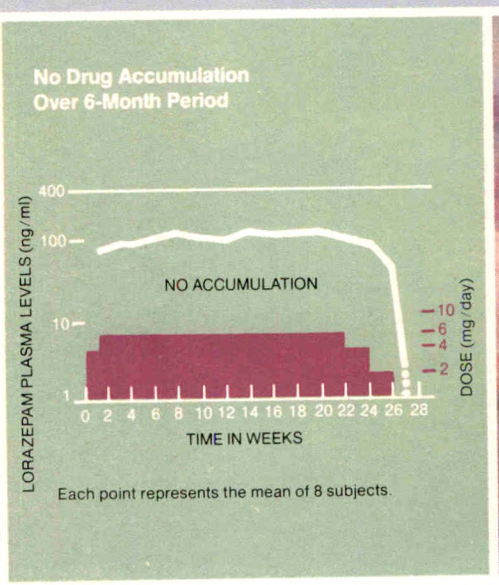
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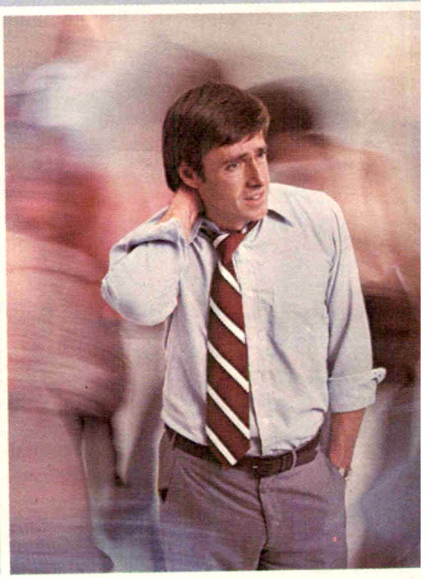
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Please see preceding page for brief summary of prescribing information.

1E May, 1979

How do you choose a minor tranquilizer?





Wyeth Laboratories
Philadelphia, PA 19101
TM

Pharmacokinetics

Ativan (lorazepam) is eliminated more rapidly than other benzodiazepines; generates no clinically active metabolites. The half-life of free lorazepam is about 12 hours; steady-state blood levels are attained in 2-3 days. (Comparable data for diazepam: 20-50 hours and at least 7-10 days.) Ativan shows no evidence of accumulation, even when given in high doses for as long as 6 months. (The pharmacokinetic profile of a drug can define such characteristics as absorption, distribution, metabolism and elimination but cannot, at present, be directly related to its therapeutic effectiveness.)

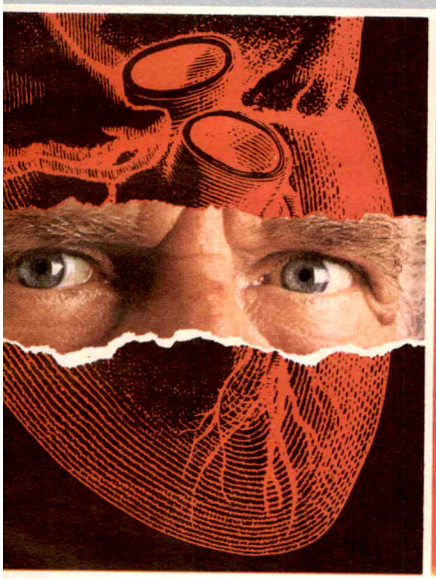
Anxiety neuroses

In many nationwide studies involving thousands of patients with anxiety neuroses Ativan (lorazepam) consistently provided significant relief of anxiety, tension, agitation and irritability as measured by standard Hamilton, Global (physician rated) and 35-Item (patient self-rated) scales.

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They're all about the same, aren't they? Until recently, that seemed to sum up the prevalent attitude about the benzodiazepines. After all, they all work similarly and all are effective anxiolytics. With seven of these compounds now available, however, differences have begun to emerge. Foremost among them: different metabolic pathways, which distinguish the relatively short-acting Ativan (lorazepam) from long-acting diazepam, clorazepate and chlordiazepoxide. At the same time, your choice can be made with confidence in the clinical record of Ativan, which confirms its value in anxiety neuroses and in significant anxiety associated with functional or organic disorders, as well as in older patients.

Consider the record of Ativan:



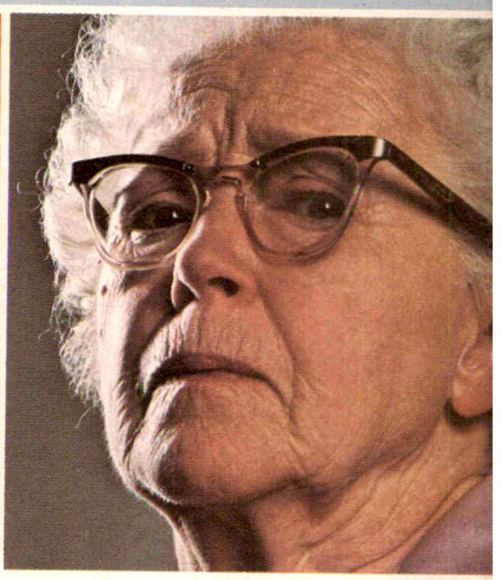
Anxiety in cardiovascular disorders

Ativan (lorazepam) has been specifically evaluated and found effective in seven common protocol, double-blind studies involving 423 patients (211 on Ativan) those anxiety was related to organic and functional cardiovascular disorders. The cardiovascular component as not, of course, been shown to be significantly benefited by such therapy.



Anxiety in gastrointestinal disorders

So far, nine common protocol, double-blind studies of Ativan have focused on anxious patients with functional or organic gastrointestinal complaints (457 patients, 234 on Ativan). Ativan was clearly effective in reducing the anxiety of these patients. The gastrointestinal component has not, of course, been shown to be significantly benefited by such therapy.



Anxiety in the aging patient

Because its simple metabolism is not readily impaired with advancing age, and accumulation is not likely to present a problem, Ativan is a good choice for older patients. Those who have trouble swallowing solid medication appreciate the small Ativan tablet, which is tasteless and disintegrates within seconds in water or fruit juice.

Ativan® (lorazepam) for Anxiety

See important information on following page.

Indications and Usage: Symptomatic relief of anxiety, tension, agitation, irritability and insomnia associated with anxiety neuroses and transient situational disturbances; anxiety associated with depressive symptoms and as a treatment of symptoms of anxiety if such symptoms are a significant feature of functional or organic disorders, particularly gastrointestinal or cardiovascular.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients on lorazepam not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation.

Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions.

Observe usual precautions with impaired renal or hepatic function.

Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component.

Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day. No effect dose was 1.25mg/kg/day (approximately 6 times the maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatric patients requires caution and frequent monitoring for symptoms of upper G.I. disease.

Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug.

In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Overdosage: In management of overdosage with any drug, bear in mind that multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring of vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

Ativan® IV for (lorazepam) Anxiety

Dosage: **Individualize for maximum beneficial effects. Increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; insomnia due to anxiety or transient situational stress, 2-4mg h.s.**

How Supplied: **0.5, 1.0 and 2.0mg tablets.**

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Philadelphia, PA 19101

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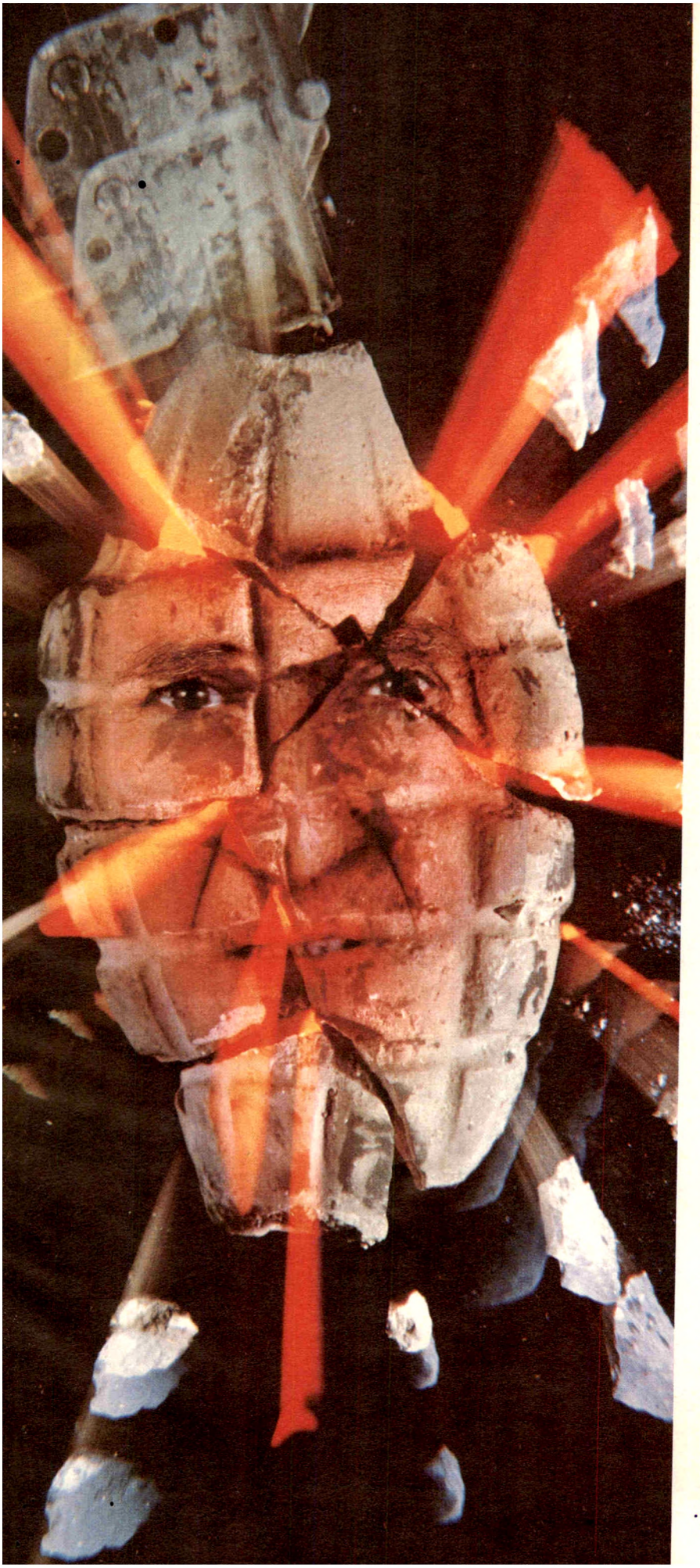
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Now, Navane Intramuscular 5 mg per ml provides a more concentrated dosage form for rapid control of severely disturbed behavior associated with acute psychosis. Navane Intramuscular, in carefully monitored doses, "...can produce significant improvement within hours and increase the accessibility of these [acutely disturbed] patients to more gradual methods of pharmacological treatment as well as to socio-environmental intervention and psychotherapy."[1]

Rapid tranquilization usually can be achieved without excessive sedation.[1] Clinically significant hypotension, often seen with other injectable antipsychotic agents, is rare with Navane Intramuscular therapy.[1-3] If extrapyramidal symptoms occur, they are usually readily controlled by dosage adjustment or antiparkinson agents.

ROERIG Pfizer
A division of Pfizer Pharmaceuticals
New York, New York 10017

For a brief summary of Navane prescribing information, including adverse reactions and contraindications, please see the following page of this advertisement.

Navane® (thiothixene) (thiothixene hydrochloride)

Capsules: 1 mg, 2 mg, 5 mg, 10 mg, 20 mg Concentrate: 5 mg/ml Intramuscular: 2 mg/ml, 5 mg/ml

BRIEF SUMMARY OF PRESCRIBING INFORMATION
Navane® (thiothixene)
Capsules: 1 mg, 2 mg, 5 mg, 10 mg, 20 mg (thiothixene hydrochloride) Concentrate: 5 mg/ml, Intramuscular: 2 mg/ml, 5 mg/ml

Contraindications. Navane (thiothixene) is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy*– Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration of Navane to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children– The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration– As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane (thiothixene). Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudo-parkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leukocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution; Navane For Injection– Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane (thiothixene) Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate– In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Stimulants that may cause convulsions (e.g. picrotoxin or pentylenetetrazol) should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg of thiothixene in bottles of 100, 1,000, and unit-dose pack of 100 (10 x 10's). Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100, 500, and unit-dose pack of 100 (10 x 10's).

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg, and in 30 ml (1 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, and 5 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

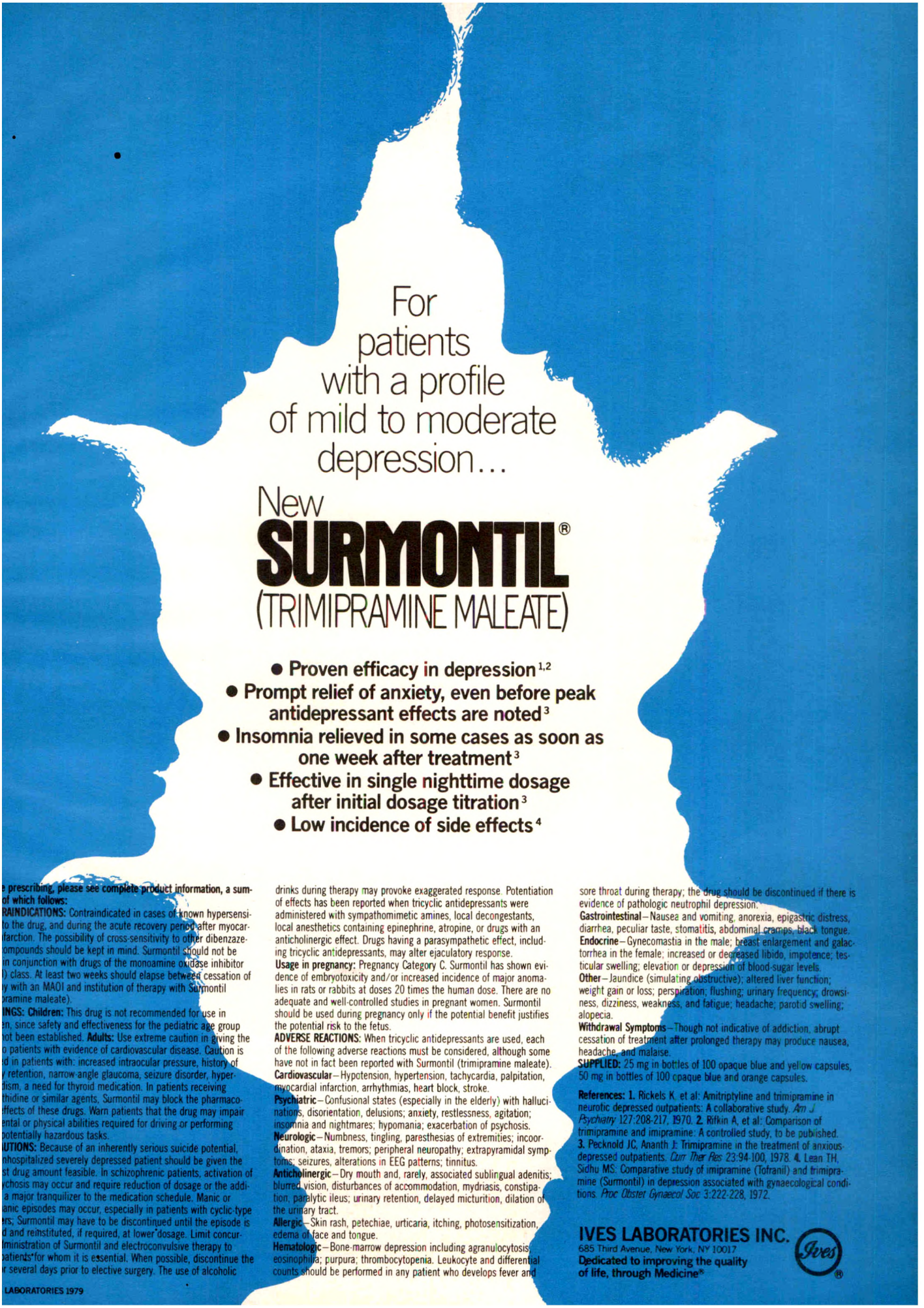
Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10 vials. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

Navane (thiothixene hydrochloride) Intramuscular For Injection is available in amber glass vials in packages of 10 vials. When reconstituted with 2.2 ml of Sterile Water for Injection, each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene, and 59.6 mg of mannitol. The reconstituted solution of Navane Intramuscular For Injection may be stored for 48 hours at room temperature before discarding.

References: 1. Stotsky BA: Relative efficacy of parenteral haloperidol and thiothixene for the emergency treatment of acutely excited and agitated patients. *Dis Nerv Syst* 38:967-973, December 1977. 2. Brauzer B, Goldstein BJ: Comparative effects of intramuscular thiothixene and trifluoperazine in psychotic patients. *J Clin Pharmacol* 8:400-403, November-December 1968. 3. Gallant DM, Bishop MP, Bishop C, et al: Thiothixene: A controlled evaluation of the intramuscular antipsychotic preparation. *Curr Ther Res* 10:561-565, November 1968.

For additional information on Navane, please consult your Roerig representative or write to: Roerig Medical Department, 235 East 42nd Street, New York, NY 10017.

ROERIG Pfizer
A division of Pfizer Pharmaceuticals
New York, New York 10017



e prescribing, please see complete product information, a sum- of which follows:

RAINDICATIONS: Contraindicated in cases of known hypersensi- to the drug, and during the acute recovery period after myocar- farction. The possibility of cross-sensitivity to other dibenzaze- ompounds should be kept in mind. Surmontil should not be in conjunction with drugs of the monoamine oxidase inhibitor I) class. At least two weeks should elapse between cessation of y with an MAOI and institution of therapy with Surmontil ramine maleate).

INGS: Children: This drug is not recommended for use in n, since safety and effectiveness for the pediatric age group ot been established. **Adults:** Use extreme caution in giving the o patients with evidence of cardiovascular disease. Caution is d in patients with: increased intraocular pressure, history of retention, narrow-angle glaucoma, seizure disorder, hyper- dism, a need for thyroid medication. In patients receiving thidine or similar agents, Surmontil may block the pharmaco- ffects of these drugs. Warn patients that the drug may impair ental or physical abilities required for driving or performing otentially hazardous tasks.

UTIONS: Because of an inherently serious suicide potential, nhospitalized severely depressed patient should be given the st drug amount feasible. In schizophrenic patients, activation of ychosis may occur and require reduction of dosage or the addi- a major tranquilizer to the medication schedule. Manic or anic episodes may occur, especially in patients with cyclic-type rs; Surmontil may have to be discontinued until the episode is d and reinstituted, if required, at lower dosage. Limit concur- dministration of Surmontil and electroconvulsive therapy to patients for whom it is essential. When possible, discontinue the r several days prior to elective surgery. The use of alcoholic drinks during therapy may provoke exaggerated response. Potentiation of effects has been reported when tricyclic antidepressants were administered with sympathomimetic amines, local decongestants, local anesthetics containing epinephrine, atropine, or drugs with an anticholinergic effect. Drugs having a parasympathetic effect, includ- ing tricyclic antidepressants, may alter ejaculatory response.

Usage in pregnancy: Pregnancy Category C. Surmontil has shown evi- dence of embryotoxicity and/or increased incidence of major anoma- lies in rats or rabbits at doses 20 times the human dose. There are no adequate and well-controlled studies in pregnant women. Surmontil should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

ADVERSE REACTIONS: When tricyclic antidepressants are used, each of the following adverse reactions must be considered, although some have not in fact been reported with Surmontil (trimipramine maleate).

Cardiovascular—Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric—Confusional states (especially in the elderly) with halluci- nations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurologic—Numbness, tingling, paresthesias of extremities; incoor- dination, ataxia, tremors; peripheral neuropathy; extrapyramidal symp- toms; seizures, alterations in EEG patterns; tinnitus.

Anticholinergic—Dry mouth and, rarely, associated sublingual adenitis; blurred vision, disturbances of accommodation, mydriasis, constipa- tion, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic—Skin rash, petechiae, urticaria, itching, photosensitization, edema of face and tongue.

Hematologic—Bone-marrow depression including agranulocytosis; eosinophilia; purpura; thrombocytopenia. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathologic neutrophil depression.

Gastrointestinal—Nausea and vomiting, anorexia, epigastric distress, diarrhea, peculiar taste, stomatitis, abdominal cramps, black tongue.

Endocrine—Gynecomastia in the male; breast enlargement and galac- torrhea in the female; increased or decreased libido, impotence; tes- ticular swelling; elevation or depression of blood-sugar levels.

Other—Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency; drowsi- ness, dizziness, weakness, and fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms—Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache, and malaise.

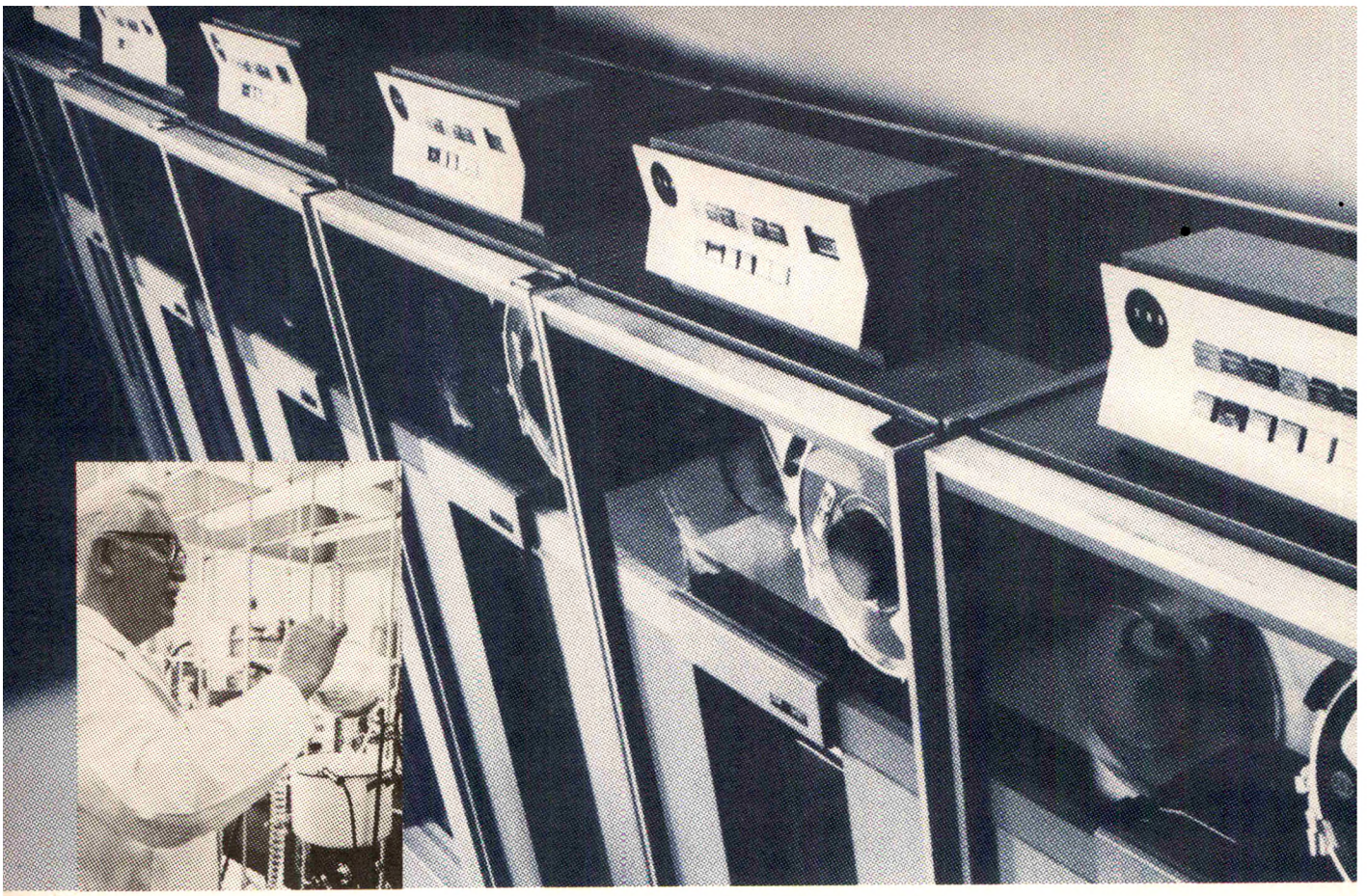
SUPPLIED: 25 mg in bottles of 100 opaque blue and yellow capsules, 50 mg in bottles of 100 opaque blue and orange capsules.

References: 1. Rickels K, et al: Amitriptyline and trimipramine in neurotic depressed outpatients: A collaborative study. *Am J Psychiatry* 127:208-217, 1970. **2.** Rifkin A, et al: Comparison of trimipramine and imipramine: A controlled study, to be published. **3.** Pecknold JC, Ananth J: Trimipramine in the treatment of anxious-depressed outpatients. *Curr Ther Res* 23:94-100, 1978. **4.** Lean TH, Sidhu MS: Comparative study of imipramine (Tofranil) and trimipra- mine (Surmontil) in depression associated with gynaecological condi- tions. *Proc Obstet Gynaecol Soc* 3:222-228, 1972.

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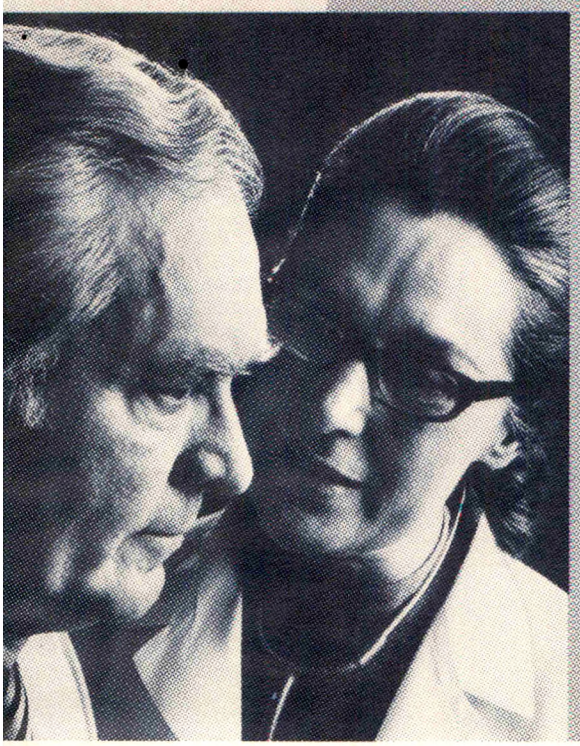
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Highly specific calming action virtually free of unwanted side effects: this was the remarkable clinical promise of Librium (chlordiazepoxide HCl). And today this promise continues to be fulfilled in a wide variety of patients you see every day.

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The published record on Librium is enormous. So large, in fact, it had to be put into a computer data bank and retrieval system. It's a record that shows Librium is highly effective in relieving anxiety; that Librium is seldom associated with serious side effects; that Librium rarely interferes with mental acuity at proper doses; that Librium is used concomitantly with primary medications. However, as with all CNS agents, patients should be warned against hazardous activities requiring complete alertness, and about possible combined effects with alcohol.

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- ☐ An unsurpassed safety record
- ☐ Minimal effect on mental acuity, in proper dosage
- ☐ Predictable patient response
- ☐ Is used concomitantly with primary medications, such as anticholinergics and cardiovascular drugs

Please see next page for summary of product information.

Librium® 5mg, 10mg, 25mg capsules
chlordiazepoxide HCl/Roche

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Relief of anxiety and tension occurring alone or accompanying various disease states. Efficacy beyond four months not established by systematic clinical studies. Periodic reassessment of therapy recommended.

Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Warn patients that mental and/or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

Usual Daily Dosage: Individualize for maximum beneficial effects. *Oral–Adults:* Mild and moderate anxiety and tension, 5 or 10 mg *t.i.d.* or *q.i.d.*; severe states, 20 or 25 mg *t.i.d.* or *q.i.d.* *Geriatric patients:* 5 mg *b.i.d.* to *q.i.d.* (See Precautions.)

Supplied: Librium® (chlordiazepoxide HCl) *Capsules*, 5 mg, 10 mg and 25 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available singly and in trays of 10. Libritabs® (chlordiazepoxide) *Tablets*, 5 mg, 10 mg and 25 mg—bottles of 100 and 500. With respect to clinical activity, capsules and tablets are indistinguishable.

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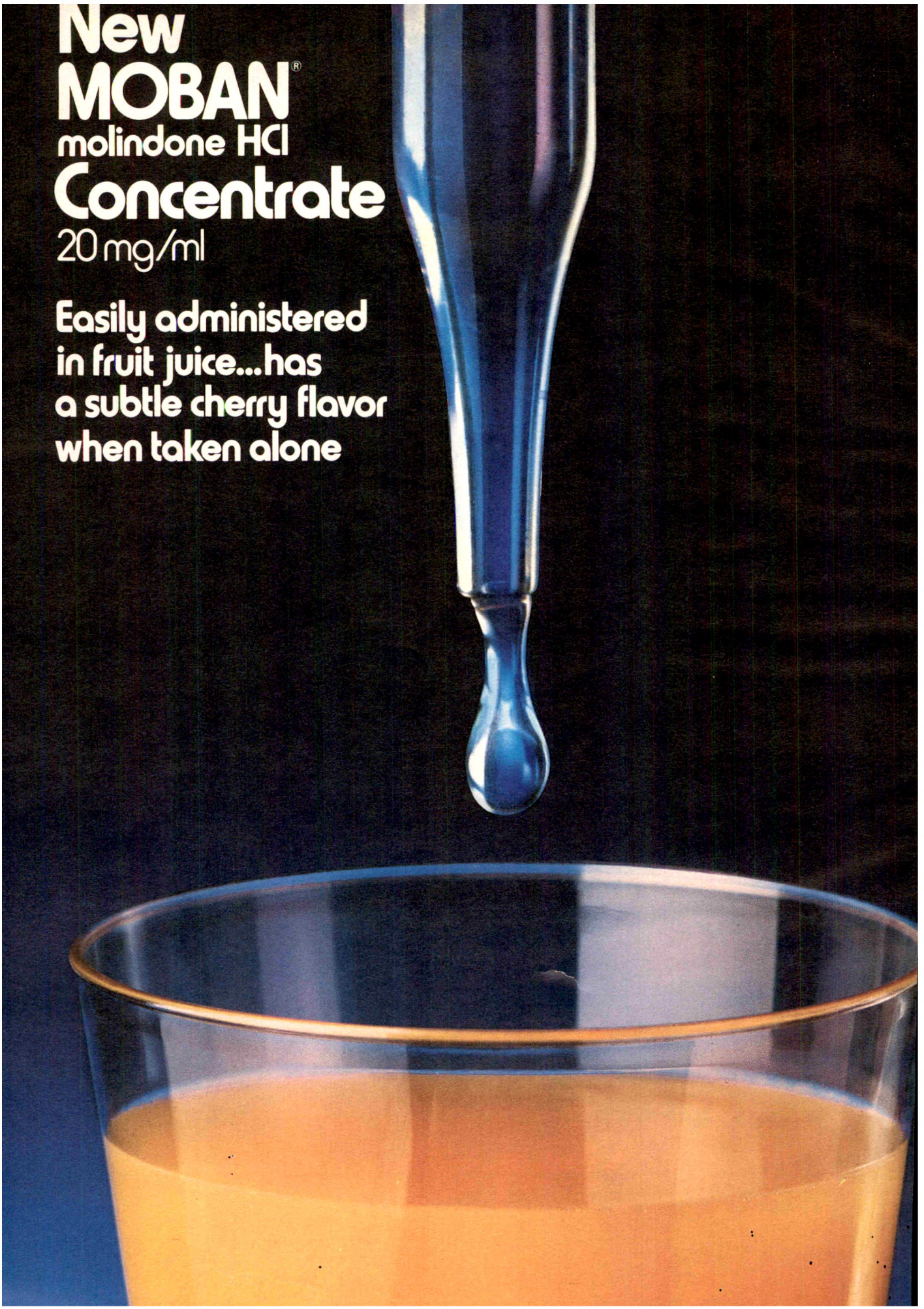
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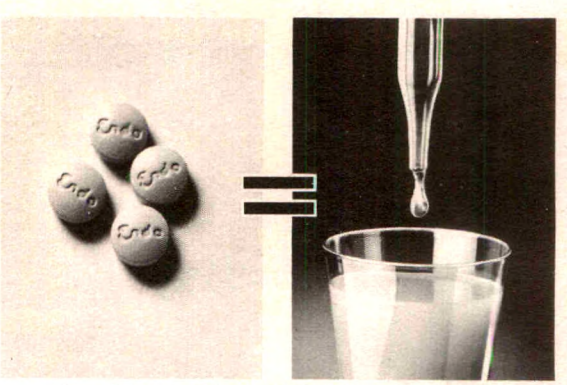
An effective antipsychotic agent with a favorable benefit/risk profile

NO incidence	excessive weight gain hepatotoxic changes ophthalmological changes persistent tardive dyskinesia skin pigmentation thyroid function changes tolerance development
LOW incidence	adverse drug interactions hematological changes hypotension nonpersistent symptoms of tardive dyskinesia sedation
MODERATE incidence	extrapyramidal reactions

This table compiled from clinical and field reports, according to the best available information.

For the schizophrenic patient

New MOBAN® molindone HCl Concentrate 20 mg/ml



four 25 mg tablets = 5 milliliters (100 mg)

Concentrated for maximum convenience of higher dosages...easy titration... enhanced compliance

Initial dosage with MOBAN®

- usual starting dose is 50-75 mg/day
- may be increased to 100 mg/day in three to four days
- may be adjusted up or down according to response
- some patients with severe symptomatology may require an increase to 225 mg/day
- elderly and debilitated patients should be started on lower dosage

Please see next page for prescribing information.

Endo Laboratories DUPONT

An effective antipsychotic agent

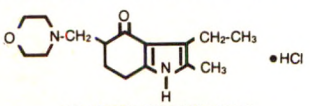
MOBAN® molindone HCl

New dosage form... Concentrate 20 mg/ml

continuing...Tablets 5 mg, 10 mg, 25 mg

DESCRIPTION MOBAN (molindone hydrochloride) is a dihydroindolone compound which is not structurally related to the phenothiazines, the butyrophenones or the thioxanthenes.

MOBAN is 3-ethyl-6, 7-dihydro-2-methyl-5-(morpholinomethyl) indol-4 (5H)-one hydrochloride. It is a white crystalline powder, freely soluble in water and alcohol and has a molecular weight of 312.67.



MOLINDONE HYDROCHLORIDE

ACTIONS MOBAN (molindone hydrochloride) has a pharmacological profile in laboratory animals which predominantly resembles that of major tranquilizers causing reduction of spontaneous locomotion and aggressiveness, suppression of a conditioned response and antagonism of the bizarre stereotyped behavior and hyperactivity induced by amphetamines. In addition, MOBAN antagonizes the depression caused by the tranquilizing agent tetrabenazine.

In human clinical studies tranquilization is achieved in the absence of muscle relaxing or incoordinating effects. Based on EEG studies, MOBAN exerts its effect on the ascending reticular activating system.

Human metabolic studies show MOBAN (molindone hydrochloride) to be rapidly absorbed and metabolized when given orally. Unmetabolized drug reached a peak blood level at 1.5 hours. Pharmacological effect from a single oral dose persists for 24-36 hours. There are 36 recognized metabolites with less than 2-3% unmetabolized MOBAN being excreted in urine and feces.

INDICATIONS MOBAN (molindone hydrochloride) is indicated in the management of the manifestations of schizophrenia.

CONTRAINDICATIONS MOBAN (molindone hydrochloride) is contraindicated in severe central nervous system depression (alcohol, barbiturates, narcotics, etc.) or comatose states, and in patients with known hypersensitivity to the drug.

WARNINGS Usage in Pregnancy: Studies in pregnant patients have not been carried out. Reproduction studies have been performed in the following animals:

Pregnant Rats oral dose— 20 mg/kg/day—10 days
no adverse effect
40 mg/kg/day—10 days
no adverse effect

Pregnant Mice oral dose— 20 mg/kg/day—10 days
slight increase resorptions
40 mg/kg/day—10 days
slight increase resorptions

Pregnant Rabbits oral dose— 5 mg/kg/day—12 days
no adverse effect
10 mg/kg/day—12 days
no adverse effect
20 mg/kg/day—12 days
no adverse effect

Animal reproductive studies have not demonstrated a teratogenic potential. The anticipated benefits must be weighed against the unknown risks to the fetus if used in pregnant patients.

Nursing Mothers: Data are not available on the content of MOBAN (molindone hydrochloride) in the milk of nursing mothers.

Usage in Children: Use of MOBAN (molindone hydrochloride) in children below the age of twelve years is not recommended because safe and effective conditions for its usage have not been established.

MOBAN has not been shown effective in the management of behavioral complications in patients with mental retardation.

PRECAUTIONS Some patients receiving MOBAN (molindone hydrochloride) may note drowsiness initially and they should be advised against activities requiring mental alertness until their response to the drug has been established.

Increased activity has been noted in patients receiving MOBAN. Caution should be exercised where increased activity may be harmful.

MOBAN does not lower the seizure threshold in experimental animals to the degree noted with more sedating antipsychotic drugs. However, in humans convulsive seizures have been reported in a few instances.

EDO 854M580

The physician should be aware that this tablet preparation contains calcium sulfate as an excipient and that calcium ions may interfere with the absorption of preparations containing phenytoin sodium and tetracyclines.

MOBAN (molindone hydrochloride) has an antiemetic effect in animals. A similar effect may occur in humans and may obscure signs of intestinal obstruction or brain tumor.

ADVERSE REACTIONS—CNS EFFECTS The most frequently occurring effect is initial drowsiness that generally subsides with continued usage of the drug or lowering of the dose.

Noted less frequently were depression, hyperactivity and euphoria.

Neurological—Extrapyramidal Reactions Extrapyramidal reactions noted below may occur in susceptible individuals and are usually reversible with appropriate management.

Akathisia Motor restlessness may occur early.

Parkinson Syndrome Akinesia, characterized by rigidity, immobility and reduction of voluntary movements and tremor, have been observed. Occurrence is less frequent than akathisia.

Dystonic Syndrome Prolonged abnormal contractions of muscle groups occur infrequently. These symptoms may be managed by the addition of a synthetic antiparkinson agent (other than L-dopa), small doses of sedative drugs, and/or reduction in dosage.

Autonomic Nervous System Occasionally blurring of vision, tachycardia, nausea, dry mouth and salivation have been reported. Urinary retention and constipation may occur particularly if anticholinergic drugs are used to treat extrapyramidal symptoms.

Hematological There have been rare reports of leucopenia and leucocytosis. If such reactions occur, treatment with MOBAN may continue if clinical symptoms are absent. Alterations of blood glucose, liver function tests, B.U.N., and red blood cells have not been considered clinically significant.

Metabolic and Endocrine Effects Alteration of thyroid function has not been significant. Amenorrhea has been reported infrequently. Resumption of menses in previously amenorrheic women has been reported. Initially heavy menses may occur. Galactorrhea and gynecomastia have been reported infrequently. Increase in libido has been noted in some patients. Impotence has not been reported. Although both weight gain and weight loss have been in the direction of normal or ideal weight, excessive weight gain has not occurred with MOBAN.

Cardiovascular Rare, transient, non-specific T wave changes have been reported on E.K.G. Association with a clinical syndrome has not been established. Rarely has significant hypotension been reported.

Ophthalmological Lens opacities and pigmentary retinopathy have not been reported where patients have received MOBAN (molindone hydrochloride). In some patients, phenothiazine induced lenticular opacities have resolved following discontinuation of the phenothiazine while continuing therapy with MOBAN.

Skin Early, non-specific skin rash, probably of allergic origin, has occasionally been reported. Skin pigmentation has not been seen with MOBAN usage alone.

MOBAN (molindone hydrochloride) has certain pharmacological similarities to other antipsychotic agents. Because adverse reactions are often extensions of the pharmacological activity of a drug, all of the known pharmacological effects associated with other antipsychotic drugs should be kept in mind when MOBAN is used. Upon abrupt withdrawal after prolonged high dosage an abstinence syndrome has not been noted.

Tardive Dyskinesia Although rarely reported with MOBAN (molindone hydrochloride) symptoms were reversible upon discontinuation of therapy.

Tardive dyskinesia associated with other agents has appeared in some patients on long-term therapy and has also appeared after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). There may be involuntary movements of extremities.

There is no known effective treatment of tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time the syndrome may not develop.

DOSAGE AND ADMINISTRATION Initial and maintenance doses of MOBAN (molindone hydrochloride) should be individualized.

Initial Dosage Schedule The usual starting dosage is 50-75 mg/day.
- —Increase to 100 mg/day in 3 or 4 days.
- —Based on severity of symptomatology, dosage may be titrated up or down depending on individual patient response.
- —An increase to 225 mg/day may be required in patients with severe symptomatology.

Elderly and debilitated patients should be started on lower dosage.

Maintenance Dosage Schedule
1. Mild—5 mg-15 mg three or four times a day.
2. Moderate—10 mg-25 mg three or four times a day.
3. Severe—225 mg/day may be required.

DRUG INTERACTIONS Potentiation of drugs administered concurrently with MOBAN (molindone hydrochloride) has not been reported. Additionally, animal studies have not shown increased toxicity when MOBAN is given concurrently with representative members of three classes of drugs (i.e., barbiturates, chloral hydrate and antiparkinson drugs).

MANAGEMENT OF OVERDOSAGE Symptomatic, supportive therapy should be the rule.

Gastric lavage is indicated for the reduction of absorption of MOBAN (molindone hydrochloride) which is freely soluble in water.

Since the adsorption of MOBAN (molindone hydrochloride) by activated charcoal has not been determined, the use of this antidote must be considered of theoretical value.

Emesis in a comatose patient is contraindicated. Additionally, while the emetic effect of apomorphine is blocked by MOBAN in animals, this blocking effect has not been determined in humans.

A significant increase in the rate of removal of unmetabolized MOBAN (molindone hydrochloride) from the body by forced diuresis, peritoneal or renal dialysis would not be expected. (Only 2% of a single ingested dose of MOBAN is excreted unmetabolized in the urine.)

However, poor response of the patient may justify use of these procedures. While the use of laxatives or enemas might be based on general principles, the amount of unmetabolized MOBAN in feces is less than 1%. Extrapyramidal symptoms have responded to the use of diphenhydramine (Benadryl*) and the synthetic anticholinergic antiparkinson agents, (i.e., Artane*, Cogentin*, Akineton*).

HOW SUPPLIED As tablets in bottles with potencies and colors as follows:

100's and 1000's:
5 mg orange
10 mg lavender
25 mg light green

As a concentrate containing 20 mg molindone hydrochloride per ml in 4 oz. (120ml) bottles.

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1. Sedation reported in normal volunteers 30 minutes after dosing. Tranxene Drug Monograph 97-0185, p. 9, 1979.
2. The elimination kinetics of an agent can be closely defined, but these cannot at present be directly related to therapeutic or adverse effects.

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Brief Summary

INDICATIONS — Symptomatic relief of anxiety and tension associated with anxiety disorders, other psychoneurotic disorders, transient situational disturbances, and functional or organic disorders. Symptomatic relief of acute alcohol withdrawal.

Effectiveness in long-term use (over 4 months) not assessed by systematic clinical studies. Physician should periodically reassess usefulness for each patient.

CONTRAINDICATIONS — Known hypersensitivity to the drug. Acute narrow angle glaucoma.

WARNINGS — Not for use in depressive neuroses or psychotic reactions. Caution patients against hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles. Advise against simultaneous use of other CNS depressants, and caution patients that effects of alcohol may be increased. Not recommended for patients under 18. Nervousness, insomnia, irritability, diarrhea, muscle aches, and memory impairment have followed abrupt withdrawal from long-term high dosage. Withdrawal symptoms were reported after abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months. Use caution in patients having psychological potential for drug dependence (dependence has been observed in dogs and rabbits).

Pregnancy and Lactation: Minor tranquilizers should almost always be avoided first trimester. Consider possibility of pregnancy before initiating therapy. Patient should consult physician about discontinuation if she becomes pregnant or plans pregnancy. Do not give to nursing mothers.

PRECAUTIONS — Observe usual precautions in depression accompanying anxiety, or in patients with suicidal tendency, or those with impaired renal or hepatic function. Do periodic blood counts and liver function tests during prolonged therapy. Use small doses and gradual increments in the elderly or debilitated.

ADVERSE REACTIONS — Drowsiness, dizziness, various g.i. complaints, nervousness, blurred vision, dry mouth, headache, mental confusion, insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression, slurred speech, abnormal liver and kidney function, decreased hematocrit, decreased systolic blood pressure.

DOSAGE — ANXIETY—Usual daily dose 30 mg or less (start the elderly or debilitated at 7.5-15 mg). Adjust gradually within 15-60 mg daily range. Capsules and scored tablets: divided doses; or once daily h.s. (start patient at 15 mg). Single Dose Tablets, 22.5 mg (for patients stabilized on 7.5 mg t.i.d.) or 11.25 mg: once daily at any hour. ALCOHOL WITHDRAWAL—In divided doses: 1st day 30 mg initially, then 30-60 mg; 2nd day 45-90 mg; 3rd day 22.5-45 mg; 4th day 15-30 mg. Then taper to 15-7.5 mg daily, and discontinue as soon as stable.

INTERACTIONS — Potentiation may occur with ethyl alcohol, hypnotics, barbiturates, narcotics, phenothiazines, MAO inhibitors, other antidepressants.

OVERDOSAGE — Take general measures as for any CNS depressant.

SUPPLIED — Tranxene (clorazepate dipotassium) 3.75, 7.5, and 15 mg capsules and scored tablets. Tranxene-SD Half Strength 11.25 and Tranxene-SD 22.5 mg single dose tablets.

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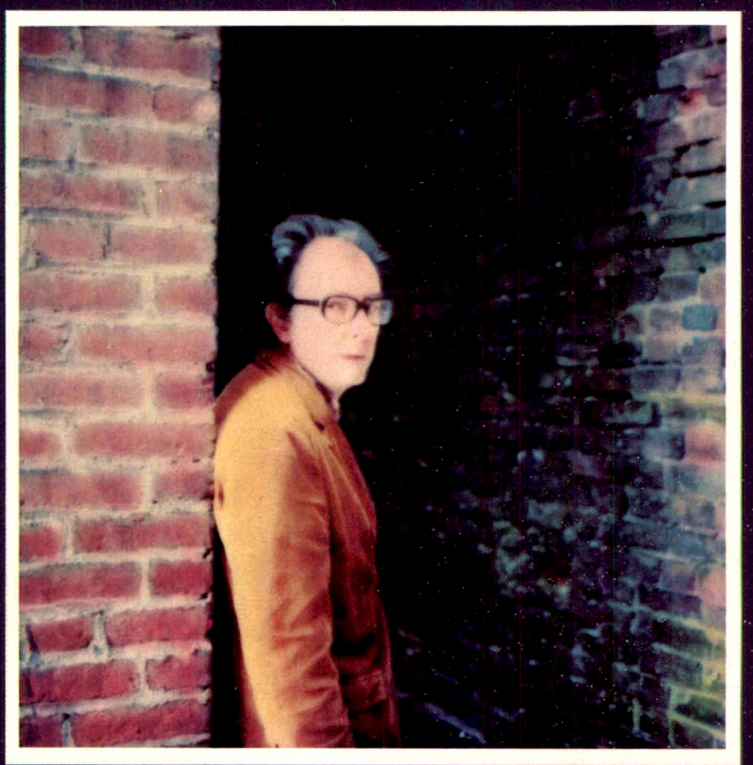
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Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Effective: For the management of the manifestations of psychotic disorders.

Possibly effective: To control excessive anxiety, tension and agitation as seen in neuroses or associated with somatic conditions.

'Stelazine' has not been shown effective in the management of behavioral complications in patients with mental retardation.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose or greatly depressed states due to C.N.S. depressants; blood dyscrasias; bone marrow depression; liver damage.

Warnings: Generally avoid using in patients hypersensitive (e.g., blood dyscrasias, jaundice) to any phenothiazine. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery), especially during the first few days' therapy.

Additive effect is possible with other C.N.S. depressants, including alcohol.

Use in pregnancy only when necessary for patient's welfare.

Precautions: Use cautiously in angina. Avoid high doses and parenteral administration when cardiovascular system is impaired. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Prolonged administration of high doses may result in cumulative effects with severe C.N.S. or vasomotor symptoms. If retinal changes occur, discontinue drug. Agranulocytosis, thrombocytopenia, pancytopenia, anemia, cholestatic jaundice, liver damage have been reported.

Patients with a history of long-term therapy with 'Stelazine' and/or other neuroleptics should be evaluated periodically for possible adjustment or discontinuance of drug therapy. Neuroleptic drugs cause elevated prolactin levels that persist during chronic administration. Since approximately one-third of human breast cancers are prolactin-dependent in vitro, this elevation is of potential importance if neuroleptic drug administration is contemplated in a patient with a previously detected breast cancer. Neither clinical nor epidemiologic studies to date, however, have shown an association between the chronic administration of neuroleptic drugs and mammary tumorigenesis.

Adverse Reactions: Drowsiness, dizziness, skin reactions, rash, dry mouth, insomnia, amenorrhea, fatigue, muscular weakness, anorexia, lactation, blurred vision. Neuromuscular (extrapyramidal) reactions: motor restlessness, dystonias, pseudoparkinsonism, persistent tardive dyskinesia.

Other adverse reactions reported with Stelazine (trifluoperazine HCl, SK&F) or other phenothiazines: Some adverse effects are more frequent or intense in specific disorders (e.g., mitral insufficiency or pheochromocytoma).

Grand mal convulsions; altered cerebrospinal fluid proteins; cerebral edema; prolongation and intensification of the action of C.N.S. depressants, atropine, heat, and organophosphorus insecticides; nasal congestion, headache, nausea, constipation, obstipation, adynamic ileus, inhibition of ejaculation; reactivation of psychotic processes, catatonic-like states; hypotension (sometimes fatal); cardiac arrest; leukopenia, eosinophilia, pancytopenia, agranulocytosis, thrombocytopenic purpura; jaundice, biliary stasis; menstrual irregularities, galactorrhea, gynecomastia, false positive pregnancy tests; photosensitivity, itching, erythema, urticaria, eczema up to exfoliative dermatitis; asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions, peripheral edema; reversed epinephrine effect; hyperpyrexia; a systemic lupus erythematosus-like syndrome; pigmentary retinopathy; with prolonged administration of substantial doses, skin pigmentation, epithelial keratopathy, and lenticular and corneal deposits. EKG changes have been reported, but relationship to myocardial damage is not confirmed. Discontinue long-term, high-dose therapy gradually. NOTE: Sudden death in patients taking phenothiazines (apparently due to cardiac arrest or asphyxia due to failure of cough reflex) has been reported, but no causal relationship has been established.

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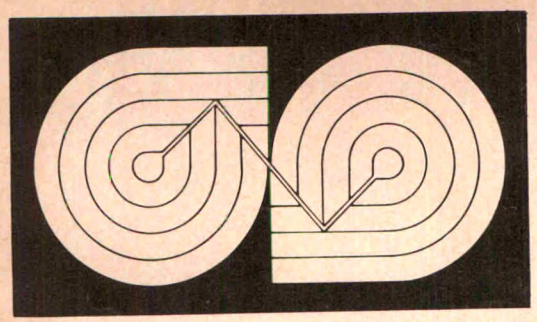
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The length of submitted material should not exceed the following specifications unless a special arrangement has been made with the Editor. Regular articles—3,800 words or the equivalent **(no more than 15 double-spaced pages, including references, tables, and figures).** Brief Communications—2,500 words **(no more than 10 double-spaced pages, including references, tables, and figures).** Clinical and Research Reports—1,000 words, 10 references, 1 table (no figures are used). Letters to the Editor—500 words, 5 references. The number of words, tables, and figures should be noted on the title page.

TYPES OF ARTICLES

Overviews

Overview articles attempt to bring together important relevant information on a topic of general interest to psychiatry. They are usually written at the invitation of the Editor; authors who have ideas for overview articles are advised to check with the Editor to ensure that a similar work is not in preparation. Overviews should not exceed 7,500 words **(no more than 25 double-spaced pages—including tables and figures—and 100 references).** All overviews, including those written by invitation, are given the same peer review received by other papers.

Regular Articles and Brief Communications

The primary difference between these two types of papers is length. The "brief communication" designation does not imply less sophisticated or complete work: it merely means that the content can be expressed within the upper limit of 2,500 words. There is no difference in the review procedure or scheduling of these articles. Authors who submit unnecessarily long articles with redundancies and loose writing style will be asked to shorten them, which can delay publication. Authors should choose their words carefully, avoiding lengthy introductions containing material that is common knowledge, summaries that merely repeat the results, unnecessary tables, figures, or references, etc. Single case reports should be submitted as Clinical and Research Reports.

Clinical and Research Reports

This section contains very brief articles (maximum of 1,000 words, 10 references, 1 table, and **no figures**) reporting new research findings, including preliminary data from pilot studies, and case reports that 1) describe new syndromes, 2) cast a new light on established ones, 3) indicate a new therapeutic procedure of potential value, or 4) describe adverse effects or previously unreported complications of drugs or therapeutic interventions. Because of the stringent criteria for this section, authors can expect more rapid publication than is possible in other sections. Submitted papers that substantially exceed the stated maximum length or contain figures will be returned to the author unreviewed.

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Articles submitted for prompt publication can be of either regular of Brief Communication length. (A "prompt publication policy" is automatically in effect for Clinical and Research Reports.) These papers are given priority in scheduling; however, authors should be aware that the minimum publication time is four months. Prompt publication papers must meet stringent criteria of originality and be of major immediate importance to the field. Authors must state their reasons for wanting rapid review in a cover letter to the Editor. It is important to think carefully about the nature of the paper before requesting prompt publication; a paper may be delayed if it is submitted inappropriately.

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Acknowledgments. Acknowledgments should be in a separate paragraph on the title page. Grant support should include the full name of the granting agency as well as the grant number. Individual acknowledgments should be as brief as possible. Acknowledgments of companies that supplied drugs are used only in the case of experimental drugs or those unavailable in this country.

Other requirements. The number of words, tables, and figures should appear in the upper right corner and a phone number for the corresponding author in the lower right.

Précis

The précis should be up to 100 words for regular articles and Brief Communications. (No précis is used in the Clinical and Research Reports section.) The précis should be a single paragraph using complete, connected sentences, active verbs, and the third person. In most cases, the précis replaces a summary.

Text

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1. Berne E: Principles of Group Treatment. New York, Oxford University Press, 1966, p 26
2. Blackwell B, Marley E, Price J, et al: Hypertensive interactions between monoamine oxidase inhibitors and foodstuffs. Br J Psychiatry 113:349-365, 1967
3. Jones AB: Long-term administration of tricyclic antidepressants: three-year follow-up. Am J Psychiatry (in press)
4. Smythe JCW (ed): Psychiatry and Human Values. Springfield, Ill, Charles C Thomas, 1971
5. Brosin H: Communication systems of the consultation process, in The Psychiatric Consultation. Edited by Mendel W, Solomon P. New York, Grune & Stratton, 1968

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Figures should be used only for data that cannot be adequately presented in text or tabular form. Figures should be submitted as glossy prints **with one photocopy attached to each manuscript copy.** The author's name and the title of the paper should appear on a gummed label affixed to the back of each glossy. All figures should be able to withstand reduction to about 8 cm (3 1/4 inches). Authors are urged to consider carefully the necessity of figures; they may be asked to delete figures that repeat information availabile in text. It is often advisable to obtain professional assistance in the preparation of figures.

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Jonathan O. Cole, M.D.

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For all practicing psychiatrists who wish to keep up to date on the uses of psychopharmacological agents and their potential side effects, this book offers a clear discussion of a range of available drugs. The information is practical and provides enough background for clinicians to practice psychopharmacology for the greatest benefit of patients. Newer agents such as lecithin, L-tryptophan, and fenfluramine are described. The book reviews treatment of such conditions as senile dementia and adult minimal brain dysfunction, and discusses such side effects of drugs as memory difficulties caused by tricyclic antidepressants, kidney changes due to lithium, and agranulocytosis resulting from antipsychotic drugs.

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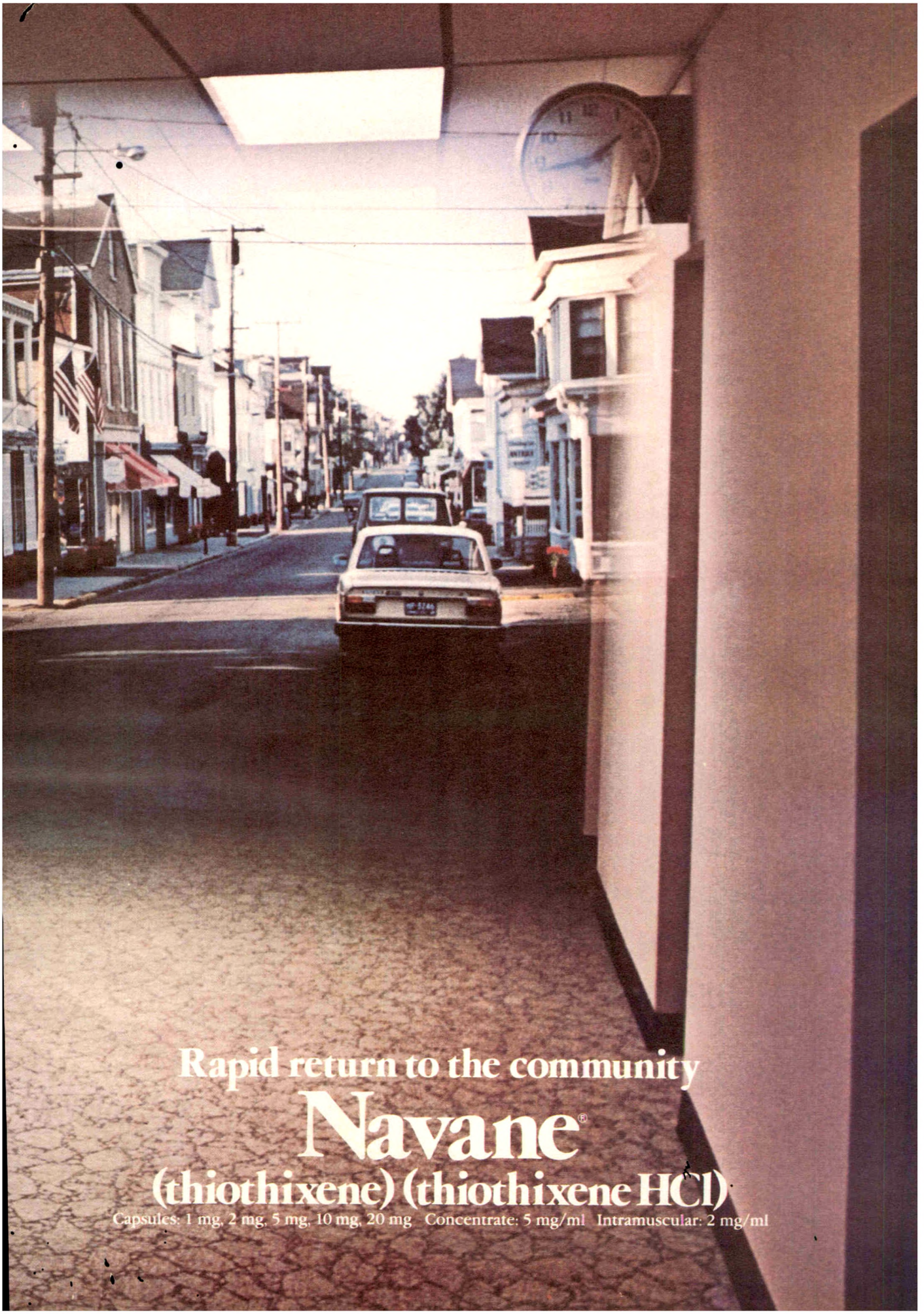
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Warnings. *Usage in Pregnancy*– Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration of Navane to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children– The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration– As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane (thiothixene). Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudo-parkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leukocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution–For Intramuscular Use Only. Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane (thiothixene) Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate– In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Stimulants that may cause convulsions (e.g. picrotoxin or pentylenetetrazol) should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg of thiothixene in bottles of 100, 1,000, and unit-dose pack of 100 (10 x 10's). Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100, 500, and unit-dose pack of 100 (10 x 10's).

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg, and in 30 ml (1 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, and 5 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10 vials. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

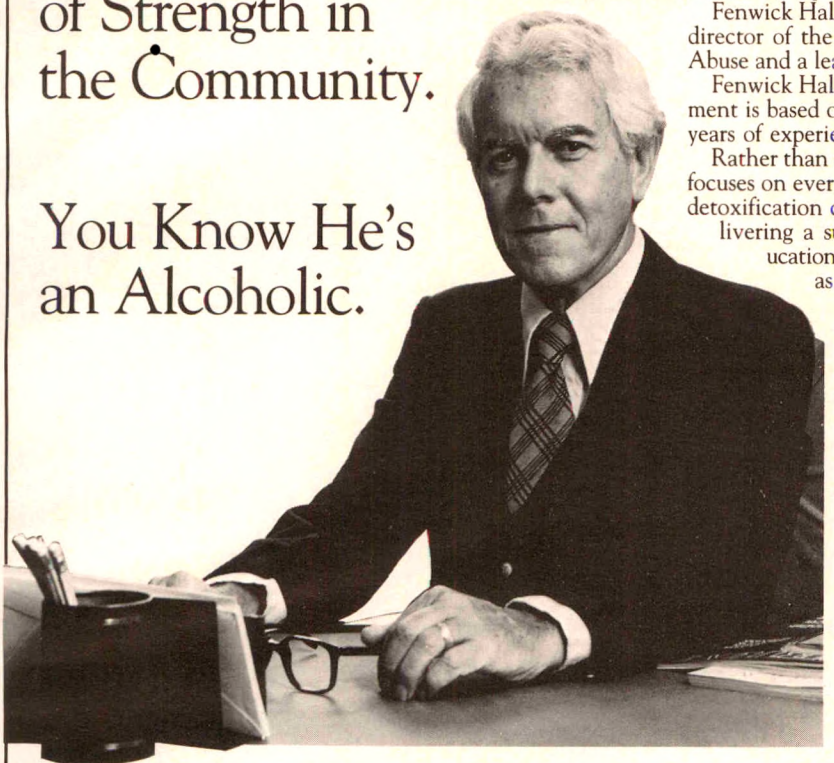
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For additional information on Navane, please consult your Roerig representative or write to: Roerig Medical Department, 235 East 42nd Street, New York, NY 10017.

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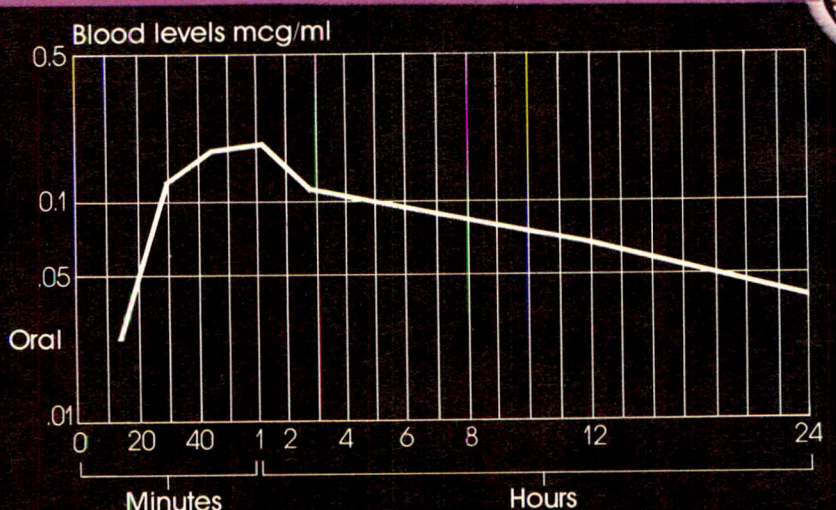
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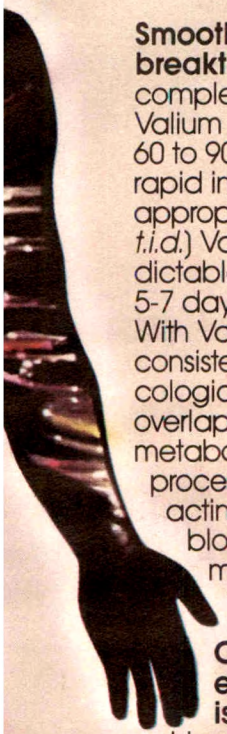
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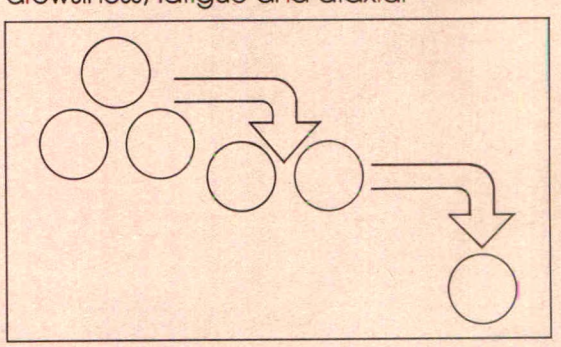
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Indications: Management of anxiety disorders, or short-term relief of symptoms of anxiety; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).
The effectiveness of Valium in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice, periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Anxiety disorders, symptoms of anxiety, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

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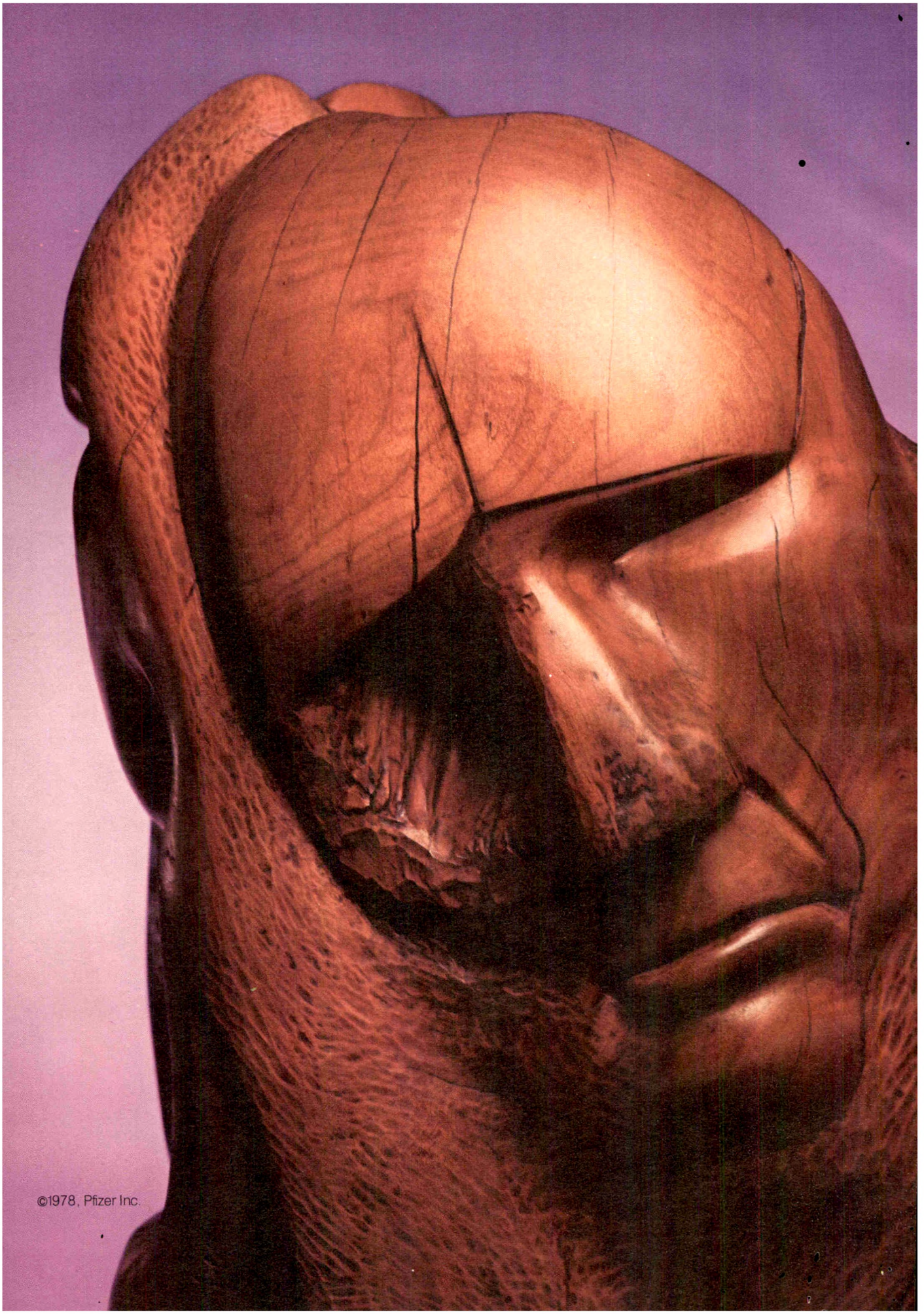
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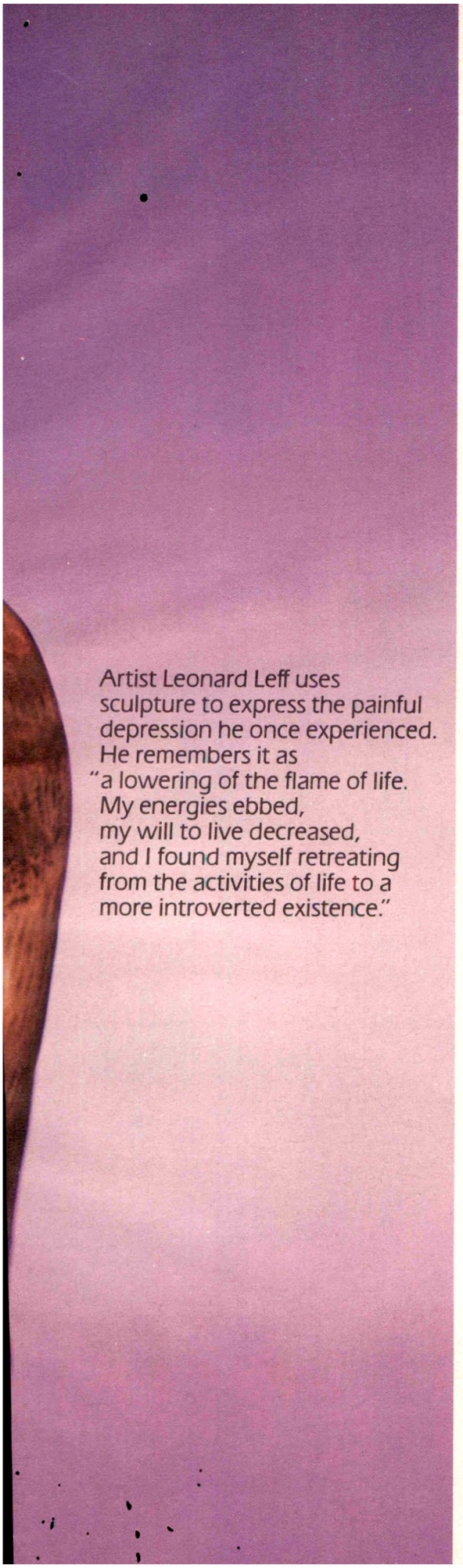
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See Brief Summary on next page for information on contraindications, warnings, precautions and adverse reactions.

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PROMINENT SEDATIVE EFFECT

which may help to relieve the difficulty in falling and staying asleep, and the early-morning awakening often associated with depression.

ESTABLISHED ANTIANXIETY ACTIVITY

to help alleviate the anxiety which often accompanies clinical depression.

USUALLY WELL TOLERATED

At doses up to 150 mg per day, Sinequan does not generally affect the antihypertensive activity of guanethidine and related compounds. Tachycardia and hypotension have been reported occasionally. Drowsiness is the most commonly observed side effect. Dry mouth, blurred vision, constipation and urinary retention have been reported.

EXTENDED RANGE OF DOSAGE STRENGTHS

for flexibility in individualizing therapy.

BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate

Contraindications. Contraindicated in individuals who have shown hypersensitivity to the drug, and in patients with glaucoma or a tendency to urinary retention. These disorders should be ruled out, particularly in older patients. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind.

Warnings. The once-a-day dosage regimen of SINEQUAN (doxepin HCl) in patients with intercurrent illness or patients taking other medications should be carefully adjusted. This is especially important in patients receiving other medications with anticholinergic effects.

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies performed in animals have shown no evidence of harm to the animal fetus. Since there is no experience in pregnant women receiving this drug, safety in pregnancy has not been established. There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.

Usage in Children: Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors. Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug. The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered and the dosage involved.

Usage with Alcohol: It should be borne in mind that alcohol ingestion may increase the danger inherent in any intentional or unintentional SINEQUAN overdosage. This is especially important in patients who may use alcohol excessively.

Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug.

Patients should also be cautioned that their response to alcohol may be potentiated.

Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy. Prescriptions should be written for the smallest feasible amount.

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.

Adverse Reactions. NOTE: Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN.

Anticholinergic Effects: Dry mouth, blurred vision, constipation, and urinary retention have been reported. If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage.

Central Nervous System Effects: Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued. Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, numbness, paresthesias, ataxia, and extrapyramidal symptoms and seizures.

Cardiovascular: Cardiovascular effects including hypotension and tachycardia have been reported occasionally.

Allergic: Skin rash, edema, photosensitization, and pruritus have occasionally occurred.

Hematologic: Eosinophilia has been reported in a few patients. There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura.

Gastrointestinal: Nausea, vomiting, indigestion, taste disturbances, diarrhea, anorexia, and aphthous stomatitis have been reported. (See anticholinergic effects.)

Endocrine: Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.

Other: Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice, alopecia, and headache have been occasionally observed as adverse effects.

Dosage and Administration. For most patients with illness of mild to moderate severity, starting daily dose of 75 mg is recommended. Dosage may subsequently be increased decreased at appropriate intervals and according to individual response. The usual optimu dose range is 75 mg/day to 150 mg/day.

In more severely ill patients higher doses may be required with subsequent gradual increase 300 mg/day if necessary. Additional therapeutic effect is rarely to be obtained by exceeding dose of 300 mg/day.

In patients with very mild symptomatology or emotional symptoms accompanying organ disease, lower doses may suffice. Some of these patients have been controlled on doses as lo as 25-50 mg/day.

The total daily dosage of SINEQUAN (doxepin HCl) may be given on a divided or once-a-d dosage schedule. If the once-a-day schedule is employed the maximum recommended dose 150 mg/day. This dose may be given at bedtime. **The 150 mg capsule strength is intended f maintenance therapy only and is not recommended for initiation of treatment.**

Antianxiety effect is apparent before the antidepressant effect. Optimal antidepressant effe may not be evident for two to three weeks.

Overdosage.

A. Signs and Symptoms

1. Mild: Drowsiness, stupor, blurred vision, excessive dryness of mouth.

2. Severe: Respiratory depression, hypotension, coma, convulsions, cardiac arrhythmias a tachycardias.

Also: urinary retention (bladder atony), decreased gastrointestinal motility (paralytic ileu hyperthermia (or hypothermia), hypertension, dilated pupils, hyperactive reflexes.

B. Management and Treatment

1. Mild: Observation and supportive therapy is all that is usually necessary.

2. Severe: Medical management of severe SINEQUAN overdosage consists of aggressi supportive therapy. If the patient is conscious, gastric lavage, with appropriate precautions prevent pulmonary aspiration, should be performed even though SINEQUAN is rapidly absorb The use of activated charcoal has been recommended, as has been continuous gastric lava with saline for 24 hours or more. An adequate airway should be established in comatose pati and assisted ventilation used if necessary. EKG monitoring may be required for several da since relapse after apparent recovery has been reported. Arrhythmias should be treated with t appropriate antiarrhythmic agent. It has been reported that many of the cardiovascular and C symptoms of tricyclic antidepressant poisoning in adults may be reversed by the slow int venous administration of 1 mg to 3 mg of physostigmine salicylate. Because physostigmine rapidly metabolized, the dosage should be repeated as required. Convulsions may respond standard anticonvulsant therapy; however, barbiturates may potentiate any respiratory depr sion. Dialysis and forced diuresis generally are not of value in the management of overdosa due to high tissue and protein binding of SINEQUAN.

Supply. SINEQUAN is available as capsules containing doxepin HCl equivalent to: 10 mg, 75 r and 100 mg doxepin: bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's). 25 mg a 50 mg doxepin: bottles of 100, 1000, 5000, and unit-dose packages of 100 (10 x 10's). 150 doxepin: bottles of 50, 500, and unit-dose packages of 100 (10 x 10's). SINEQUAN O Concentrate (10 mg/ml) is available in 120 ml bottles with an accompanying dropper calibrat at 5 mg, 10 mg, 15 mg, 20 mg, and 25 mg. Each ml contains doxepin HCl equivalent to 10 doxepin. Just prior to administration, SINEQUAN Oral Concentrate should be diluted w approximately 120 ml of water, whole or skimmed milk, or orange, grapefruit, tomato, prune pineapple juice. SINEQUAN Oral Concentrate is not physically compatible with a numbe carbonated beverages. For those patients requiring antidepressant therapy who are methadone maintenance, SINEQUAN Oral Concentrate and methadone syrup can be mi together with Gatorade®, lemonade, orange juice, sugar water, Tang®, or water; but not w grape juice. Preparation and storage of bulk dilutions is not recommended.

More detailed professional information available on request.

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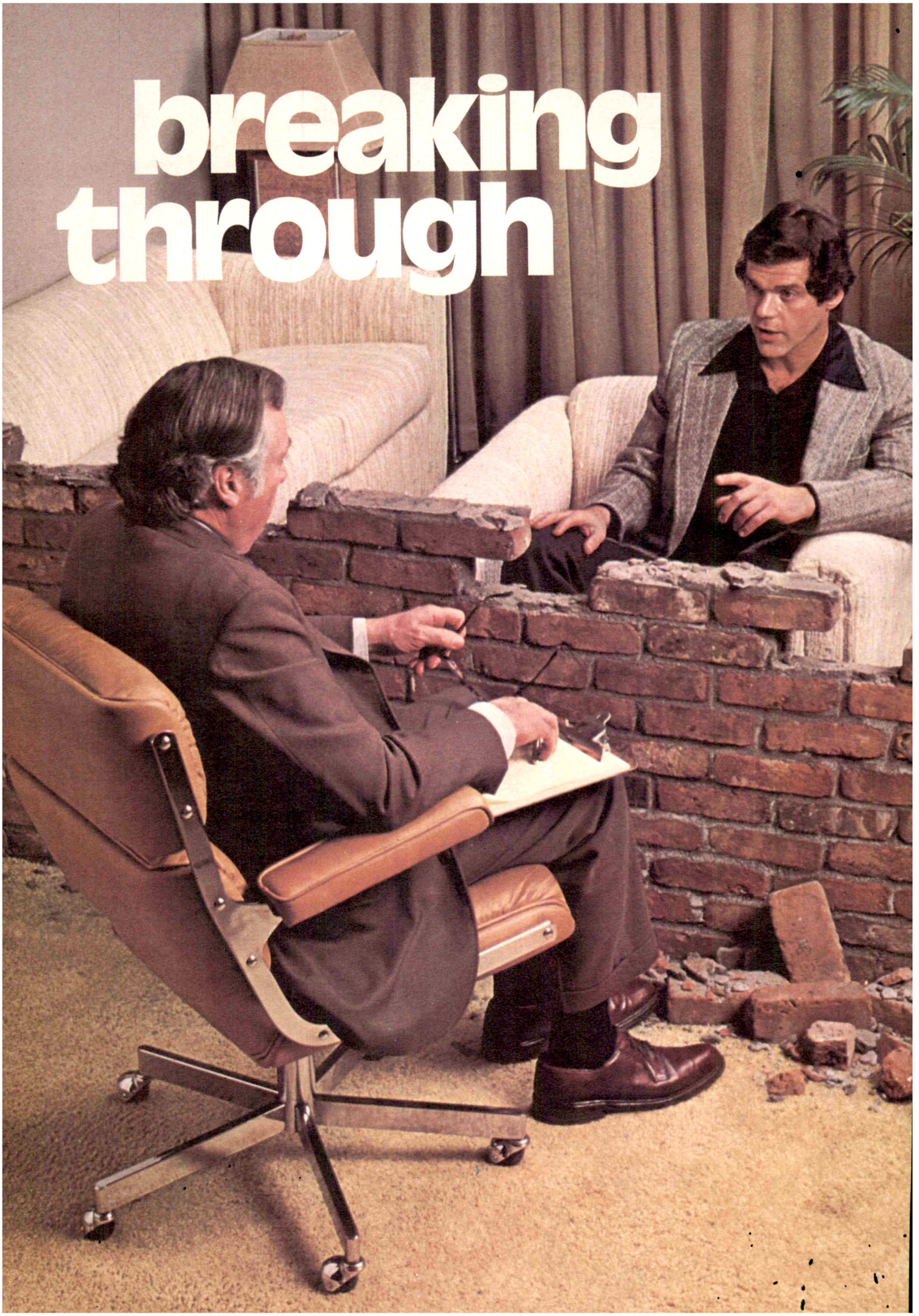
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breaking
through

In marked agitation with depression symptomatic relief may make the patient more accessible and responsive.

Rapid relief of marked agitation in many patients
The tranquilizer component alleviates symptoms of agitation and anxiety within a few days, without apparent dulling of mental acuity. Hypnotic effects from the tranquilizer component appear to be minimal, particularly in patients permitted to remain active. However, TRIAVIL may impair mental and/or physical abilities required for performance of hazardous tasks.

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The antidepressant component relieves symptoms of depression such as poor concentration and feelings of hopelessness as well as early morning awakening; adequate relief of symptoms may take a few weeks or even longer.

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There are now *five* tablet strengths of TRIAVIL for ease of dosage adjustment. For many patients with agitation and depression, you can now initiate therapy with one TRIAVIL® 4-50, containing 4 mg perphenazine and 50 mg amitriptyline HCl, b.i.d. The regimen is simple, economical, and may well enhance patient compliance.

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For marked agitation with depression

dual-action **Triavil®**

containing perphenazine and amitriptyline HCl



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Please see following page for a brief summary of prescribing information.

More dosage strengths than any other formulation containing a tranquilizer and an antidepressant

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TRIAVIL® 2-25: Each tablet contains
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TRIAVIL® 4-10: Each tablet contains
4 mg perphenazine and 10 mg amitriptyline HCl.

CONTRAINDICATIONS: Central nervous system depression from drugs (barbiturates, alcohol, narcotics, analgesics, antihistamines); evidence of bone marrow depression; known hypersensitivity to phenothiazines or amitriptyline. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred from such combinations. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with TRIAVIL. Therapy should then be initiated cautiously with gradual increase in dosage until optimum response is achieved. Not recommended for use during acute recovery phase following myocardial infarction.

WARNINGS: TRIAVIL should not be given concomitantly with guanethidine or similarly acting compounds since TRIAVIL may block the antihypertensive action of such compounds. Use cautiously in patients with history of urinary retention, angle-closure glaucoma, increased intraocular pressure, or convulsive disorders. Dosage of anticonvulsive agents may have to be increased. In patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely. Tricyclic antidepressants, including amitriptyline HCl, have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time, particularly in high doses. Myocardial infarction and stroke have been reported with tricyclic antidepressant drugs. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. TRIAVIL may enhance the response to alcohol and the effects of barbiturates and other CNS depressants. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Not recommended in children or during pregnancy.

PRECAUTIONS: Suicide is a possibility in depressed patients and may remain until significant remission occurs. Such patients should not have access to large quantities of this drug.

Perphenazine: Should not be used indiscriminately. Use with caution in patients who have previously exhibited severe adverse reactions to other phenothiazines. Likelihood of some untoward actions is greater with high doses. Closely supervise with any dosage. The antiemetic effect of perphenazine may obscure signs of toxicity due to overdosage of other drugs or make more difficult the diagnosis of disorders such as brain tumor or intestinal obstruction. A significant, not otherwise explained, rise in body temperature may suggest individual intolerance to perphenazine, in which case discontinue.

If hypotension develops, epinephrine should not be employed, as its action is blocked and partially reversed by perphenazine. Phenothiazines may potentiate the action of central nervous system depressants (opiates, analgesics, antihistamines, barbiturates, alcohol) and atropine. In concurrent therapy with any of these, TRIAVIL should be given in reduced dosage. May also potentiate the action of heat and phosphorous insecticides. Neuroleptic drugs elevate prolactin levels; the elevation persists during chronic administration. Tissue culture experiments indicate that approximately one-third of human breast cancers are prolactin dependent *in vitro*, a factor of potential importance if the prescription of these drugs is contemplated in a patient with a previously detected breast cancer. Although disturbances such as galactorrhea, amenorrhea, gynecomastia, and impotence have been reported, the clinical significance of elevated serum prolactin levels is unknown for most patients. An increase in mammary neoplasms has been found in rodents after chronic administration of neuroleptic drugs. Neither clinical studies nor epidemiologic studies conducted to date, however, have shown an association between chronic administration of these drugs and mammary tumorigenesis; the available evidence is considered too limited to be conclusive at this time.

Amitriptyline: In manic-depressive psychosis, depressed patients may experience a shift toward the manic phase if they are treated with an antidepressant. Patients with paranoid symptomatology may have an exaggeration of such symptoms. The tranquilizing effect of TRIAVIL seems to reduce the likelihood of this effect. When amitriptyline HCl is given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs.

Caution is advised if patients receive large doses of ethchlorvynol concurrently. Transient delirium has been reported in patients who were treated with 1 g of ethchlorvynol and 75-150 mg of amitriptyline HCl.

Concurrent administration of amitriptyline HCl and electroshock therapy may increase the hazards associated with such therapy. Such treatment should be limited to patients for whom it is essential. Discontinue several days before elective surgery if possible. Elevation and lowering of blood sugar levels have both been reported. Use with caution in patients with impaired liver function.

ADVERSE REACTIONS: Similar to those reported with either constituent alone.

Perphenazine: Extrapyramidal symptoms (opisthotonus, oculogyric crisis, hyperreflexia, dystonia, akathisia, acute dyskinesia, ataxia, parkinsonism) have been reported and can usually be controlled by the concomitant use of effective antiparkinsonian drugs and/or by reduction in dosage, but sometimes persist after discontinuation of the phenothiazine.

Tardive dyskinesia may appear in some patients on long-term therapy or may occur after drug therapy with phenothiazines and related agents has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw. Involuntary movements of the extremities sometimes occur. There is no known treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms. It is advised that all antipsychotic agents be discontinued if the above symptoms appear. If treatment is reinstituted, or dosage of the particular drug increased, or another drug substituted, the syndrome may be masked. Fine vermicular movements of the tongue may be an early sign of the syndrome. The full-blown syndrome may not develop if medication is stopped when lingual vermiculation appears.

Other side effects are skin disorders (photosensitivity, itching, erythema, urticaria, eczema, up to exfoliative dermatitis); other allergic reactions (asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions); peripheral edema; reversed epinephrine effect; hyperglycemia; endocrine disturbances (lactation, galactorrhea, gynecomastia, disturbances of menstrual cycle); altered cerebrospinal fluid proteins; paradoxical excitement; hypertension, hypotension, tachycardia, and ECG abnormalities (quinidine-like effect); reactivation of psychotic processes; catatonic-like states; autonomic reactions, such as dry mouth or salivation, headache, anorexia, nausea, vomiting, constipation, obstipation, urinary frequency or incontinence, blurred vision, nasal congestion, and a change in pulse rate; other adverse reactions reported with various phenothiazine compounds, but not with perphenazine, include grand mal convulsions, cerebral edema, polyphagia, pigmentary retinopathy, photophobia, skin pigmentation, and failure of ejaculation.

The phenothiazine compounds have produced blood dyscrasias (pancytopenia, thrombocytopenic purpura, leukopenia, agranulocytosis, eosinophilia); and liver damage (jaundice, biliary stasis).

Pigmentation of the cornea and lens has been reported to occur after long-term administration of some phenothiazines. Although it has not been reported in patients receiving TRIAVIL, the possibility that it might occur should be considered.

Hypnotic effects, lassitude, muscle weakness, and mild insomnia have also been reported.

Amitriptyline: Note: Listing includes a few reactions not reported for this drug, but which have occurred with other pharmacologically similar tricyclic antidepressant drugs and must be considered when amitriptyline is administered. *Cardiovascular:* Hypotension; hypertension; tachycardia; palpitation; myocardial infarction; arrhythmias; heart block; stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth; blurred vision; disturbance of accommodation; increased intraocular pressure; constipation; paralytic ileus; urinary retention; dilatation of urinary tract. *Allergic:* Skin rash; urticaria; photosensitization; edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis; leukopenia; eosinophilia; purpura; thrombocytopenia. *Gastrointestinal:* Nausea; epigastric distress; vomiting; anorexia; stomatitis; peculiar taste; diarrhea; parotid swelling; black tongue. Rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido; elevated or lowered blood sugar levels. *Other:* Dizziness, weakness; fatigue; headache; weight gain or loss; increased perspiration; urinary frequency; mydriasis; drowsiness; alopecia. *Withdrawal Symptoms:* Abrupt cessation after prolonged administration may produce nausea, headache, and malaise. These are not indicative of addiction.

OVERDOSAGE: All patients suspected of having taken an overdosage should be admitted to a hospital as soon as possible. Treatment is symptomatic and supportive. However, the intravenous administration of 1–3 mg of physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage of physostigmine should be repeated as required particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine. On this basis, in severe overdosage with perphenazine-amitriptyline combinations, symptomatic treatment of central anticholinergic effects with physostigmine salicylate should be considered.

1180 (DC6613217)

For more detailed information, consult your MSD Representative or see full Prescribing Information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486.

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MEDICAL DIRECTOR'S PAGE

Bruce H. Medd, M.D., Director of Professional Services



THE FAMILY PHYSICIAN AND THE PROBLEM DRINKER

Recognizing the alcoholic patient

It is estimated that one out of every 20 patients seen by the family physician is suffering from the adverse physical and emotional effects of alcohol abuse.[1] However, data suggest that only 1 in 10 alcoholics is diagnosed and treated as such.[2] It is believed that the diagnosis is often missed because most patients rarely complain about or wish to discuss their alcoholism. Instead, they present complaints relating to excessive anxiety and tension, gastrointestinal disturbances, sleep problems and other general physical or psychological symptoms.[1]

The increased emphasis on the disease concept of alcoholism has encouraged the physician to improve diagnostic and treatment skills in this area. This can best be initiated with the recognition by all professionals that alcohol is a widely abused *psychoactive drug* of the sedative/depressant class and that it is highly *addictive,* exhibiting all four parameters of drug dependence as defined by the World Health Organization: *psychic* dependence, *physical* dependence, development of *tolerance* and characteristic *abstinence* (withdrawal) *syndrome*.[2]

As alcoholism and alcohol-related disorders tend to follow the same pattern as other diseases—including onset, acute episodes, remission, relapse, deterioration and even death—the sooner diagnosis is made, the better the prognosis. Prompt identification is also important as alcoholics generally tend to misuse all medication in the same way that they abuse alcohol, often increasing dosages and refilling prescriptions given for the presenting complaints without their physician's knowledge.[3] It should also be noted that with continued drinking an enzyme system—the microsomal ethanol oxidizing system (MEOS)—is induced, which is associated with a nonspecific enhancement of a wide variety of microsomal enzymes. This results in an altered metabolism of many commonly prescribed medicines, *i.e.,* half-life may be markedly shortened in the absence of alcohol, whereas it may be prolonged when the same medication is taken in combination with alcohol.[4] Therefore, when use of any other psychoactive medication, *i.e.,* sedatives, hypnotics and minor tranquilizers, is deemed necessary in these dependency-prone individuals, close supervision should be maintained.

No one is in a better position to detect the patient with alcohol problems than the family physician, and there are many physical and psychological manifestations of alcoholism that can be detected long before the appearance of such classic signs as red face, bloodshot eyes, shaky hands, enlarged liver or frank malnutrition.[1] The accompanying table lists some of the more common *early* indications of alcohol abuse as presented by several authorities in the field.[1,2,5]

GUIDELINES FOR EARLY DETECTION OF ALCOHOLISM

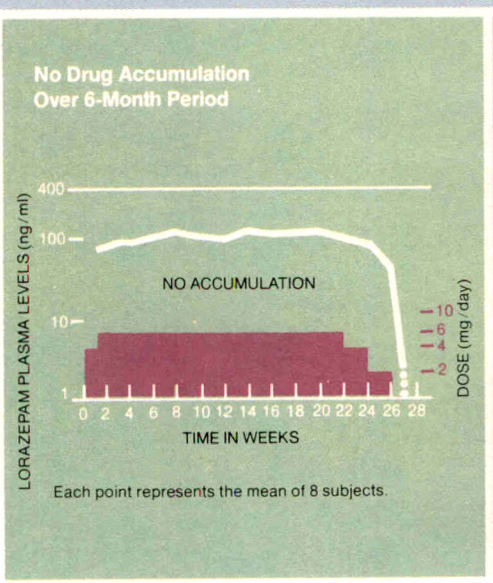
Physical clues	
Gastrointestinal	vomiting (often before breakfast); nausea; dysphagia; diarrhea; vague abdominal complaints
Neurologic	blackouts; insomnia, headaches
Cardiovascular	palpitations; tachycardia
Traumatic	frequent accidents; falls or injuries of vague origin; cigarette burns on hands, chest
Psychological clues	
Behavioral	frequent financial difficulties; excessive absenteeism and poor performance at work; marital discord or multiple divorces; immoderate use of coffee, tea, tobacco, drugs
Emotional	anxiety; panic attacks; hallucinations; depression, suicidal ideation
Laboratory findings	hyperlipidemia; abnormal liver and/or kidney function; anemia; blood alcohol of 0.15% (almost pathognomonic for alcoholism)[1]

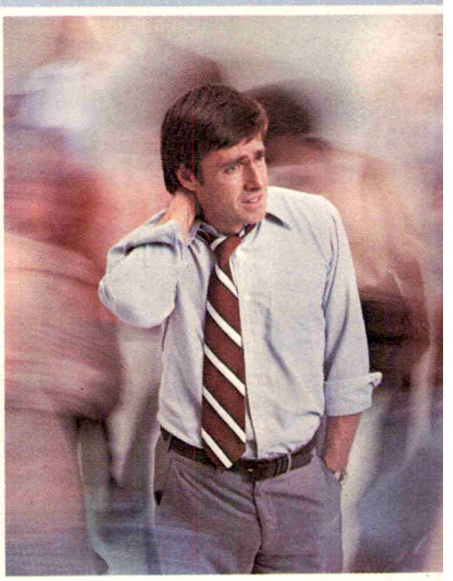
If you would like to receive a copy of a screening chart offering a wide range of clues to the early detection of alcoholism, ask your Roche representative or write to me: Bruce H. Medd, M.D., Director of Professional Services, Roche Laboratories, Division of Hoffmann-La Roche Inc., Nutley, New Jersey 07110.

References: 1. Ewing JA: *Am Fam Physician 18*:107-114, Nov 1978. **2.** Whitfield CL, Williams K: *The Patient with Alcoholism and Other Drug Problems, Medical Aspects for Physicians and Other Helpers,* 3rd ed, Medical College of Wisconsin Medical Education Program, 1977, pp. 10, 13, 26, 29, 30. **3.** Fox R: *Aspects of Alcoholism,* vol. 1. Philadelphia, J.B. Lippincott Company, 1966, p. 13. **4.** Graham DY: *Hosp Med 14*:71-88, Jan 1978. **5.** Knott DH: *Hosp Med 15*:50-65, Jan 1979.

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How do you choose a minor tranquilizer?





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Pharmacokinetics

Ativan (lorazepam) is eliminated more rapidly than other benzodiazepines; generates no clinically active metabolites. The half-life of free lorazepam is about 12 hours; steady-state blood levels are attained in 2-3 days. (Comparable data for diazepam: 20-50 hours and at least 7-10 days.) Ativan shows no evidence of accumulation, even when given in high doses for as long as 6 months. (The pharmacokinetic profile of a drug can define such characteristics as absorption, distribution, metabolism and elimination but cannot, at present, be directly related to its therapeutic effectiveness.)

Anxiety neuroses

In many nationwide studies involving thousands of patients with anxiety neuroses, Ativan (lorazepam) consistently provided significant relief of anxiety, tension, agitation and irritability as measured by standard Hamilton, Global (physician rated) and 35-Item (patient self-rated) scales.

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They're all about the same, aren't they? Until recently, that seemed to sum up the prevalent attitude about the benzodiazepines. After all, they all work similarly and all are effective anxiolytics. With seven of these compounds now available, however, differences have begun to emerge. Foremost among them: different metabolic pathways, which distinguish the relatively short-acting Ativan (lorazepam) from long-acting diazepam, clorazepate and chlordiazepoxide. At the same time, your choice can be made with confidence in the clinical record of Ativan, which confirms its value in anxiety neuroses and in significant anxiety associated with functional or organic disorders, as well as in older patients.

Consider the record of Ativan:



Anxiety in cardiovascular disorders

Ativan (lorazepam) has been specifically evaluated and found effective in seven common protocol, double-blind studies involving 423 patients (211 on Ativan) whose anxiety was related to organic and functional cardiovascular disorders. The cardiovascular component has not, of course, been shown to be significantly benefited by such therapy.



Anxiety in gastrointestinal disorders

So far, nine common protocol, double-blind studies of Ativan have focused on anxious patients with functional or organic gastrointestinal complaints (457 patients, 234 on Ativan). Ativan was clearly effective in reducing the anxiety of these patients. The gastrointestinal component has not, of course, been shown to be significantly benefited by such therapy.



Anxiety in the aging patient

Because its simple metabolism is not readily impaired with advancing age, and accumulation is not likely to present a problem, Ativan is a good choice for older patients. Those who have trouble swallowing solid medication appreciate the small Ativan tablet, which is tasteless and disintegrates within seconds in water or fruit juice.

Ativan® (lorazepam) for Anxiety

See important information on following page.

Brief Summary of Prescribing Information

Indications and Usage: Symptomatic relief of anxiety, tension, agitation, irritability and insomnia associated with anxiety neuroses and transient situational disturbances; anxiety associated with depressive symptoms and as a treatment of symptoms of anxiety if such symptoms are a significant feature of functional or organic disorders, particularly gastrointestinal or cardiovascular.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients on lorazepam not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation.

Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions.

Observe usual precautions with impaired renal or hepatic function.

Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component.

Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day. No effect dose was 1.25mg/kg/day (approximately 6 times the maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatric patients requires caution and frequent monitoring for symptoms of upper G.I. disease.

Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug.

In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Overdosage: In management of overdosage with any drug, bear in mind that multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring of vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

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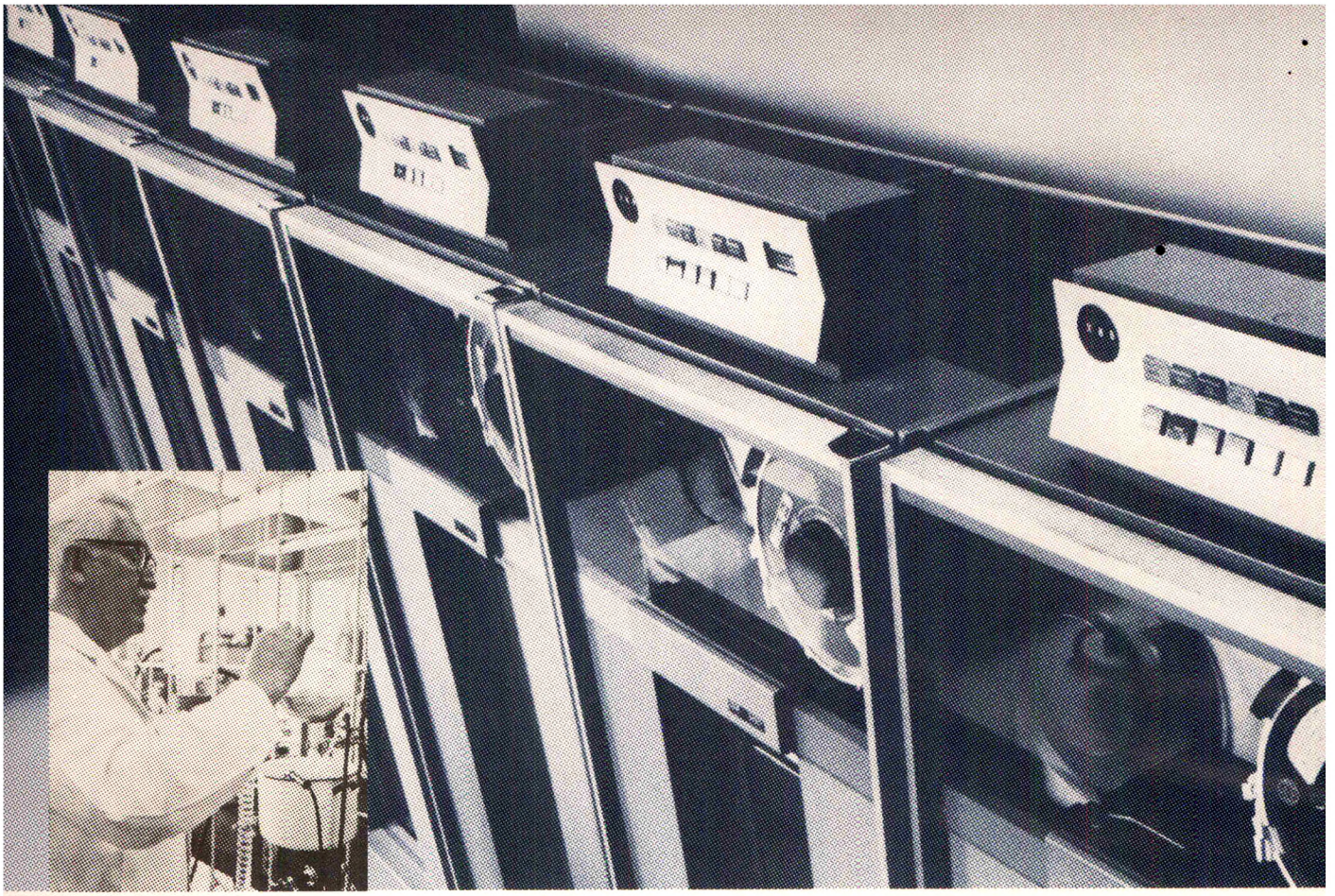
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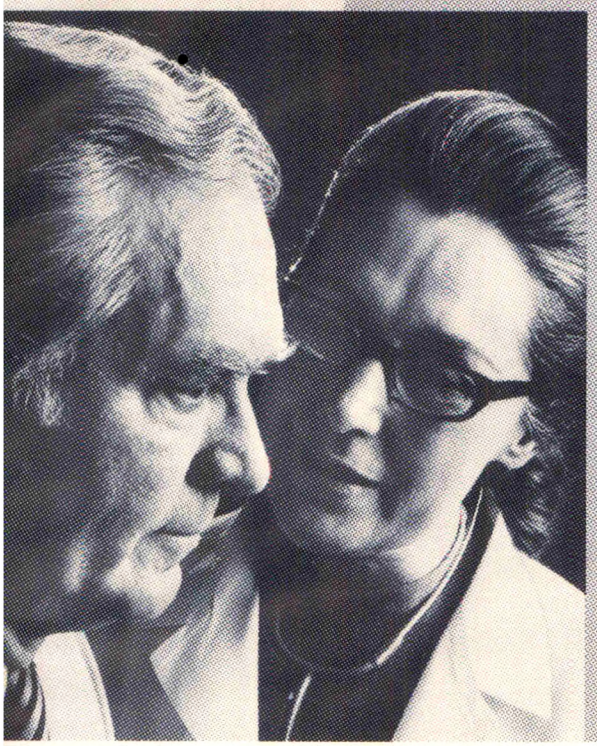
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THE CHRONIC MENTAL PATIENT

Report of a Conference held in January 1978

Edited by

John A. Talbott, M.D.

with contributions by James T. Barter, M.D., Paul R. Friedman, J.D., Raymond Glasscote, M.A., Trevor D. Glenn, M.D., Norman V. Lourie, M.S.W., W. Walter Menninger, M.D., Arthur T. Meyerson, M.D., Kenneth Minkoff, M.D., Samuel Muszynski, M.S.W., Lucy D. Ozarin, M.D., Gordon L. Paul, Ph.D., Ronald Peterson, Steven S. Sharfstein, M.D., Judith Clark Turner, Jane Bloom Yohalem, J.D.

This 277-page Report of the Conference, sponsored by APA and President Carter's Commission on Mental Health, identifies the chronic mental patient population, spells out where they are and what their needs and rights are. It specifies what programs work and what programs do not work in meeting the needs of these patients. It elaborates on the obstacles to implementing effective programs and the economic issues involved. It delineates the pros and cons of case management and specifies responsibility for coordinating, implementing, and monitoring services to chronic mental patients.

Finally, it proposes a *Call to Action* which opens with this statement: "There is no more urgent concern than the needs of the chronic mentally ill who suffer from severe, persistent, or recurrent mental illnesses with residual social and vocational disabilities. As a result of the deinstitutionalization programs of the past decade and the continuing growth of high risk populations that generate chronically ill, the problems associated with the care of these patients constitute a national crisis."

Since the Conference, the APA Assembly and the Board of Trustees have both approved the "Call to Action" which calls upon the APA to take the lead in undertaking programs to elevate the prestige and value of work with chronic mentally ill patients.

It follows that all APA members should be thoroughly versed in the current problems of this chronic patient population. This is best accomplished by reading this Conference Report now available from APA Publications Services at $11.00 a copy.

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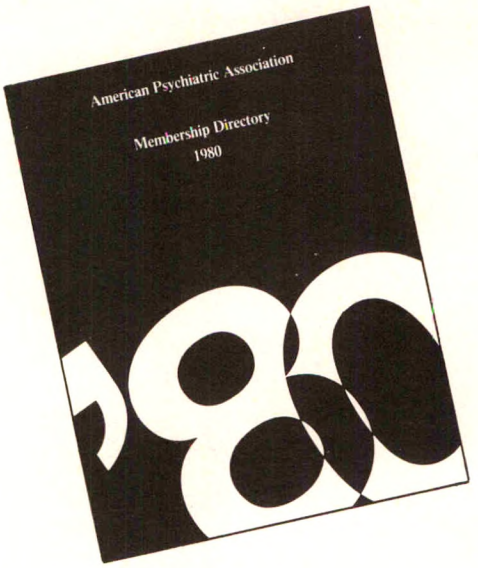
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Contraindications: Severe central nervous system depression, comatose states from any cause, hypertensive or hypotensive heart disease of extreme degree.

Warnings: Administer cautiously to patients who have previously exhibited a hypersensitivity reaction (e.g., blood dyscrasias, jaundice) to phenothiazines. Phenothiazines are capable of potentiating central nervous system depressants (e.g., anesthetics, opiates, alcohol, etc.) as well as atropine and phosphorus insecticides; carefully consider benefit versus risk in less severe disorders. During pregnancy, administer only when the potential benefits exceed the possible risks to mother and fetus.

Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy, observed primarily in patients receiving larger than recommended doses, is characterized by diminution of visual acuity, brownish coloring of vision, and impairment of night vision; the possibility of its occurrence may be reduced by remaining within recommended dosage limits. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving), and increase dosage gradually. Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension since phenothiazines may induce a reversed epinephrine effect on occasion. Daily doses in excess of 300 mg should be used only in severe neuropsychiatric conditions.

Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; rarely, nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—ECG changes (see *Cardiovascular Effects* below). *Other*—Rare cases described as parotid swelling.

It should be noted that efficacy, indications and untoward effects have varied with the different phenothiazines. It has been reported that old age lowers the tolerance for phenothiazines; the most common neurologic side effects are parkinsonism and akathisia, and the risk of agranulocytosis and leukopenia increases. The following reactions have occurred with phenothiazines and should be considered whenever one of these drugs is used. *Autonomic Reactions*—Miosis, obstipation, anorexia, paralytic ileus. *Cutaneous Reactions*—Erythema, exfoliative dermatitis, contact dermatitis. *Blood Dyscrasias*—Agranulocytosis, leukopenia, eosinophilia, thrombocytopenia, anemia, aplastic anemia, pancytopenia. *Allergic Reactions*—Fever, laryngeal edema, angioneurotic edema, asthma. *Hepatotoxicity*—Jaundice, biliary stasis. *Cardiovascular Effects*—Changes in terminal portion of electrocardiogram, including prolongation of Q-T interval, lowering and inversion of T-wave, and appearance of a wave tentatively identified as a bifid T or a U wave have been observed with phenothiazines, including Mellaril (thioridazine); these appear to be reversible and due to altered repolarization, not myocardial damage. While there is no evidence of a causal relationship between these changes and significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients showing characteristic electrocardiographic changes while taking the drug. While proposed, periodic electrocardiograms are not regarded as predictive. Hypotension, rarely resulting in cardiac arrest. *Extrapyramidal Symptoms*—Akathisia, agitation, motor restlessness, dystonic reactions, trismus, torticollis, opisthotonus, oculogyric crises, tremor, muscular rigidity, and akinesia. *Persistent Tardive Dyskinesia*—Persistent and sometimes irreversible tardive dyskinesia, characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and sometimes of extremities may occur on long-term therapy or after discontinuation of therapy, the risk being greater in elderly patients on high-dose therapy, especially females; if symptoms appear, discontinue all antipsychotic agents. Syndrome may be masked if treatment is reinstituted, dosage is increased, or antipsychotic agent is switched. Fine vermicular movements of tongue may be an early sign, and syndrome may not develop if medication is stopped at that time. *Endocrine Disturbances*—Menstrual irregularities, altered libido, gynecomastia, lactation, weight gain, edema, false positive pregnancy tests. *Urinary Disturbances*—Retention, incontinence. *Others*—Hyperpyrexia; behavioral effects suggestive of a paradoxical reaction, including excitement, bizarre dreams, aggravation of psychoses, and toxic confusional states; following long-term treatment, a peculiar skin-eye syndrome marked by progressive pigmentation of skin or conjunctiva and/or accompanied by discoloration of exposed sclera and cornea; stellate or irregular opacities of anterior lens and cornea; systemic lupus erythematosus-like syndrome.

Dosage: Dosage must be individualized according to the degree of mental and emotional disturbance, and the smallest effective dosage should be determined for each patient.

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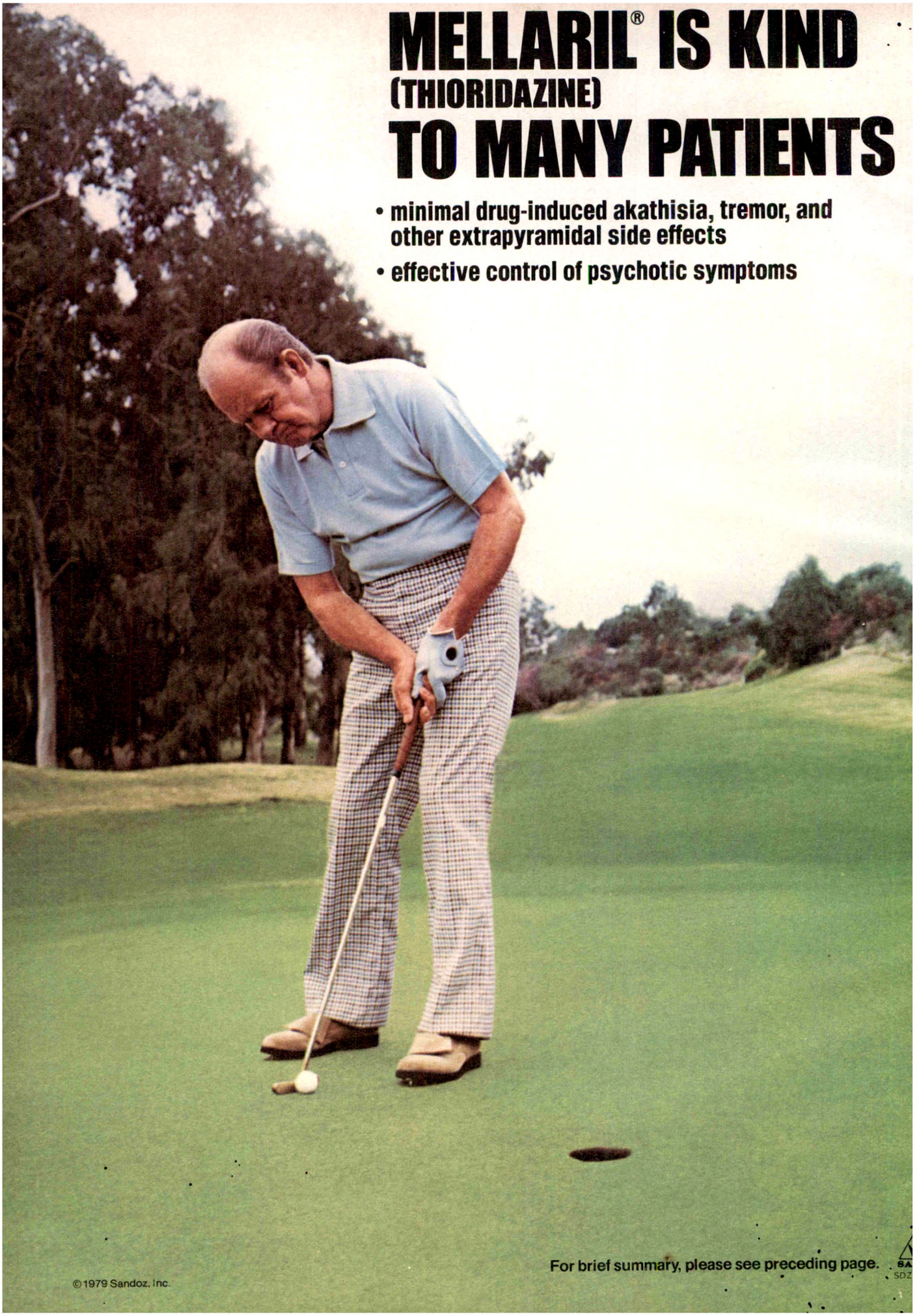
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MELLARIL® IS KIND (THIORIDAZINE) TO MANY PATIENTS

- minimal drug-induced akathisia, tremor, and other extrapyramidal side effects
- effective control of psychotic symptoms

For brief summary, please see preceding page.

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THE AMERICAN JOURNAL OF PSYCHIATRY

Psychophysiologic Factors in Sudden Cardiac Death

BY BERNARD LOWN, M.D., REGIS A. DESILVA, M.D., PETER REICH, M.D., AND BENJAMIN J. MURAWSKI, PH.D.

Sudden cardiac death due to ventricular fibrillation is the leading cause of fatality in the industrially developed world. A considerable body of evidence indicates that the higher nervous system modifies electrical activity of the heart and may trigger sudden death. The evidence for increased risk for ventricular fibrillation due to psychophysiologic factors is supported predominantly by animal studies, but increasing evidence is forthcoming from human studies. The involvement of psychiatrists, psychologists, and cardiologists in a multidisciplinary approach to managing patients at risk for sudden death from ventricular fibrillation is yielding significant insights and prolonging their lives.

Recognition of an association between sudden death and profound emotion dates to the very dawn of recorded history. Comprehension of such a relation is embedded in the matrix of common speech and documented in the most ancient of scripts, scrolls, and papyri. Yet until quite recently medical scientific inquiry stood aloof from the entire subject of sudden death, let alone examine the possible role of psychologic factors in its pathophysiology. This neglect was not by virtue of incomprehension of the magnitude of the problem. Rather it stemmed from the more general malaise to be found in any scientific field when methodologic tools have not yet been developed to probe adequately a given field of inquiry. Sudden death had been rationalized as the irreversible culmination of advanced coronary atherosclerosis, and since it occurred unpredictably in the community, it was seemingly outside the sphere of the physician's responsibility. However, the advent of direct-current defibrillation, cardioversion, coronary care units, heart rhythm monitoring devices, and, especially, cardiopulmonary resuscitation emphasized the reversible nature of sudden death due to ventricular fibrillation.

The initial questions posed regarding identification of the patient at increased risk for sudden death suggested that neural factors were implicated. In 1970, Lown and Ruberman (1) postulated that certain types of ventricular premature beats are harbingers of fatality. These extrasystoles are sporadic and often emerge without apparent physiologic basis. The cardiologist necessarily considers a hemodynamic explanation, wherein the ectopic beat is deemed to be an expression of enhanced myocardial ischemia. However, young and healthy subjects without demonstrable heart disease are by no means spared such rhythmic irregularities (2). A hemodynamic explanation, furthermore, does not account for the fact that there is a greater prevalence and severity of arrhythmia during a 24-hour electrocardiographic monitoring period, when the patient is largely passive, than during maximal exercise stress testing (3). Exercise testing induces peak heart rates and rises in blood pressure far in excess of the levels observed during passive monitoring. What then are the provocative factors for arrhythmia that are even more potent than a vigorous hemodynamic stress? It has been our working hypothesis that higher nervous activity triggered by psychologic factors can significantly affect cardiac electrophysiologic properties and may result both in ventricular premature beats and in lowering the threshold for ventricular fibrilla-

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tion, the underlying basis for sudden cardiac death (4, 5).

Whenever patients dying suddenly have been electrocardiographically monitored at the time of death, the mechanism almost invariably has been ventricular fibrillation. This rhythm disorder is characterized by a chaotic disorganization of the heart beat, veritably an electrical storm of tumultuous and ineffectual colliding and dispersing wavelets of depolarization that annul synchronized activation of the contractile process. The result is abrupt cessation of cardiac pumping action, with irreversible brain damage ensuing within several minutes. It has long been entertained that this catastrophic arrhythmia leading to arrest of cardiac activity is precipitated by coronary thrombosis, myocardial infarction, or both. Yet in a majority of those who die suddenly fresh morphologic lesions, either in vessels or in heart muscle, are remarkable for their absence. Furthermore, patients who have been resuscitated from sudden cardiac death generally do not immediately reexperience ventricular fibrillation, although recurrence may occur at a later time. Thus, ventricular fibrillation may be deemed to be due to a sudden but momentary disruption of the electrical function of the heart. Sudden cardiac death may therefore be viewed as a reversible electrical accident. Lown and coworkers (6, 7) have postulated that transient risk factors momentarily destabilize the heart already afflicted with ischemic disease and thereby predispose it to self-regenerating repetitive electrical activity resulting in ventricular fibrillation. Among the major sources of transient risk factors is the central nervous system.

The validity of this hypothesis is of profound contemporary medical significance. Sudden cardiac death is the leading cause of fatality in the industrially developed world. It claims more lives than cancer, and about 1,200 individuals are stricken daily in the United States alone. The victims are predominantly male and frequently at the prime of life. Studies in animals as well as clinical observations sanction the view that neural and psychologic inputs to the heart may indeed trigger ventricular fibrillation. These findings deserve careful examination.

ANIMAL MODELS

In studying the role of psychologic stresses on predisposition of the heart to ventricular fibrillation, the arrhythmia of sudden death, a number of methodologic problems had to be overcome. Until recently no experimental data were available relating to the effect of psychologic variables on cardiac susceptibility to ventricular fibrillation. This was largely due to the absence of suitable methods for repeatedly determining cardiac vulnerability in the intact unanesthetized animal. Such assessment required the use of painful stimuli, induction of ventricular fibrillation, and use of traumatic resuscitation procedures that preclude meaningful investigation of psychologic variables. It was necessary to devise an appropriate electrical end-point that could serve as an index of vulnerability.

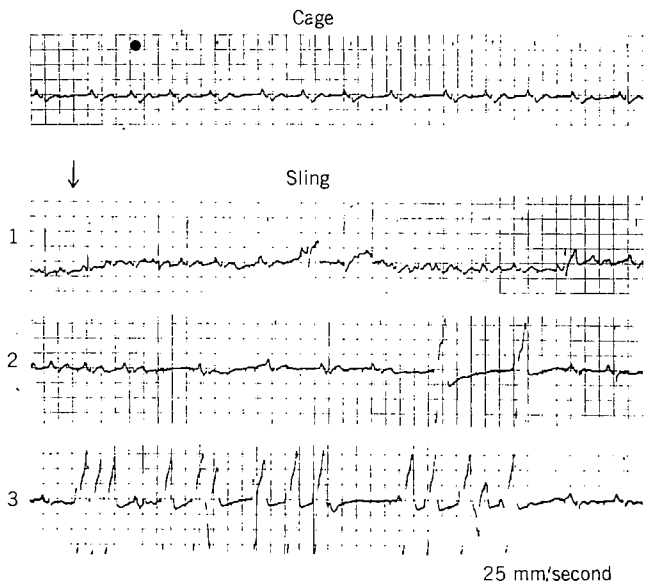
The physiologist measures cardiac vulnerability by applying increasing intensities of electrical pulses during the 20-millisecond interval of the cardiac cycle (referred to as the ventricular vulnerable period), coinciding with the apex of the T-wave of the surface electrocardiogram. The minimum current discharged during the vulnerable period that induces ventricular fibrillation is designated the ventricular fibrillation threshold. This index serves as a measure of electrical instability of the heart and its predisposition to malignant arrhythmias. A high threshold indicates a low vulnerability to fibrillation and vice versa. To obviate the psychologic and physical trauma attending precipitation of ventricular fibrillation and subsequent defibrillation, we have developed another marker for electrical stability of the myocardium, known as the repetitive extrasystole threshold (8–11). This method consists of delivering a graded electrical impulse to the heart early in diastole and coincident with the vulnerable period. Normally, one impulse evokes but a single ventricular response. As the current strength is progressively increased and approaches the ventricular fibrillation threshold, one stimulus will evoke two or more successive extrasystoles. The minimum current required to evoke two ventricular responses is designated the repetitive extrasystole threshold (8). This threshold is consistently 65% of the lowest current required to evoke ventricular fibrillation. The repetitive extrasystole threshold reliably tracks electrical current requirements for ventricular fibrillation, without awareness on the part of the animal (11).

Psychologic Stress and Ventricular Vulnerability

Using the repetitive extrasystole method, we have shown in dogs that psychologic stress profoundly affects vulnerability to ventricular arrhythmias and fibrillation (9–14). One group of dogs was left undisturbed in a cage, and in another cage dogs were immobilized in a Pavlovian sling and subjected to mild electric shocks. No shocks were administered after the first 3 days. The dogs that had been conditioned in these two environments were studied during recovery from acute myocardial infarction. During the period of recovery, at a time when heart rhythm was normal, return of an animal to the stressful sling environment provoked ventricular tachycardia (see figure 1). Acute coronary artery occlusion in the stressful environment resulted in an incidence of ventricular fibrillation that was three times higher than that in the nonstressful environment (14). Placing the animal in the stressful environment also resulted in elevations in blood levels of norepinephrine and total catecholamines.

Using the same cage and sling model, we also in-

FIGURE 1
Ventricular Arrhythmia Provoked by Psychologic Stress in a Dog with Previous Coronary Artery Occlusion[a]



[a]Several weeks earlier the dog had been placed in a Pavlovian sling and subjected to mild electrical shocks. The electrocardiogram indicates no arrhythmia when the dog was left undisturbed in the cage and, 1) immediately after transfer to the sling (arrow), acceleration in heart rate and unsteadiness of baseline with a single fusion beat, 2) ventricular premature beats two minutes later, and 3) paroxysms of ventricular tachycardia.

vestigated dogs with normal hearts. When a dog was placed in a sling where it had received an electrical shock the previous day, its threshold for repetitive extrasystoles was 40%–50% lower than when it was in the tranquil environment of the cage. This difference in response indicates increased vulnerability to fibrillation in the presence of increased stress (9–12). These studies also documented that the onset of increased vulnerability was extremely rapid, occurring within a few minutes of exposure to stress. On the other hand, restoration of control conditions after withdrawal from stress required a much longer time (12). This is consistent with clinical observations relating to latency of the cardiac response and recovery when an individual is subjected to psychologic stress.

Similar findings have been reported by Skinner and associates (15), who studied the effects of familiar and unfamiliar environments on the occurrence of ventricular fibrillation caused by acute coronary occlusion in pigs. In the unfamiliar setting fibrillation occurred a few minutes after the induction of coronary occlusion. When the same experiment was performed in an environment to which the pigs had previously adapted, onset of fibrillation was greatly delayed and even entirely prevented in some animals. We have found also that a shock avoidance paradigm in dogs markedly lowered the threshold for ventricular fibrillation (10). Thus a variety of psychologic stresses induce profound changes in cardiac electrophysiologic properties that favor the development of ventricular fibrillation.

Neural Mechanisms

It is pertinent to consider the neural mechanisms by which psychologic stresses lower the ventricular vulnerable period threshold and predispose to ventricular fibrillation. The mammalian ventricle is richly innervated by the autonomic nervous system. Both the sympathetic and the parasympathetic divisions are represented. A large number of studies spanning a hundred years indicate that cardiac electrical activity is influenced by neural inputs (5). Stimulation of peripheral nerve structures such as the cardiac sympathetic nerves and the stellate ganglia increases susceptibility to ventricular fibrillation. Earlier in this century it was shown that injection directly into the brain of drugs or chemicals such as nicotine, barium chloride, caffeine, epinephrine, acetyl choline, and strophanthidin produced ventricular arrhythmias including fibrillation. Subsequent studies using more precise methods with the aid of stereotaxically placed electrodes demonstrated the occurrence of ventricular arrhythmias during stimulation of the frontal, orbital, motor and premotor cortical areas, the anterior part of the temporal lobe, the insula, and the cingulate gyrus. Korteweg and colleagues (16) found that arrhythmias were more readily provoked from subcortical areas such as the posterior hypothalamus and the quadrigeminal bodies.

Myocardial ischemia is the most potent predisposing factor for the development of ventricular fibrillation. Satinsky and coworkers (17) studied the effect of stimulation of the cardiac accelerator and vasomotor centers in the posterior hypothalamus during acute myocardial ischemia in anesthetized dogs. Stimulating electrodes were stereotaxically placed in the hypothalamus. In the absence of hypothalamic stimulation, during ligation of the left anterior descending coronary artery only 2 of the 32 dogs (6.3%) developed ventricular fibrillation. This low incidence of ventricular fibrillation was increased ten-fold to 62.5% when the hypothalamus was stimulated immediately after acute ischemia was induced by ligation of the same vessel. This study indicated that the profibrillatory effect of myocardial ischemia can be potentiated by neural traffic originating in hypothalamic nuclei. The pathway through which cortical and subcortical inputs affect the heart is likely to be the sympathetic nervous system since susceptibility to ventricular arrhythmias is ablated by cardiac sympathectomy or sympathetic blockade with beta-adrenergic blocking drugs (4). Also, stimulation of stellate ganglia replicated the results obtained with midbrain stimulation even in experiments wherein blood pressure and heart rate effects of enhanced sympathetic activity were precluded (18, 19).

Are sympathetic-adrenergic mechanisms also implicated in the susceptibility to ventricular fibrillation resulting from psychologic stress? Animals trained to avoid programmed electric shock showed a 50% re-

duction in repetitive extrasystole threshold persisting throughout the three days in which they were exposed to this biobehavioral stress (10). However, when the animals received intravenously the beta-1 cardioselective drug Tolamolol in doses sufficient to annul the heart rate response to isoproterenol, the change in cardiac vulnerability for repetitive response was prevented. Thus, beta-adrenergic blockade, which inhibits the effects of sympathetic autonomic discharge, obviated the changes in cardiac vulnerability induced by instrumental aversive conditioning.

There has been less clarity about the role of the parasympathetic nervous system in the genesis of ventricular arrhythmias. The investigations of Richter (20) have exerted profound influence in directing psychologic investigators to the vagus as a cause of sudden death. Rats made to swim in water tanks died from bradycardia and asystole rather than from ventricular fibrillation or from drowning, and the mechanism was believed to be enhanced vagus nerve activity. While the simple faint in man is caused by this means, sudden cardiac death is largely the result of ventricular fibrillation. In fact, experimental evidence (21–29) argues a converse thesis, namely that the vagus exerts antiarrhythmic action on the ventricular myocardium. The protective effect of the vagus is demonstrable only in the presence of enhanced adrenergic drive, whether elicited by stellate ganglion stimulation (28) or catecholamine infusion (29). That the action of the vagus depends on increased sympathetic tone is further indicated by the observation that when beta-adrenergic blockade was induced, changes in ventricular excitability (30) and vulnerability (28) were completely precluded. The antiarrhythmic vagal effect is due to its muscarinic properties and can be annulled by atropine (29).

Thus, the vagus nerve exerts but an indirect action on cardiac vulnerability by antagonizing augmented sympathetic tone. The basis for the cholinergic antagonism is due to muscarinic stimulation that inhibits the release of norepinephrine from sympathetic nerve endings and attenuates the response to catecholamines at receptor sites. Vagal afferent activity also exerts a protective influence on vulnerability by tonic inhibition of sympathetic neural discharge. These mechanisms also operate in the conscious state and modulate ventricular electrical stability in response to psychophysiologic stimuli (31).

Neurochemical Aspects

Sympathetic and parasympathetic neural activity continuously modulate the excitable properties of the heart. It may therefore be worthwhile to probe the role of brain neurotransmitters in the generation of autonomic neural traffic that affects cardiac vulnerability to ventricular fibrillation. This approach is especially inviting in view of the seminal investigations of Wurtman and Fernstrom (32, 33), which indicate that neurotransmitter synthesis in the brain can be modified by dietary means.

There is evidence that accumulation of serotonin in the brain may reduce sympathetic neural activity (34, 35). It has been demonstrated that concentration of central serotonin depends on plasma levels of tryptophan, the essential dietary amino acid and the physiologic biochemical precursor of serotonin. When tryptophan alone is administered, it is hydroxylated and then decarboxylated to form serotonin throughout the body (36). Monoamine oxidase then catalyzes a rapid degradation of serotonin. However, serotonin can be concentrated in the brain by following tryptophan administration with the simultaneous monoamine oxidase inhibitor phenelzine and the L-aromatic amino acid decarboxylase inhibitor carbidopa. Phenelzine crosses the blood barrier and favors the accumulation of serotonin wherever it is formed. However, carbidopa is limited to the peripheral circulation and thereby restricts serotonin formation to the brain.

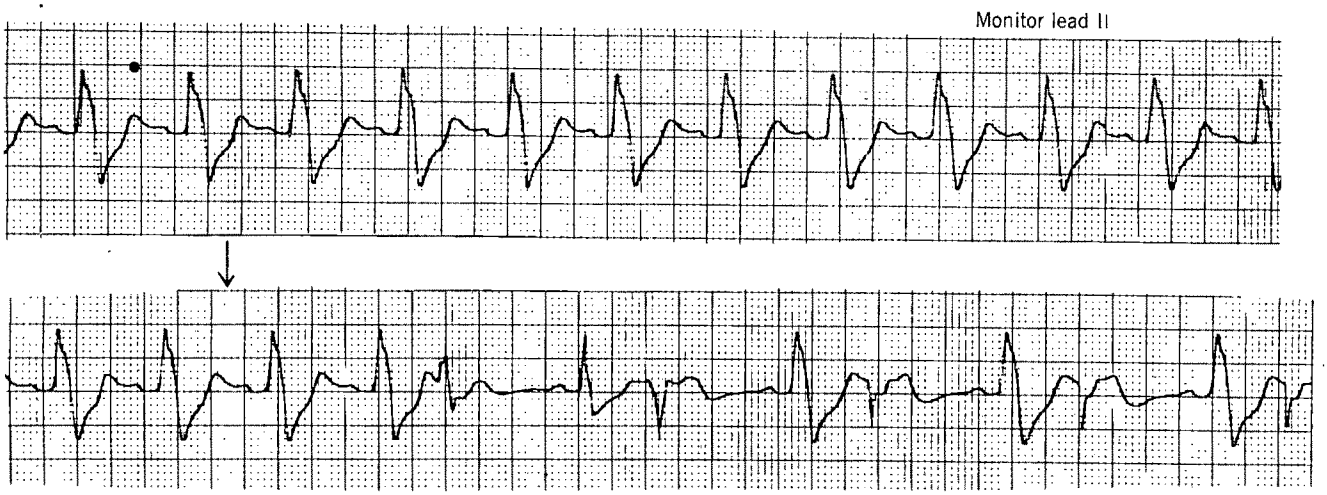
Rabinowitz and Lown (37) used these methods to determine their effect on cardiac susceptibility to repetitive extrasystoles, which is a measure of ventricular vulnerability to fibrillation. When tryptophan, phenelzine, and carbidopa were given separately no change in ventricular threshold for repetitive response was noted. However, a sustained increase of 50% in this threshold resulted from the use of biochemical measures that presumably increase serotonin levels in the central nervous system. When 5-hydroxytryptophan was administered instead of its parent compound tryptophan, thus bypassing the rate-limiting step of hydroxylation, the threshold increased within one hour, compared to 2½ hours after tryptophan.

A further corroboration of the above observations is provided by the recent findings of Blatt and coworkers (38). Systemic administration to dogs of the three central serotonergic agents, including melatonin, induced significant increases in the threshold of the ventricular vulnerable period for repetitive myocardial electrical activity. Such changes were blocked by the specific serotonin antagonist metergoline. These findings have significant clinical implications. Modern neuropharmacology is providing agents that enable precise alterations in brain neurochemistry and thereby permit elicitation of discrete cardiovascular effects. This approach may provide a new method for reducing ventricular vulnerability to fibrillation by neuropharmacologic measures.

CLINICAL STUDIES

While one may design adequate animal models to simulate biologic problems in man, the ultimate validation of putative causal connections requires direct investigation of the human condition. That emotions

FIGURE 2
Ventricular Arrhythmia in a Woman with Previous Ventricular Fibrillation When Conversation Was Shifted to Domestic Conflict (arrow)[a]



[a]The conversation occurred 48 hours after the woman had been resuscitated from ventricular fibrillation. She had been entirely free of ventricular arrhythmia during continuous monitoring in a coronary care unit, but when, during the fourth minute of conversation, the topic was shifted to domestic conflict with her family, ventricular bigeminy with T-wave interruption emerged and persisted until the discussion was shifted to less psychologically aversive content. This arrhythmic response was consistently reproducible.

play a role in sudden death has gained currency by repetition if not by precise documentation. The role of psychologic and emotional factors in the provocation of sudden death is chronicled in the earliest folklore of diverse cultures. Engel (39) has compiled anecdotal reports gleaned from the daily press of 170 sudden deaths that occurred in association with profound emotions such as bereavement, real or symbolic danger, loss of status, and even joy. However, such a conjunction of events, while compelling attention and speculation, is but meager evidence of causality.

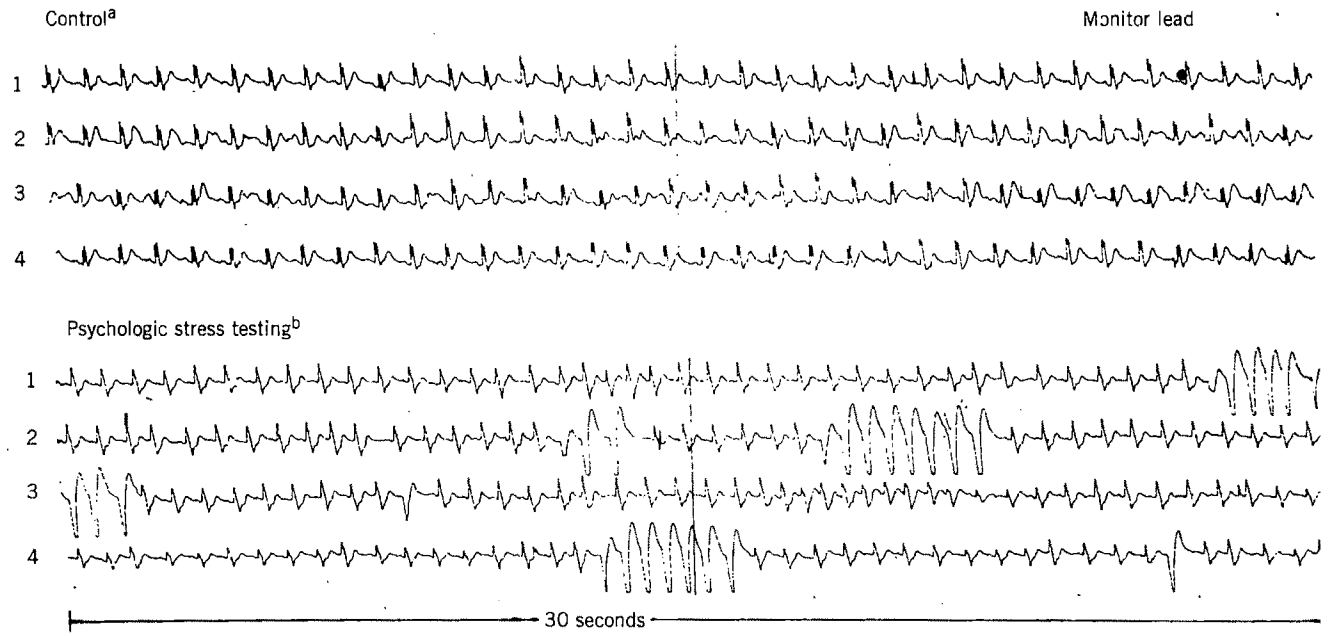
The problem can be approached more readily by considering the antecedent mechanisms that predispose to ventricular fibrillation, the underlying basis for sudden cardiac death. The hypothesis that certain complex forms of ventricular ectopic (or premature) beats may portend sudden fatality among patients with ischemic heart disease (1, 6) has now been confirmed in extensive epidemiologic and clinical investigations (40–45). It is thus inviting to examine interactions between psychologic factors and the occurrence of ventricular arrhythmias that may predispose to sudden death. The study of arrhythmias affords several advantages over investigating other disease states. The deranged heart rhythm is directly exposed by electrocardiographic monitoring, is sporadic and recurrent, and has a precise time of onset and offset. These factors not only simplify appreciation of end-organ changes but provide opportunity for defining possible causal connections between psychologic stresses and occurrence of arrhythmia.

While the cardiac characteristics required for initiating ventricular fibrillation in man can be stated with some precision, definition of the trigger factors emanating from the brain have proved elusive. The clinician frequently intuits a causal connection between a particular psychologic state or emotional stress and the onset of ventricular arrhythmia. It is recognized, though, that the predisposing psychologic condition is subjective, not readily measureable, uniquely individual, and derived from a complex weave of past experiences and diverse processes of psychologic conditioning. The factors triggering arrhythmia may appear to be innocuous stimuli to the clinical observer but in the particular patient gain their currency and strength from symbolic meaning deeply enmeshed in a prior conditioning matrix. Thus in relation to arrhythmias, as in other attempts to study mind-body links, methodologic deficiencies will prevail until scientific advances permit classification of these fundamental psychologic processes. Nevertheless, data are now accumulating which indicate that psychologic factors and biobehavioral stresses may provoke ventricular ectopic activity.

Ventricular Ectopic Activity and Psychologic Stress

A number of early observers have commented on the relationship between emotion and the occurrence of ventricular premature beats. Sigler (46) noted that discussion of emotionally charged events during electrocardiographic monitoring evoked significant ventricular arrhythmias. Similar data were presented by Stevenson and coworkers (47). Taggart (48, 49) in a series of studies showed that stresses induced by car racing, driving in traffic, public speaking, or thermal and emotional stresses provoked ventricular arrhythmias. Public speaking was associated with emergence of ventricular premature beats in 6 of 23 healthy individuals and in 5 of 7 patients with coronary artery disease (49). This relation of psychologic stress and the

FIGURE 3
Ventricular Premature Beats in a 66-Year-Old Man During Psychologic Stress Testing



[a]Heart rate=65 beats/minute; no ventricular premature beats.
[b]Heart rate=92 beats/minute. High grades of ventricular premature beats consisting of couplets and recurrent runs of ventricular tachycardia appeared during the interview relating to his illness. Subsequent monitoring (not shown) revealed insignificant arrhythmia.

occurrence of ventricular arrhythmia is frequently noted in patients continuously monitored in coronary care units (see figure 2).

Attempts to objectify psychologic stress, namely, development of testing procedures that will predictably provoke arrhythmias by exposure to some adverse but ethically permissible biobehavioral experience has so far proved unachievable except in the occasional patient. Since changes in autonomic neural traffic in the experimental animal alter cardiac excitability, we examined such an approach in a series of 19 patients already exhibiting ventricular arrhythmia, 8 of whom had been resuscitated from sudden death. Autonomic neural testing consisted of carotid sinus stimulation, posture changes on a tilt table, Valsalva's maneuver, hyperventilation, breath-holding, and dive-reflex activation. All of these proved ineffective in provoking ventricular ectopic activity (50, 51).

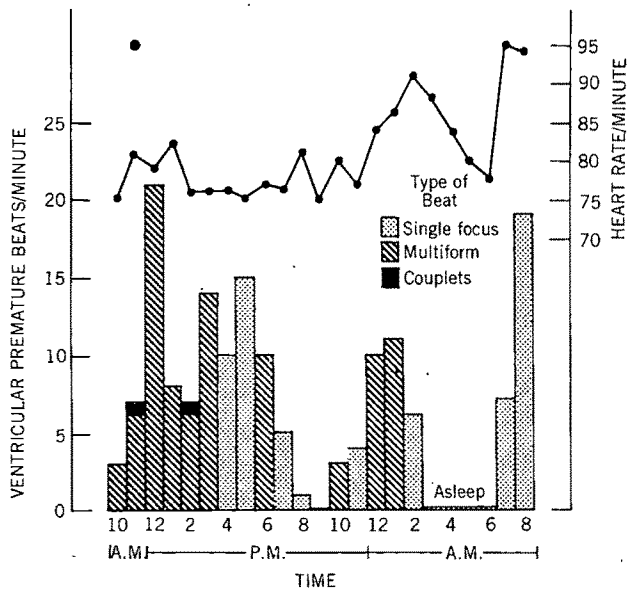
We also administered a structured psychologic stress test to the same group of patients. It consisted of three parts: mental arithmetic for 5 minutes, reading from Stroop color cards for 5 minutes, and a brief psychologically charged discussion for 10 to 15 minutes. The discussion centered around the patient's illness, marriage, financial burdens, work-related problems, and death. The patient was continuously monitored electrocardiographically by means of telemetry throughout the procedure. In 11 of the 19 patients there was a substantial increase in frequency of ventricular premature beats during the study. The mean level of premature beats for the entire group increased from 2.8 per minute during the control period to 6.1 per minute ($p<.05$, Wilcoxon's sign ranked test) during the stress test. In one patient, discussion of his illness provoked tears and life-threatening ventricular tachycardia, which abated with reassurance alone (see figure 3).

We have now extended these studies to 31 patients with ventricular arrhythmias. The frequency of ventricular premature beats during stress testing was compared to that noted during three random hours of a 24-hour monitoring session while the patients were awake. The frequency of ventricular ectopic activity was significantly greater ($p<.05$) during the psychologically stressful interview than during any other of the three control periods. This was all the more remarkable in view of the fact that the stress was mild. Furthermore, each control period itself was probably not free of psychologic tension as these were hospitalized patients. These studies emphasize that "passive" alterations in peripheral autonomic neural tone are not sufficient to precipitate ventricular arrhythmias, but psychologic stress is. While the mechanisms remain to be defined, enhanced sympathetic neural activity is undoubtedly implicated, yet additional pathways must be involved for cerebrocortical-mediated effects on cardiac excitability.

Reduction in Neural Input

While increased sympathetic outflow associated with psychological stress may evoke arrhythmias, it is relevant to examine the effects of reduction in sympa-

FIGURE 4
Ventricular Premature Beats and Heart Rate in a Man with Alcoholic Cardiomyopathy[a]



[a]When the patient finally slept from 2 to 6 a.m., nearly all ventricular arrhythmia disappeared. Although the heart rate decreased during sleep, it was more rapid than during the majority of hours the patient was awake.

thetic tone such as occurs during sleep or with relaxation states. Lown and coworkers (7) demonstrated a striking decrease in ventricular premature beat frequency and grade while patients were asleep in their homes. These investigations were subsequently extended to 30 hospitalized patients who had EEG as well as electrocardiographic monitoring during sleep (see figure 4). During sleep, rapid eye movement (REM) periods showed the highest prevalence of ventricular extrasystoles. The most significant reductions in ventricular premature beats occurred during stages II and III (R.A. DeSilva, Q. Regestein, B. Lown, unpublished observations).

The effect on ventricular arrhythmia of a noncultic form of meditation involving relaxation has also been studied (52, 53). In one such study (52) the results suggested a reduction in ventricular premature beat frequency, while a variable response was noted by another group (53). Our own experience with this form of meditation (R.A. DeSilva and B. Lown, unpublished observations) involved 52 patients. Twenty of them showed more than one ventricular ectopic beat per minute and were selected for analysis. Meditation produced a significant but slight decrease in heart rate (4%) and a decrease in the frequency of ventricular premature beats (37%; $p<.01$; paired t test, one-tailed). No correlation was demonstrable between shifts in heart rate and ventricular premature beat frequency. In one patient having ventricular tachycardia at the time of testing, the arrhythmia terminated during meditation. In 14 of the 20 patients, assays for blood epinephrine, norepinephrine, and dopamine showed no statistically significant alterations. The response of arrhythmia to this meditation is, however, an individual response and in some patients no effect or an actual increase in ventricular ectopic beats may occur.

Sudden Cardiac Death

The fact that sudden death is unexpected and most frequently instantaneous and occurs in the seemingly healthy subject who is in the midst of usual chores confounds the possibility of meaningful study, especially when it is usually a unitary event. Nevertheless, diverse studies are pointing to a possible causal relation between biobehavioral factors and the provocation of sudden fatality. A number of studies suggest that particular lifestyles and the occurrence of traumatic life events may predispose to both myocardial infarction and sudden death (54–61). For example, Wolf and Goodell (54) found in the Roseto community in Pennsylvania that rural Italians, although consuming a diet rich in lipids, had a low death rate from myocardial infarction. The families were stable, closely knit, and relatively secure. With increasing urbanization and disruption of the family unit, the sudden death rates increased and are now approaching that of other adjoining communities. Rahe and coworkers (55–57) have shown that significant life changes have an impact on longevity. Events such as recent bereavement, loss in job status, divorce, and imprisonment were assigned specific life-change unit weights. These data were scored on an internal subjective metric by 1,279 survivors of documented myocardial infarctions and, by their spouses, for 226 sudden cardiac death victims. The life-change units for these individuals were higher during the preceding six months than during the same time interval one year earlier. Higher scores were particularly evident for sudden death victims. Statistical correlations between recent bereavement and sudden death have been shown by Rees and Lutkins (58), especially among males. In the year following bereavement, widowers had a mortality rate of 12.2% as compared to 1.2% in a carefully matched control group. Similar findings have been reported by Parkes and associates (59).

Among patients recovering from myocardial infarction, significantly more patients died in conjunction with ward rounds conducted by the chief surgeon than at other times (60). The stresses engendered by excitement, anxiety, and disappointments in relation to the patient's progress and discharge from hospital were considered as factors provoking sudden death. Transfer from a coronary care unit to a general ward has been associated with emotional disturbances, reinfarction, and serious ventricular arrhythmias (61). Among 7 patients, 5 experienced an increase in urinary catecholamine excretion, and of these 2 developed ventricular tachycardia and ventricular fibrillation. When 7 subsequently treated patients were psychologically

prepared before transfer out of the coronary care unit, no cardiac or emotional complications were noted and only 2 developed increases in catecholamine excretion.

Some cogent insights are being provided in studies relating educational levels to mortality from heart disease. Although using educational level as an index for the magnitude of psychosocial stress poses certain problems (62), Kitagawa and Hauser (63) cite a number of reasons for accenting it as a measure of socioeconomic status as well as psychosocial stress. Educational level, to a large extent, determines the individual's position in society, including income and opportunities for upward social mobility. In contemporary United States society, the lack of education is profoundly handicapping and excludes a person from achieving those personal goals on which our culture places highest value. It furthermore traps the individual in a tracking course of poverty without ready exit. The result is unmitigated frustration and unrelenting psychosocial stress. Comstock (64) showed that among 189 deaths in males aged 45–64 years in Washington County, Md., a low level of education, a history of smoking, and lack of church attendance correlated with cardiac mortality. A greater than expected death rate from ischemic heart disease occurred among men with fewer than 8 years of education compared to a cohort that had achieved a higher educational level. Kitagawa and Hauser (63) in their massive demographic studies found a consistent inverse relationship in adults between cardiovascular mortality and educational attainment. The effect of education was more noteworthy among persons 25 to 64 years of age than among older age groups. As an example, among white men 25–64 years old, mortality ratios for men with hypertension who had fewer than 8 years of schooling was 1.27 as compared to 0.70 for men with one or more years of college, a differential of 55%.

The classical investigations of Kitagawa and Hauser assume special relevance in view of the recently reported finding from the ongoing cardiovascular epidemiologic survey by the Health Insurance Plan of New York (65). These workers found a significant association between education level, the presence of complex forms of ventricular premature beats, and sudden cardiac death. Among 1,739 men who survived myocardial infarction and were monitored electrocardiographically for one hour, men with 8 years of schooling or less exhibited over 3 times the risk of sudden cardiac death if complex ventricular arrhythmias were present. By contrast, better educated men who had the same arrhythmia had a significantly lower mortality rate. The cumulative mortality rates were 33% and 9%, respectively, for the two groups over a 3-year follow-up period. Neither standard risk factors nor a variety of clinical characteristics accounted for the observed difference in mortality.

Direct evidence on the relationship between psychophysiologic stresses and the occurrence of malignant ventricular arrhythmias is provided by several carefully studied case reports. Wellens and associates (66) observed the recurrent onset of ventricular fibrillation in a young girl whenever she was awakened by an alarm clock. The initial episode of fibrillation occurred after she was awakened by a thunderclap. Recently, Lown and coworkers (67) described in detail the case of a 39-year-old man who experienced ventricular fibrillation on two occasions, once while awake and once while asleep. The first episode occurred when he was roughhousing with his sexually mature daughters. When a neighbor rang the doorbell he looked up, said "I'm sorry," and had a cardiac arrest that was demonstrated to be due to ventricular fibrillation. After successful resuscitation he underwent cardiac catheterization and coronary angiography; there was no evidence of structural heart disease. Extensive psychiatric, psychologic, and sleep electroencephalographic with simultaneous electrocardiographic evaluations were carried out. He was defensive and covertly hostile and denied being depressed or angry, yet his lifestyle pattern was one of controlled aggression. Although stating an aversion to violence and repressed sexuality, these were frequently the subject of his dreams. During initial psychiatric interviews he developed increases in ventricular premature beat frequency and grade, although he was outwardly calm and well controlled. A second cardiac arrest occurred during sleep at 4:00 a.m., and a sleep electroencephalogram subsequently showed marked increase in arrhythmia during REM sleep occurring at this very time of night. He was taught to meditate using a noncultic method, and during periods of meditation he could suppress arrhythmia successfully. The management of this patient's case included not only a comprehensive antiarrhythmic drug program but meticulous attention to psychiatric and psychologic factors as well. In the four years since this report, he has remained well and nearly free of any ventricular ectopic activity.

Donlon and associates (68) reported the case of a 38-year-old obsessive-compulsive man with conflicts in relation to aggression, work, and sexuality who was studied six weeks after an inferior wall myocardial infarction. He was interviewed for an hour and heart rhythm was continuously monitored while his expressions were filmed. Periods of stress as judged from the filmed interview appeared to correlate well with the number of ventricular premature beats per minute. During treadmill exercise stress testing there was a paucity of ventricular arrhythmias, suggesting that the heart was more sensitive to emotional than hemodynamic stress.

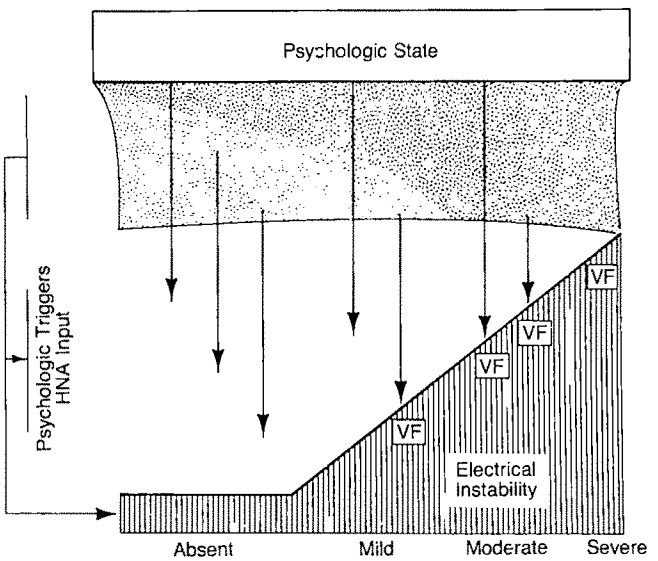
Until quite recently the victim of sudden death was not available for study. But now the Biblical occurrence of having "risen from the dead" is becoming common. This provides a unique opportunity for investigating the psychophysiologic factors implicated in

sudden death. The successfully resuscitated patient continues to have electrical instability of the myocardium, exhibits a high prevalence of ventricular arrhythmia, and has an extreme predisposition for reexperiencing ventricular fibrillation (69). Over the past few years we have studied 150 patients with oft-recurring life-threatening ventricular arrhythmias. Of these, 117 were closely evaluated and followed by a "higher nervous activity" task force consisting of a psychiatrist, a psychologist, and members of the cardiovascular group. Among them, 62 patients had had one or more episodes of ventricular fibrillation.

In a number of these patients three sets of conditions that contributed to the occurrence of malignant ventricular arrhythmia appeared to be operating. The first was the presence of electrical instability of the myocardium. In the majority of these patients such instability was due to antecedent disease, usually ischemic heart disease. Second was the presence of a psychologic state intense enough to pervade and burden daily life. Most frequently it was either an affective depression or a sense of psychologic entrapment without possible exit. The third condition was a proximate psychologic trigger for the ventricular arrhythmia—either 1) occurrence within the 24 hours preceding the malignant arrhythmia of a highly meaningful and clearly defined psychologic event that may be judged as a precipitant of the rhythm disorder, or 2) significant emotional stresses that provoke ventricular arrhythmia while the patient is in the hospital. The dynamic interrelationship of these three factors is illustrated in figure 5. In the presence of extreme electrical instability of the myocardium, a minor psychologic trigger may suffice to provoke a major arrhythmia even in the absence of a substantial psychologic state. At the other extreme of the spectrum of electrical instability, when there is no myocardial derangement, ventricular fibrillation cannot be precipitated irrespective of the magnitude of the psychologic trigger or the extent of the psychologic state.

In our initial investigations we searched for a psychologic trigger in the 117 patients who had been studied by the interdisciplinary task force (70). We identified 25 patients (21%) who were judged independently by members of the task force to have had a psychologic trigger for their arrhythmia. The "trigger" group was differentiated from the other 92 patients with malignant arrhythmia by having less demonstrable cardiac pathology while experiencing more serious ventricular arrhythmias. In the majority of "trigger" patients (15 of 25), the psychologic event occurred less than one hour before the onset of the malignant arrhythmia. The major affect was anger, which was noted in 17 of the 25 patients. These studies are as yet at their inception but hold promise in helping to clarify the essential psychophysiologic factors which may play a role in the genesis of ventricular arrhythmias in patients predisposed to sudden death.

FIGURE 5
Interactions Between Psychologic Variables and Myocardial Electrical Instability[a]



[a]When electrical instability of the myocardium is absent, as shown at left of figure, neither psychologic state nor trigger can provoke ventricular fibrillation (VF). At the other end of the spectrum, when electrical instability is marked, as shown at the extreme right of figure, no psychologic trigger is required to induce ventricular fibrillation. In the intermediate zone an interplay of these three factors determines the predisposition to ventricular fibrillation. Thus lesser magnitude of triggers is required with increased psychologic state or with a greater degree of myocardial electrical instability.

FINAL COMMENTS

Considerable evidence lends credence to the hypothesis that inputs from the central nervous system constitute transient risk factors for sudden cardiac death from ventricular fibrillation. While data from animal studies are direct and persuasive, evidence in man is as yet incomplete and circumstantial. The neuroeffector sequence impinging on the myocardium is the summated expression emanating from complex interactions between immediate sensory perception, recall of past emotional events, preexisting conditioned reflex pathways, concentration of neurochemical precursors at various brain sites, and a host of other factors as yet to be identified. The available evidence, however, leaves little doubt that psychophysiologic factors can no longer be ignored if the trigger mechanisms for the sporadically occurring ventricular arrhythmias are to be comprehended. There are sufficient data to attribute the immediate precipitation of sudden cardiac death in many instances to psychophysiologic factors. The lack of conclusive evidence need not hamper the development of an integrated approach involving cardiologist, psychiatrist, and neuroscientist for studying and managing patients afflicted with recurring life-threatening ventricular arrhythmias. Indeed the exercise of such multidisciplinary cooperation has enabled us to prevent cardiac arrest and

prolong the lives of victims afflicted with repeated episodes of ventricular fibrillation (69, 71).

In animal models with cardiac ischemia, a primary role in the occurrence of ventricular arrhythmias can be ascribed to the sympathetic limb of the autonomic nervous system. However, enhanced sympathetic neural traffic to the heart does not constitute the sole basis for the genesis of ventricular arrhythmias in man. Indeed, beta-adrenergic blocking drugs alone have proved inadequate for preventing malignant ventricular rhythm disorders. No doubt numerous central mechanisms are involved in the precipitation of such arrhythmias. Current developments in neurochemistry and psychopharmacology promise new insights into the underlying mechanisms regulating cardiac activity as well as clarification of the basis for a number of arrhythmias. In any case, resolution of the problem of sudden death requires that the central nervous system be restored to its premier integrating role of cardiac function.

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Overview: The "Wife-Beater's Wife" Reconsidered

BY ELAINE HILBERMAN, M.D.

Although not a new phenomenon, wife-beating has been largely ignored by mental health professionals. The author describes a context in which the physical abuse of women as wives can be understood and suggests alternative theoretical constructs to traditional theories of masochism as explanations of why women stay in violent marital relationships. She discusses the societal attitudes that normalize the use of family violence as well as attitudes about women, men, and sex roles that leave women vulnerable to assault; summarizes the research on abused wives; identifies a specific stress response syndrome caused by violent abuse; and elaborates on the problems posed for clinicians working with abused women.

Wife-beating, although not a new phenomenon, has been largely ignored by mental health professionals. Until recently the literature and research on family violence were limited to child abuse and murder, since these categories alone were generally accepted as serious problems deserving of public attention and intervention. Although extensive research and service efforts have focused on family dynamics that foster child abuse and alcoholism, there has been inadequate recognition of the aggression that is often directed toward the wives and mothers in such families. This "selective inattention" has been noted by most contemporary researchers and writers (1-3) and was documented by O'Brien's finding (4) that the index of the *Journal of Marriage and the Family* from its inception in 1939 through 1969 contained no references to "violence."

Wife abuse, when it was identified, was generally assumed to represent some intrapsychic liability on the part of the victim. This psychiatric labeling or attribution of blame reflected and reinforced the societal belief that spouse abuse was an isolated problem in unusually disturbed couples in which the violence was viewed as "fulfilling masochistic needs of the wife and necessary for the wife's (and the couple's) equilibrium" (5). Several important clinical implications follow from these explanations of causality. First, when spouse abuse is perceived as occurring only infrequently and in deviant relationships, it is defined as a private rather than a public problem. Second, if the victim can be seen as provoking or needing the abuse, mental health professionals can more comfortably focus on the "meaning" of the violence than on the fact of the violence per se. Third, since women themselves are aware of and even share these attitudes, they are reluctant to reveal the abuse and are most usually "silent victims." Thus there has been a covert alliance between victims and clinicians, in which treatment of symptoms is offered as an alternative to the more direct identification of the problem and the appropriate intervention and protection of abused women (3, 6, 7).

Because spouse abuse is usually wife abuse, I will focus on the female victims of marital violence. The intent of this overview is to present a context in which the physical abuse of women as wives can be understood. A review of the pertinent literature is summarized in terms of information that is essential for the assessment and treatment of victims in danger. Although the sociological and feminist literature on this topic has expanded rapidly in the last decade (1-3, 7-13), the clinical and psychiatric literature is relatively new and is often found outside traditional sources (6, 14-22).

I will discuss societal attitudes that normalize the use of family violence as well as attitudes about women, men, and sex roles that leave women vulnerable to assaults by significant other men. I will summarize clinical research reports about abused wives; these suggest alternative theoretical constructs to explain why women remain in violent marital relationships. A more detailed account of my own clinical experiences is offered and includes the identification of a specific stress response syndrome that is the direct result of violent abuse (19). I will discuss treatment implications and recommendations, clarify the role of the mental health professional, and elaborate the problems posed for clinicians working with abused wives. I hope that this article will mobilize the interests and capabilities of the psychiatric community for urgently needed clinical services and research in this area.

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THE SOCIETAL CONTEXT

Violence as a Norm

There is considerable irony in our lack of attention to family violence at a time of great public concern and fear about violence in general. As Straus noted in the foreword to Gelles' *The Violent Home* (1), the social definition of the family as nonviolent "causes a perceptual blackout of the family violence going on daily all around us in 'normal' families." Although the concept of family is cherished as a source of nurturance and mental health, the family is the most frequent single locus for violence of all types, including homicide (23). Violence is said to occur in 50% of American families, so that the marriage license might be viewed as a "hitting license" (1, 21, 23, 24).

Family violence, in contrast to violence outside the family, is more often perceived as normal, legitimate, and instrumental (1, 2, 23). This attitude is most evident in the corporal punishment of children. That violence between spouses is also socially condoned is reflected in results of the 1968 interview survey conducted for the National Commission on Causes and Prevention of Violence. This survey of a representative national sample of adult women and men found that 20% approved of husband- and wife-hitting (25). Spouse abuse is not limited to a particular social class or ethnic group, although the highest reported incidence is among the poor. Poor people are more likely to come to the attention of a public agency, while the privacy of middle- and upper-class women is protected by their personal physicians or attorneys. In a study of 600 couples who were in the process of divorce (26), 40% of lower-class women and 23% of middle-class women reported physical abuse by spouses. A 1979 survey of wife abuse in Kentucky (27) found that income levels were not good predictors of family violence. School dropouts with less than an eighth-grade education appeared to be less violence-prone than those who had some college education.

Family violence has several important societal ramifications beyond the suffering of individuals (28):

1. Injury and death. Of all murders in this country, 20%–50% occur within the family. Police are called to intervene in domestic disputes more often than in all other criminal incidents combined (2, 29–32), and 20% of all police fatalities occur during such interventions (3, 28). A Kansas City, Mo., police study (33) reported that 40% of the city's homicides were between spouses. In more than 85% of these homicides police had been called in at least once before the fatal episode, and in half of these cases they had been called in five times during the 2 years before the murder (33).

Although reports consistently reveal that the home is a violent setting, Dobash and Dobash (30, 34) have observed that researchers often fail to note that this violence is not evenly distributed among family members but is disproportionately directed toward women. Female victims of homicide are more often killed by a spouse, while the majority of male victims are killed by someone outside the family. According to Dobash and Dobash (30, 34), about 40% of all female homicide victims were killed by husbands, but only 10% of male victims were killed by wives. It has also been reported (35) that among those who murder spouses, wives were seven times more likely than husbands to have murdered in self-defense. Thus, men are much more likely to commit homicide than women and women victims are usually married to their assailants.

2. Violence as learned behavior. Gelles and Straus (23) noted that the family is the primary training ground for violent behavior: "A person is more likely to observe, commit, and to be the victim of violence within the family than in any other setting." Beating of women in the home is often accompanied by physical and/or sexual abuse of the children (15, 19, 20). Children who are abused often grow up to abuse their offspring, and children who see violent parental interactions often have physically abusive relationships in adulthood. Thus violence breeds violence (1, 7, 23).

Violence Against Women

The recent attention to wife abuse is the direct result of the women's movement and follows on the heels of the anti-rape movement. It is best understood as an expansion of the concern about female sexual abuse to encompass the broader category of violence against women. The large majority of victims of spouse abuse, incest, and rape are female, and their assailants are most often men who (with the possible exception of rapists) are trusted relatives and intimates. In all of these acts of violence, women and girls are perceived by their assailants as legitimate targets for male aggression (3, 36–40). The basic fact of the victim's femaleness is used by the offender to justify the assault, and the complaining woman is suspected of fabrication, provocation, or seduction, while the assailant is protected.

Wife-beating has had social and legal sanction throughout history (3, 30, 36, 41, 42). Martin (3, 43) noted that the word "family," derived from the Latin *familia,* in Roman culture connoted the totality of slaves belonging to an individual. As early as 753 B.C., married women were defined as "necessary and inseparable possessions" of their husbands (30). There is ample documentation of the wife's obligation to obey her husband and of the husband's legal and moral responsibility to control his wife. The husband's authority to chastise his wife was explicitly written into the laws of church and state and later incorporated into English common law. Blackstone, in 1763, explained the husband's right of chastisement: "For, as he is to answer for her misbehaviours, the law thought it reasonable to entrust him with his power of restraining her by domestic chastisement" (30).

The husband's right to hit his wife was written into

law in the United States in 1824 with the restriction that "he . . . [use] a switch no bigger than his thumb" (3). This law was overturned in 1874 when the North Carolina Supreme Court ruled that "the husband has no right to chastise his wife under any circumstances." The importance of this judgment was unfortunately undone by the court's qualifying statement: "If no permanent injury has been inflicted, nor malice, cruelty nor dangerous violence shown by the husband, it is better to draw the curtain, shut out the public gaze, and leave the parties to forget and forgive" (3).

Although wife-beating is no longer legitimized by statute, current criminal justice procedures are consistent with "drawing the curtain." Field and Field (44) commented that

> the evolution of a system in which criminal enforcement is left entirely to the whim of the victim . . . implies official acceptance of our acquiescence to the policy that violence between "consenting" adults is a private affair. . . . The poles of enforcement are the rigorous prosecution of the person accused of assaulting a stranger and the nonprosecution of the one who assaults a marital partner.

Violent marriages are currently maintained by pervasive patterns of sex discrimination (3, 38, 39, 43). Men use violence as a way of maintaining a superior power position in the family. Their wives understand that neither separation nor legal intervention will necessarily end the violence (28, 44, 45). The man is protected, and the abused woman comes to believe that marital violence is the norm and that she deserves the abuse. This self-blame is reinforced by "helping" agencies that ignore and demean the victim or accuse her of provoking the assault (3, 7). The woman who leaves a violent home is denied child-care facilities; equal educational, vocational, and economic opportunities; and a legitimate self-supporting and autonomous role outside the home (38, 39).

DEFINING WIFE ABUSE

Most clinicians who study spouse abuse have arrived at similar working definitions of marital violence (6, 15, 18, 20). (The term "marital" does not imply a legal relationship but includes any relationship involving cohabitation and sexual intimacy.) In general, an abused or battered wife is one who is subjected to serious and/or repeated physical injury as a result of deliberate assaults by her spouse. Scott (20) graded the severity of such abuse along the following continuum: 1) not requiring medical attention, 2) requiring outpatient attention, and 3) requiring hospitalization. He also categorized the frequency of abuse as 1) regular, 2) episodic (situational), 3) increasing, and 4) terminal.

There is considerable semantic and attitudinal confusion about these definitions of violence. Pushing and shoving may not constitute a threat to life and may be considered permissible by a couple, but the same behaviors would be considered violent acts if the assailant were a stranger. As another example, Gelles (1) commented that individuals do not label the use of physical force on children as "violence" because of the "powerful pro-use-of-physical-force-on-children norms." He asserted that if violence is defined as an act with the intent of physically injuring the victim, then the physical punishment of children is violent.

THE FAMILY CONTEXT AND THE CONSEQUENCES

In this section I will describe the family setting and the psychological and behavioral consequences of wife-beating, as derived from clinical research reports (1, 7, 14–21, 46–48). One of the largest clinical samples has been reported by Gayford (15–17), who studied 100 battered women admitted to a shelter in England. British psychiatrists have been somewhat more aware of this problem because of Pizzey's efforts to initiate a network of shelters for abused women and their children (7, 46). Gelles' 1974 sociological study of 80 families (1) was the first important clinical research effort in the United States.

A 1977–1978 study by Munson and myself of 60 battered women in a general medical clinic setting (19) is similar to work reported by Dewsbury (6) in that the abused women were not selected by virtue of admission to a shelter or by awareness that the abuse they suffered warranted attention. The history of violence was known to the referring clinician in only 4 of our 60 cases, although most of these women and their children had received ongoing medical care at the clinic. Information about the women was acquired by dialogue with them, while material about spouses was almost always indirect because these men adamantly rejected attempts to engage them in medical or psychiatric treatment. Involvement with husbands was further limited by the women's need to maintain silence about clinic visits and by our concern that conjoint treatment in the absence of provisions for safety might result in an escalation of violence. Other researchers have also found that husbands are unwilling to participate in interviews about family violence. Gelles (1) abandoned initial plans for conjoint interviews because early interviews with couples suggested the potential for precipitation of further violence.

Families of Origin

Munson and I (19) found that lifelong violence was a frequent pattern for many abused wives. Half of the women we studied reported violence between parents (usually the father assaulting the mother), paternal alcoholism, and their own physical and/or sexual abuse as children. The women's husbands were said to have had even more early exposure to emotional deprivation, alcoholism, lack of protection, and violence, both

as witnesses and as objects of abuse. Suicides and homicides among family members and neighborhood acquaintances were common occurrences and were usually committed with guns, which were normal household possessions. These findings about violence in families of origin are consistent with other reports. Of the sample studied by Gayford (15), 23% gave histories of parental violence; violent backgrounds were even more prevalent for their husbands, half of whom were said to come from violent families. Scott (20) also described a pattern of violent fathers and passive mothers in families of origin.

Most of the women we studied (19) left home at an early age to escape from violent, jealous, and seductive fathers who kept their wives and daughters imprisoned. Marriage during teenage years was the norm for these women, and many were pregnant at the time of marriage or had had children before marriage. They viewed pregnancy as the only way they would be allowed to leave the family—the alternative to paternal control was paternal rejection and a way out of the home. In Gayford's sample (15), 60% of the women reported premarital pregnancies. Rounsaville (48) described these marriages as occurring precipitously and during stressful periods. Most usually, the future husbands did not exhibit violent behavior toward the women until they were married, and the women ignored other evidence of the men's potential for violence because of the urgent need to marry in order to "escape" from home. Once married, these women found themselves with jealous and assaultive husbands, often replicating their lives before marriage.

Alcohol and Violence

An association between alcohol use by the batterer and marital violence has been noted in all of the relevant studies. Drinking accompanied the violence in 44% of Gelles' sample (1) and 93% of our sample (19). Gayford (15) described drunkenness as occurring regularly in 52% of the men and occasionally in an additional 22%. In a survey of 100 wives of alcoholics, none of whom had been identified as victims of marital violence, Scott (20) found that 72% of the women had been threatened, 45% were beaten, and 27% described potentially lethal assaults. Drunkenness is not always accompanied by violent behavior, however, and wife abusers who drink heavily also beat their wives when they are sober. Because of this variability, Gelles (1) hypothesized that wife abusers become intoxicated in order to carry out a violent act. Drinking is thus used to disavow the deviant behavior and to provide a "time out" during which the assailant is not responsible for his actions.

The Marriage

The violent relationship is one of extraordinary intensity (19, 21). When they are not behaving aggressively, wife abusers have been described as childlike, remorseful, and yearning for nurturance. This picture of fragility was confirmed in our study (19) by occasional reports of a husband's suicidal or psychotic behavior when his wife threatened to dissolve the relationship. The women felt quite sorry for their spouses because of their histories of deprivation and abuse. These men could never understand or acknowledge a termination without murderous rage, so marriage became a life sentence for their wives (7, 19). When husbands threatened homicide, they were taken literally because threats and wishes became a reality with explosive suddenness in these relationships. Many women left their marriages for brief periods but invariably returned because of economic and emotional dependence on husbands and threats of further violence from which they had no protection. For example, one woman made active plans to leave her marriage despite her husband's threats of violence but retreated abruptly when her closest friend, also an abused woman, was murdered by a violent spouse.

In many violent marital relationships, there seems to be a cycle of violence, with three phases that vary in time and intensity. There is a phase in which tension builds, a phase in which violence erupts, and a phase of relief from tension. In this last phase, the husband is often kind, loving, and remorseful. This postviolence reconciliation leaves the women hopeful that her husband will change and serves as a powerful reinforcer for her to continue the marital relationship (21, 49). Battered women are most often seen by clinicians toward the end of the first phase or during the last phase. When the anticipatory anxiety becomes intolerable, women sometimes act to precipitate the inevitable assault. In contrast, during the reconciliation period victims often appear unconcerned about the violence. It is possible that both of these behaviors may be misinterpreted as masochistic by clinicians.

Jealousy

Extreme jealousy was reported in 57 of the 60 marriages that Munson and I studied (19), with husbands making active and successful efforts to keep their wives ignorant and isolated. If the women left the house for any reason, they were met with accusations of infidelity that culminated in assault. Clinic visits were often made in secrecy, and some women were beaten regularly when they returned from the clinic. Other channels of communication were also closed off. Friendships with women were discouraged either by the husband's embarrassing his wife in front of her friends or by his accusations that the wife's friends were lesbians or "trash." Many husbands refused to allow their wives to work, and others tried to ensure that both partners worked at the same place so they could monitor the woman's activities and friends. Thus for these women isolation was complete and potential support unavailable. The women were painfully aware of this; one commented, "He'd do anything he

could to get me down to where I would not go out in the world."

This extraordinary possessiveness was apparent in Gayford's sample (15)—66% of the women said their husbands accused them of infidelity or checked on their activities. Gelles (1) and Scott (20) have identified the "third degree," a phenomenon in which a husband interrogates his wife for hours until she admits to some infidelity simply to end the argument. In reality, many of the husbands studied engaged in extramarital liaisons, while few of the women did so. Gayford (15) and Scott (20) have both suggested that extreme cases of delusional jealousy warrant immediate separation because of the potential for homicidal violence.

Marital Violence

The women we studied (19) reported that violence erupted any time a husband did not immediately get his way. A common pattern was for the husband to come home late after being with another woman and to goad his wife into an argument that ended in violence. The assaults usually occurred at night and on weekends, so children were witnesses and participants (1, 19). In many cases, the oldest child had not been fathered by the husband, and violence toward the wife was extended to the "bastard kid." Older children also became involved when they attempted to defend or protect the mother.

Some women were assaulted daily, while others were beaten intermittently; they therefore lived in constant anticipatory terror. There was also a subgroup of victims who had sustained one life-threatening injury, e.g., a subdural hematoma or a gunshot wound to the chest. Although there was no further violence in these cases, this was a powerful learning experience for the victim, who behaved thereafter as if she were always in danger.

Assault weapons included hands, feet, fists, rocks, bottles, telephones, iron bars, knives, and guns. Scratching, slapping, punching with fists, throwing down, and kicking were prevalent, with faces and breasts the most frequently mentioned sites of assault (1, 3, 6, 14, 15, 19–21). These assaults led to multiple bruises, black eyes, fractured ribs, subdural hematomas, and detached retinas. Some victims also reported having been strangled and choked until consciousness was impaired. Sexual assaults were common, and women described being beaten and raped in front of the children. Guns were available in most of these homes and were constant threats—some men kept guns in bed to intimidate their wives (19). We know of one woman who slept with her shoes on to permit a "fast getaway" (19).

Most women reported changes in the pattern of violence during pregnancy (19). There was increasing abuse for some, with the abdomen replacing the face and breasts as the target for battering. This often led to abortions and premature births (1, 15, 19, 21). It has been hypothesized that this behavior represents a prenatal form of infanticide or child abuse (1, 50). A smaller number of women reported less abuse with pregnancy, and one woman deliberately stayed pregnant to avoid violence (19).

The coping strategy used most often during the assault was to get away temporarily, although the women found there were few places to go. Some women sought recourse through the criminal justice system, but these attempts were frustrated by the unresponsiveness of magistrates and law enforcement officials, as well as the husband's retaliative violence (8, 19, 21, 28, 42, 44). The few women who resorted to counterviolence did so in desperation when other options had failed. In contrast to the husband's violent behavior, these women used violence in response to a direct threat to life. Their behavior usually came as a surprise to them because they had been unaware of the extent of their rage and their own capacity for violence (19).

The Children

Almost all clinicians are impressed with the correlation between child abuse and spouse abuse. Gayford (15) reported, in his study of battered women, that 37% of the women and 54% of the men beat the children. Munson and I (19) identified the physical and/or sexual abuse of children in a third of the families we studied. Whether the children were themselves battered or were onlookers to parental violence, they were deeply affected by the climate of violence in which they lived (51). Emotional neglect, abuse, and frequent separations were the norms—the children were never certain who would leave and when. Thus, children in violent homes, as witnesses and targets of abuse, are quite vulnerable (1, 15, 19–21).

A high incidence of somatic, psychological, and behavioral dysfunctions has been described in such children (15, 19). Psychosomatic illnesses were especially prominent in the children we studied and included headaches, abdominal complaints, asthma, peptic ulcer, rheumatoid arthritis, stuttering, and enuresis (19). Depression, suicidal behavior, and overt psychosis were seen in a few of these children and adolescents.

The following portrait of the children emerged from our study (19). Preschool and young school children in violent homes had somatic complaints, school phobias, enuresis, and insomnia. The insomnia was often accompanied by intense fear, screaming, and resistance to going to bed at night. This behavior was temporally related to the violence in the home—much of the wife-beating occurred when the children were in bed. Most children had impaired concentration and difficulty with school work. Older children began to show differential behavior patterns that divided along gender lines. The most frequently reported cluster for boys—aggressive disruptive behavior, stealing, temper tantrums, truancy, and fighting with siblings and schoolmates—was notably absent in girls.

As Gayford (15) observed, "Some children were so disruptive that they could not be contained at normal schools, primarily because of their violence towards other children and, in some cases, their teachers." In a 1977 personal communication Pizzey confirmed the effectiveness of this training for violence, noting that her network of crisis shelters could not contain the adolescent boys from violent homes because of their uncontrollable aggression. Separate housing and personnel were necessary for these boys, who invariably destroyed furniture and possessions and broke windows until they had gone through a period of resocialization.

In contrast, teenage girls continued to have an increasing array of somatic symptoms and became withdrawn, passive, clinging, and anxious (this pattern also occurred in a smaller number of boys). Teenage girls further suffered from the perpetual surveillance and accusations of sexual activity by their fathers, who were seductive and, in some cases, overtly incestuous. Finally, there were reports of married daughters who were battered and of grown sons who were alcoholic and violent, thus completing the cycle.

Battered Women: Psychological Consequences

In our sample (19), the reasons for psychiatric referral usually clustered around physical symptoms or mixed anxiety/depressive symptoms that the women related to marital problems. Marital problems were generally defined in terms of the husband's alcoholism, financial irresponsibility, and promiscuity; the battering was never mentioned. Women with physical symptoms had been referred because of chronic use or abuse of tranquilizers or analgesics, unremitting symptomatology, treatment noncompliance, and frequent clinic visits.

Somatic complaints, conversion symptoms, and psychophysiologic reactions were abundant, as evidenced by frequent clinic visits for headaches, choking sensations, hyperventilation, asthma, gastrointestinal symptoms, allergic phenomena, and chest, pelvic, and back pain. Symptoms were often connected to previous sites of battering. One husband regularly scratched and gouged his wife's back to the point of bleeding and scarring; when there was increased tension at home her back "broke out" in giant urticaria (19).

Treatment of preexisting chronic disease was complicated by the affective states of these women. For example, an epileptic woman required multiple hospitalizations for seizures. Noncompliance in the use of anticonvulsives was her only escape from a savagely brutal husband who refused to allow her to leave the house unless she needed medical attention. Some women were unable to leave battering marriages because of physical disabilities that left them helpless, such as blindness and Huntington's chorea (19).

Psychiatric histories suggested prior psychological dysfunction for more than half of the women in our study (19). Depressive illness was the single most frequent diagnostic category, with manic-depressive psychosis, schizophrenia, alcoholism, and personality disorders all represented to a lesser degree. There were relatively few alcoholic women in this sample, but the women patients in a nearby alcoholic rehabilitation program were almost all victims of parental and marital violence. Some women in our study had been hospitalized repeatedly, often because of the acute onset of a reactive psychosis in which they lost control of aggressive impulses. Others had sought help from crisis lines, outpatient mental health facilities, and emergency rooms. Almost all of these women had made frequent visits to local physicians for somatic complaints, anxiety, insomnia, or suicide attempts, usually by drug overdoses. Most had been treated intermittently or chronically with sedative-hypnotics, tranquilizers, and/or antidepressants. Although these women had had multiple contacts with various clinicians over the years, they did not tell the physicians of the violence, nor did the physicians ask them (6, 19).

These findings of a high incidence of depressive symptoms, treatment with psychoactive drugs, and suicidal behavior are consistent with other reports (6, 15–17, 19, 20, 48). Of Gayford's sample of 100 women (15), 71 had symptoms that were treated with antidepressants and tranquilizers, 42 had attempted suicide, usually by overdose, 46 at some time had been referred to a psychiatrist, and 21 had a diagnosis of depression. Half of Dewsbury's sample of 15 women (6) had a history of psychiatric hospitalization.

The Impact of Violence: A Stress-Response Syndrome

Munson and I (19) have described a uniform psychological response to violence that was identical for the entire sample of battered women. These women were a study in paralyzing terror that was reminiscent of the rape trauma syndrome (52), except the stress was unending and the threat of assault ever present. Agitation and anxiety bordering on panic were almost always present. Events even remotely connected with violence—sirens, thunder, a door slamming—elicited intense fear. There was chronic apprehension of imminent doom, of something terrible always about to happen. Any symbolic or actual sign of potential danger resulted in increased activity, agitation, pacing, screaming, and crying. The women remained vigilant, unable to relax or to sleep. Sleep, when it came, brought no relief. Nightmares were universal, with undisguised themes of violence and danger.

In contrast to their dreams, in which they actively attempted to protect themselves, the waking lives of these women were characterized by overwhelming passivity and inability to act. They were drained, fatigued, and numb, without the energy to do more than minimal household chores and child care. They had a pervasive sense of hopelessness and despair about themselves and their lives. They saw themselves as in-

competent, unworthy, and unlovable and were ridden with guilt and shame. They thought they deserved the abuse, saw no options, and felt powerless to make changes.

Like rape victims, battered women rarely experienced their anger directly (53), although their stories elicited anguish and outrage in the listener. It is probable that the constellation of passivity, guilt, intense fear of the unexpected, and violent nightmares reflected not only fear of another assault but a constant struggle with the self to contain and control aggressive impulses. The violent encounter with another person's loss of control of aggression precipitates great anxiety about one's own controls. We know from rape victims that one such encounter is sufficient to bury aggression (54). In the life experiences of battered women, there is little perceived or real difference between affect, fantasy, and action. Thus it is not surprising that fear of loss of control was a universal concern. These fears were often expressed in vague, abstract terms but were unmistakably linked to aggression.

A minority of women did lose control of aggressive impulses. Some women became frankly homicidal and others fantasized detailed plans for murdering their husbands as a way of coping with their anger. They locked up guns and knives to prevent easy access to weapons, without knowing why they did so. Some displayed aggression by becoming adept at verbal retaliation, while others fought back physically. However, these cases were the exceptions; passivity and paralysis of action more accurately described the majority of these women. Aggression was most consistently directed against themselves, in the form of suicidal behavior, depression, grotesque self-imagery, alcoholism, or self-mutilation. One woman's face and body were covered with self-inflicted scratches and scars. Passivity and denial of anger do not imply that the battered woman is adjusted to or likes her situation. These are the last desperate defenses against homicidal rage (19).

TREATMENT ISSUES

The Clinician's Response

The response of health professionals to wife-battering has generally been characterized by inattention, blame, and disbelief. Explanations for the extraordinary difficulty experienced by clinicians in confronting these issues are complex and multidetermined. First, both male and female clinicians share the pervasive cultural biases and myths that stereotype and denigrate women (e.g., the ambivalent image of woman as virgin or seductress). Second, female victims of male aggression may not feel safe in revealing the abuse to male clinicians. Third, even when clinicians are sensitive to the possibility of such victimization, they may find it difficult to initiate direct questions because the details of such savage abuse can be overwhelming, and the clinician may feel as anguished or helpless as the patient. Finally, the frequency with which family violence occurs makes it likely that an individual clinician may have personally experienced a violent family or may have a friend or family member who was abused. It is not surprising, then, that the pressure on the clinician is to achieve some distance from the victim. This distancing is achieved by disbelief, labeling, accusations that the victim "needs" the abuse or could have controlled or prevented it by being less provocative, and the unfounded assumption that most victims come from poor or black families and are thus different from the clinician.

Identification

Clinicians must be aware of the prevalence of family violence and the variety of ways in which victims may present themselves. The portal of entry to health care is determined by the nature of the presenting symptom. Victims with physical injuries are seen on emergency and trauma services and are then referred to surgical subspecialists and dentists (14, 22). Those with chronic psychosomatic symptoms, anxiety, and insomnia make multiple visits to general practitioners and family doctors. Battered women who are severely depressed, anxious, or suicidal are referred to crisis intervention units or psychiatrists for evaluation, while those with functional psychoses enter psychiatric hospitals. Victims who are substance abusers may come to the attention of drug or alcohol programs. Some victims can be identified by virtue of their relationship to other identified patients, such as alcoholic husbands and abused or disturbed children. A high index of suspicion facilitates victim identification, but there is no substitute for a direct inquiry about violence. Clinicians must be aware that victims for whom violence has become a way of life do not define the abuse as "violence" and will deny its occurrence. Thus it is more helpful and productive to base questions on behavioral descriptions, e.g., "Is anyone at home hitting you?"

Once established, marital violence tends to escalate, and the possibility of a lethal outcome must be considered throughout the diagnostic and treatment process. This is especially important in the usual clinical situation, in which only the abused wife is in treatment and the violence at home is continuing. In theory, the aim of treatment might be to terminate the violence or the marital relationship. In practice, however, the husband usually does not perceive his behavior as a problem requiring intervention, and his wife maintains secrecy about any intervention to avoid further violence or interference with her treatment. Removal of women and children from the home to a safe environment is therefore essential to adequate long-term treatment and rehabilitation (3, 7, 12, 15, 19, 21, 39, 46, 55–58). In the absence of such shelter, battering men will con-

tinue to follow, harrass, and assault their families wherever they go.

Why Do They Stay?

Women are trapped in violent homes by a complex interplay of intrapsychic and external constraints. Gelles (59) cited three major factors that influence the abused woman's likelihood of staying in the marriage:

1. The less severe and less frequent the violence, the more likely the woman is to stay.
2. The more a woman was struck by her parents as a child, the more likely she is to stay.
3. The fewer her resources and the less her power, the more probable it is she will stay.

Clinicians must identify the woman's internal and external barriers to self-protection in order to help her become capable of action to terminate the violence or the marriage.

Among contemporary researchers, there has been little enthusiasm or supporting data for the theories of masochism that have been traditionally invoked to explain why women remain in violent relationships (15, 20, 39, 48, 49, 59, 60). Clinicians and social scientists highlight the absence of economic resources as a factor that severely limits the options of women attempting to leave their marriages. Most women are economically dependent on their husbands and have neither the job skills nor opportunities that are necessary for financial independence. The economic realities for female-headed single-parent families are well documented (58).

In an elegant critical review of theories of female masochism, Waites (60) argued,

> Female motivation, like motivation generally, must be studied in the context of external constraints such as social choices available and the consequences of particular choices. In this light, the masochistic woman often appears as an individual who has almost no choice, given the lack of social support for extricating herself from her situation or who is confronted with alternatives which are, for the most part, negatively valued.

She concluded that theories of female masochism are inadequate as explanations of actual behavior because "in situations in which choice is externally restricted, the question of internal motivation approaches irrelevance" (60).

The combination of continuing threats and violence by the husband and the absence of provisions for safety is universally identified as a deterrent to action. As Martin (3) asserted, "Battered women give many reasons or rationalizations for staying, but fear is the common denominator. Fear immobilizes them, ruling their actions, their decisions, their very lives." She cited Maccoby and Jacklin (61), who suggested that while fear is an arousal state, "the possibility exists that females show immobilization and other 'passive' behavior primarily when they are afraid." In a paper presented to the Association for the Advancement of Psychoanalysis in October 1977 Symonds also concluded, "The response of women to the violence of the immature, impulsive and exploitive husband is to be profoundly terrorized and traumatized by this violence"; they are "brainwashed by terror." He conceptualized this fear as a form of traumatic psychological infantilism or frozen fright, which in other reports (62, 63) he described as the acute psychological response to rape and other violent crimes.

There has also been increasing interest on the part of several authors (21, 49, 60, 64) in Seligman's concept of learned helplessness (65) as an explanation of the abused wife's continued passivity when external options seem possible or are made available:

> When an organism has experienced trauma it cannot control, its motivation to respond in the face of later trauma wanes. Moreover, even if it does respond and the response succeeds in producing relief, it has trouble learning, perceiving, and believing that the response worked. (65, pp. 22–23)

Battered women do not believe that they can escape from the batterer's domination (49). This expectation of powerlessness and inability to control one's destiny, whether real or perceived, prevents effective action. Traditional sex role socialization norms further reinforce the idea that women's needs are fulfilled and their identities derived only indirectly through their men. As a consequence women have inadequate preparation for independence and inadequate skills for assertive problem-solving behavior.

These descriptions of the victim's immobilization and inability to leave the violent home are reminiscent of the pathological transference that develops between hostages and hostage-takers. According to Ochberg (66), the "Stockholm syndrome" includes positive feelings on the part of the hostage toward the captor, negative feelings on the part of the hostage toward the authorities responsible for rescue, and reciprocation of positive feelings by the hostage-taker toward the captive. He suggested that this pathological transference is based on the captive's terror, infantile dependence, and gratitude and that this relationship may, in fact, promote the survival of the hostage.

The constructs offered as explanations of why women remain in destructive relationships are not mutually exclusive and have important implications for the choice of treatment models and the availability and nature of programs for battered women (49, 57, 60, 64, 67). The most significant treatment implication derives from the way in which the locus of the problem is defined. Theories of masochism, which locate the problem within the victim, lead to certain assumptions about victim-offender relationships and programmatic responses that have been described by Lynch and Norris (67). For example, if the victim is held responsible for the abuse, there will be no support for spouse

abuse programming, and existing helping agencies will set the victim up for further abuse (67).

Clinicians working with battered women comment on the frequency with which helpers become frustrated and angry with their clients (7, 19, 49). Even victims who initiate the request for assistance and shelter and who are provided with resources often show a pattern of secrecy, continued protection of the assailant, and failure to follow through. It often takes four or five separations from an abusive spouse before the fantasy of change within the marriage ends and a decisive separation occurs (19, 21).

As Walker (49) pointed out, "It is probable that battered women do not accept the helper's assistance because they do not believe it will be effective. This can be attributed to the learned helplessness hypothesis in which their cognitive set tells them that no one can help them." One treatment implication of this learning theory is that clinicians may need to view changes in behavior and cognitive sets as primary, with the expectation that motivational and affective correlates will follow. In order to reverse this conditioning for helplessness, clinicians must find new ways to motivate clients to try new behaviors (45).

Clinicians who work with battered women are aware of the temptation to take excessive responsibility for the victim's treatment in the response to her presentation of herself as incompetent and unable to help herself. These attempts to "rescue" the victim may lead her to shift her dependence from the abusive husband to the therapist or agency (57, 64), thus reinforcing her passivity and helplessness. Personnel in refuges and shelters are especially sensitive to the importance of achieving "a reconceptualization of self from 'victim' and 'failure' to competent, autonomous person" and have developed a variety of environmental models in which women "come to define their own acts, to accept responsibility for them, and see themselves as capable of acting independently" (57).

Thus the evaluation of battered women must include a careful assessment of those intrapsychic and environmental factors which limit or can be mobilized to facilitate constructive solutions. Limiting factors may be intrapsychic, such as severe depression, passivity, or self-blame, or external, such as economic dependence on the spouse, physical disability, or homicidal threats by the husband. Potential resources include personal strengths, talents, and capacities, as well as concerned family and friends and institutional means for economic, legal, educational, and vocational support. In describing the direct treatment situation, Waites (60) concluded that

> an awareness of external constraints can be helpful in raising the self-esteem of the client as well as in helping her sort out the extent to which her own actions do interact with circumstances to keep her in a destructive situation. . . . Once reality is acknowledged . . . irrational behavior patterns can be examined both from the standpoint of how such patterns were originally acquired and how they may be changed.

Victims' Needs for Intervention and the Clinician's Role

Victims have both short- and long-term needs that include immediate protection and crisis intervention; food, clothing, and shelter; medical, psychiatric, and legal interventions; psychotherapy and counseling; and the mobilization of resources that support an alternative choice to living with an abusive spouse. The extent of these needs will vary according to an individual's life circumstances, adaptive capacities, and psychological status—one client may need all of these resources, while another may need few.

Emergency protection/crisis intervention. An understanding of the immediate determinants of a call for help is crucial to any assessment. Some women come to law enforcement or health facilities when the violence has escalated to a point where their lives are endangered. Others come when they are losing control of their aggressive impulses toward self, spouse, or children. These are emergencies in which immediate protection is imperative. It may be necessary for the clinician to intervene directly with hospitals, law enforcement, child protection, and social service agencies, and/or emergency shelters. The lack of emergency housing for women in crisis has complicated disposition planning enormously (68), and the experiences of the current shelter movement underscore the necessity for a safe milieu in which a woman can begin the process of regaining control of her life without fear. When emergency protection is not needed early in treatment, it may become necessary later, as will be discussed in the section on psychotherapy and counseling issues. Personnel who work with battered women are keenly aware that intervention involves "anticipating the worst and preparing for it" (64).

Treatment of mental illness and stress syndromes. Diagnosis of any clinical psychopathologic syndrome for which a specific treatment modality exists will obviously enhance the battered woman's ability to take control of her life. Hospitalization is indicated when there is acute decompensation or loss of control. I have not found hospitalization useful beyond these urgent needs, perhaps because the hospital environment often tends to reinforce helpless and dependent behavior. As noted earlier, depressive reactions are especially prevalent among abused women and respond to tricyclic antidepressant treatment. However, clinicians should be aware when prescribing such medications of the frequency with which abused women attempt suicide by drug overdose.

The stress syndrome that occurs in response to the violence itself seems to abate only with removal of the threat of violence. The agitation and sleep disturbances have been treated with low doses of antianxiety agents and antidepressants. However, when

there may be dangerous consequences if the woman goes to sleep or decreases her vigilance, medication is usually ineffective and is contraindicated.

Psychotherapy and counseling issues. The battered woman has often experienced a lifetime of abuse and neglect by significant others and in her contacts with medical, social, and criminal justice agencies. She thus comes to believe that she deserves such treatment and to expect no empathy from clinicians. Her feelings of helpless rage, which she may have acted out against her children or herself, further intensify her belief that she is unworthy or bad. To tell the clinician about the spouse's violence or her own frightening impulses is to risk yet another confirmation of her harsh self-judgment. The core issues for psychotherapeutic work, then, are the woman's markedly impaired self-esteem, emotional isolation, and mistrust.

There is a complex mythology about wife-beating that must be identified and challenged early in the treatment of battered women. The victim uses this group of beliefs to "explain" the brutality:

1. The violence is "normal," a perception seen most often in victims who come from violent families of origin.
2. The violence is rationalized: the husband is not responsible because he is sick, mentally ill, alcoholic, unemployed, or under stress.
3. The violence is justified: the woman deserves it because she is bad, provocative, or challenging.
4. The violence is controllable: if the woman is good, quiet, and compliant, her husband will not abuse her.

These beliefs reinforce the battered woman's tenuous denial and protect her husband and her marriage, at the expense of her self-esteem and autonomy and, possibly, her life. They allow her to remain totally enslaved while she believes that she is in control.

In a therapeutic relationship, the battered woman becomes aware of the extent of her rage and its relationship to her guilt, passivity, and fears of losing control. Understandably, this is a crisis point for some women, who may require hospitalization or intensive outpatient work to support their controls. This is also a time when the woman begins to take a more assertive position, with tentative plans for employment, vocational training, education, and termination of the marriage. Because the woman's behavioral changes may lead to escalation of her husband's violent behavior, she should not be pushed to move beyond her perceived "safe boundaries" in acknowledging her anger or confronting her husband. The clinician must assume that the woman is accurate in her assessment of her controls, the extent of the danger to herself and her children, and her husband's potential for violence.

This discussion of treament issues has been based on the assumptions that the abused woman's living situation continues to leave her vulnerable to violent abuse and that her husband does not think he needs treatment, which are the most common conditions in cases of spouse abuse. Treatment options may expand if the victim is protected from further violence and/or her husband is motivated to seek help. The latter situation is more likely to occur when the assailant no longer has access to the victim. Treatment modalities may then include couples work, individual or group therapy with the abusing spouse, or a shelter for violent husbands. Our knowledge of these treament options is preliminary and beyond the scope of this paper. However, some observations on this subject are relevant.

Commenting on her research with couples therapy, Walker (49) noted that battered women are more successful at reversing helplessness when they leave the relationship than when they remain with their partners and try to change the relationship to a nonbattering one. She suggested that before couples therapy can begin, the symbiotic dependency bonds that have developed must be severed by treating the couple as two individuals, with a focus on enhancing independent behavior and teaching new communication skills. In a study designed to test the hypothesis that treatment approaches which encourage ventilation of anger may be dangerous, Straus (69) found that increases in verbal aggression resulted in dramatic increases in physical aggression. Thus clinicians must exert caution in devising treatment strategies for aggressive clients.

Identification of life alternatives. Adequate rehabilitative efforts often require information and assistance from a variety of community resources with which both the battered woman and the clinician may have contact, including 1) medical facilities for reproductive control and medical and mental health care, 2) social service agencies for financial aid, housing, emergency shelter, food stamps, clothing, day care, and child protective services, 3) criminal justice agencies for protection against further violence, 4) legal aid for assistance with warrants, court procedures, and separation and divorce agreements, 5) vocational rehabilitation programs for financial aid and information about educational pursuits, job training, and employment counseling, and 6) women's groups for information, support, and shelter (19, 67).

Consultation and collaboration. Thus far, the role of mental health professionals has been discussed in terms of direct services to victims. There is also a broad spectrum of consultative roles with other clinicians and agencies and in collaborative efforts to develop innovative programs. Within the hospital setting, mental health professionals can use their consultative and liaison skills to educate the staff of emergency, trauma, and general medical services in the identification and assessment of "silent victims" and in the variety of treatment options and community programs.

Outside of the medical setting, there is a growing need for mental health consultation to criminal justice agencies. Appropriate areas for consultation include

crisis intervention training for law enforcement personnel, with emphasis on strategies to de-escalate violence and to mediate disputes, and prosecutor and court diversion programs that provide victims and offenders with referrals to helping agencies and special treatment facilities (28, 70–72). A newer area for psychiatric consultation and education is the courtroom. Attorneys are increasingly requesting pretrial psychiatric evaluations and consultations in preparing the defense of the battered wife who kills her husband after years of abuse (73). These women usually do not have extensive criminal records, and the homicide often reflects the failure of law enforcement personnel and the courts to protect such women, especially those who are poor and/or minority group members. Psychiatrists can help to educate prosecutors, defense attorneys, judges, and juries about the social and psychological aspects of wife-beating and the context in which a fatality can occur in the absence of insanity or premeditation. Through testimony, they can assist in the determination of a therapeutic rather than a punitive disposition.

The widespread development of shelters for abused women and their children has given rise to another area for mental health consultation. Evaluation services for victims and their children are needed, as are programs that focus on unlearning aggressive behavior patterns and teaching nonviolent models for relationships. Shelters are a singularly important therapeutic and rehabilitative experience for battered women and their children. As Vaughan (56) noted,

> A shelter is not a treatment center, residents are not described as clients, battering is not described as a syndrome. . . . In the operation of the house every attempt is made to avoid labels that stereotype. A woman at the shelter said once, "It belittles you to admit you're a battered woman. I don't like the name. I'd rather be referred to as temporarily abused."

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Natural History of Male Psychological Health: IX. Empirical Evidence for Erikson's Model of the Life Cycle

BY GEORGE E. VAILLANT, M.D., AND EVA MILOFSKY, M.S.

The authors examine Erikson's model of the life cycle by reviewing two 40-year prospective studies, one of 392 men from high-crime core-city neighborhoods and the other of 94 successful college students. The empirically defined developmental stage of each man at age 47 and many other facets of each man's life course were rated by judges blind to other data. The studies identified a well-defined, career consolidation stage that falls between Erikson's stages of intimacy and generativity. The results support three hypotheses. First, the stages of men's life cycle must be passed through sequentially; failure to master one stage usually precludes mastery of subsequent stages. Second, the age at which a given stage is mastered varies enormously. Third, the stage attained by middle life appears quite independent of childhood social class or education, although adult maturation is correlated with whether childhood was conducive to basic trust, autonomy, and initiative.

> It is evident that purely external causes can only give rise to mechanical motion, that is, to changes in scale or quantity, but cannot explain why things differ qualitatively . . . and why one thing changes into another.
>
> —Mao Tse-Tung (1, p. 26)

There is no model for conceptualizing developmental changes over the lifespan that commands consensus. In recent years, however, several studies have suggested that the vicissitudes of adult life reflect more than external perturbations. Because there have been few prospective studies of adult development, models have been theoretical (2) or derived from cross-sectional (3–6) or retrospective studies (7, 8). The most influential of these studies has been Erikson's theoretical model, first described in 1950 in *Childhood and Society* (2). Although this model has had a powerful influence on other work, there have been very few studies (9) in which it has been validated empirically in adults. Investigators working on the two longest lived prospective studies of human development (at the Fels Institute, Yellow Springs, Ohio, and the University of California Institute of Human Development at Berkeley) have chosen to focus on more manageable and more scientifically testable questions rather than models of adult development.

The purpose of this paper is to report early findings from two prospective studies of male development that empirically support and further illuminate Erikson's inductively derived model of adult development. This paper reviews two 40-year prospective studies, one of 392 undereducated men from high-crime inner-city neighborhoods, and the other of 94 highly educated men selected for psychological health during their sophomore year at college.

THE MODIFIED ERIKSONIAN MODEL

The model we used to test Erikson's view of the adult life cycle is portrayed as a spiral in figure 1. (Comprehensive reviews of alternative models of adult development have been provided elsewhere; see references 4, 10, and 11.)

The model is basically the same as Erikson's except for two changes based on recent work. First, certain facets of Erikson's fifth stage, identity versus identity diffusion, have been split off and relabeled stage 6a, career consolidation versus self-absorption. Second, facets of Erikson's seventh stage, generativity versus stagnation, have been recategorized as stage 7a, keepers of the meaning versus rigidity. (The rationale for this is described elsewhere; see reference 12). Because of the wide use of "stage" by Erikson and others, the term has been retained in this paper, but it should be recognized that in adult development the use of

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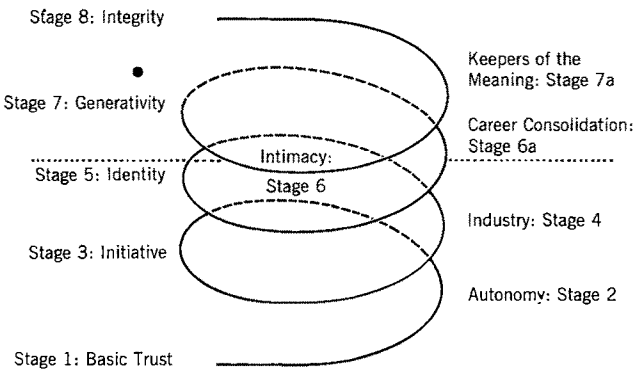
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FIGURE 1
Schematic Diagram of the "Stages" in the Modified Eriksonian Model



"stage" is metaphorical and terms like "developmental task" or "developmental level" would be more correct.

The implicitly sequential nature of this model was summed up by one subject who wrote, "At 20 to 30, I think I learned how to get along with my wife; and at 30 to 40 I learned how to be a success in my job. At 40 to 50 I worried less about myself and more about the children."

In discussing adult development, we will ignore Erikson's first three stages (basic trust, autonomy, and initiative). Prospective study of the sample we will describe did not begin until the men were in junior high school or college, so their struggles to negotiate the earliest Eriksonian stages were not observed. Also, because the tasks faced by adults are so different from a child's, no valid commonsense tests of these stages in adulthood seemed possible.

Stages 4 to 7 in figure 1 were defined in terms relevant to the available data that would also lend themselves to empirical analysis. Admittedly, by concretizing Erikson's elegant (if platonic) language, one loses much of its cultural universality and clinical depth.

It is obvious that cultural differences alter the tasks appropriate to each stage. For example, in cultures that do not provide highly trained women with help in raising small children, the process of career consolidation in women will be hampered. In cultures in which marital bonding is unimportant or postponed until late in life, one would have to devise some measure of intimacy other than marriage. After all, over the ages marrying for love has been the exception, not the rule.

Stage 4 included men who never fully achieved independence from their family of origin or from institutional ties. In middle life such men remained dependent on others and never committed themselves to occupational specialization or sustained friendship. Not all such men came to psychiatric attention, and several had insights into how far removed their lives were from what Erikson called generativity. One man wrote, "I don't have any stress. I don't get that involved. By the same token, I don't have any great happiness."

Stage 5 included men who had mastered the subordinate task of adolescence—social, residential, and economic independence for more than 10 years from the family of origin. Such independence derives from identification and internalization of important childhood figures and is not just a product of egocentricity and running away from home. This subordinate task was substituted for Erikson's term "identity" because his concept defies empirical definition and includes many issues that underlie the later stage of career consolidation (vide infra).

Stage 6, intimacy, included men who had achieved tasks associated with stage 5 and had lived with another person, other than a relative, in an interdependent and intimate fashion for 10 years or more. For almost all of the men this person was a wife. We thought it obvious that Erikson's original formulation of "mutual orgasm" as a criterion for intimacy was far too narrow and would include many sociopaths while excluding some very mature individuals.

In contrast to Piagetian models that focus primarily on cognitive maturation (13, 14), Erikson's psychosocial model reflects the individual's increasing capacity to relate to an ever-expanding life space of people and institutions. Thus in figure 1 the capacity for adult intimacy is represented as the dividing line between childhood and adulthood. We will present evidence that this decision has some scientific legitimacy.

Stage 6a, career consolidation versus self-absorption, included men who, in addition to establishing interdependence with one individual, had made a clear, specialized career identification as measured by occupational satisfaction, commitment, and skill. Career consolidation is often achieved through the fresh internalizations of mentors. It involves the transformation of preoccupation with self, of the child's hobby, and of "seeking the bauble reputation e'en in the cannon's mouth" into a specialized role valued by both self and society. Most summaries of adult development during the fourth decade (8, 15, 16) have focused on the process of career consolidation.

Although the somewhat egocentric passage from apprentice to master craftsman is an important facet of identity, it is rarely achieved until intimacy has been mastered. A vivid, if concrete, illustration is Erik Homburger, erstwhile teacher and therapist to young children, who did not achieve his stable identity of Erik Erikson, teacher to and investigator of young adults, until he had long since established a stable marriage. Over the years Erikson puzzled about the placement of this facet of development (17, 18), and popular writers on female development (19, 20) have pointed out recently that what Erikson called "distantiation" (which others have called "self-actualization") is intrinsic for adult growth beyond intimacy. However,

further psychosocial development may be inhibited to the extent that this process excludes care and leads the adult into self-absorption—the obverse of career consolidation—or "excessive self-love based on a too-strenuously self-made personality" (18).

Stage 7, generativity, included men who, having "found themselves" a second time, demonstrated a clear capacity for "care," "productivity," and "establishing and guiding the next generation" (Erikson's terms). The definition does not mean merely raising crops and small children; it involves assuming sustained responsibility for the growth, well-being, and leadership of others. Depending on the opportunities a society provides, this can mean serving as consultant, guide, mentor, or coach to young adults or to the larger society.

Stage 7a, keepers of the meaning versus rigidity, is tentatively inserted in the model to account for a phenomenon that has appeared consistently in the lives of the college men, but not until they have achieved generativity (12). A 55-year-old subject described the stage succinctly: "Passing on the torch and exposure of civilized values to children has always been of importance to me, but has increased with each ensuing year."

Since most of the sample are not of an age when this phenomenon occurs, stage 7a and Erikson's final stage 8, integrity versus despair, will not be discussed in the empirical part of this paper. In theoretical terms, however, stage 7a removes from Erikson's concept of generativity the development of those characteristics that have less to do with "care" and more to do with "wisdom" and preservation of culture. During this period there is tension between the development of what a culture calls wisdom (21) and the increasing rigidity (22, 23) that reflects changes in intellectual function often seen after middle life.

In adult human development, the relative roles of biology and environment remain as obscure as they are in the evolution of object relations. Obviously, mastery of each stage is a complex process that can be broken down further. Just as Erikson (17) provides the best account of the inner sequences involved in the mastery of identity, the substages of intimacy have been explicated by Goethals and Klos (24) of career consolidation by Levinson (8), and of wisdom by Clayton (21).

THE MODEL AS A SPIRAL

There are several heuristic advantages to depicting the model as a spiral (figure 1). First, this representation underscores that during adult life individuals evolve rather than merely grow and that traits and interests change qualitatively. Second, a spiral conveys the concept that with maturational change, unlike the transformation of caterpillar to butterfly, past progress is not lost. Third, this depiction suggests that as in climbing a spiral staircase, failure to attain one landing makes it unlikely that one will attain the next.

Finally, the left and right sides of the spiral reflect rhythmic changes in malleability of character and intraceptiveness that have been observed, at least in our culture. By clinical impression—if not by hard data—the stages on the left side of the spiral (the "oedipal" years, adolescence, middle life, and if judged by vigorous septuagenarians like Pablo Picasso and William Butler Yeats, old age) are times of change and instability. These are times for reworking both ascribed identity and conventional morality and for experiencing heightened intraceptiveness, affective lability, and fresh instinctual conflict (12, 25). They are periods that provide excellent copy for serious dramatists and popular psychologists. They are also the stages during which a person may seem most like a hysteric.

On the right side of the spiral are represented the four stages of autonomy, latency, career consolidation, and Shakespeare's judge "with eyes severe and beard of formal cut, full of wise saws and modern instances." Clinical impression of individuals during these periods indicates preoccupation with the preservation of sameness and autonomy and with following and maintaining rules. These are periods marked by relatively less intraception. Individuals are more likely to be concerned with being and with absorbing, observing, and promulgating the traditions of their culture than with reworking those traditions. Both dramatist and popular psychologist gloss over these periods, during which an individual might seem most like an obsessional.

A spiral model avoids the issue of transitions. Several investigators have provided empirical data to suggest that the "crisis" aspects of adolescence (26), menopause (27), and retirement (28) may have been overemphasized. Transitional crises may have more to do with individual pathology (e.g., adolescent turmoil), with role changes that are poorly mediated by the culture, (e.g., unplanned retirement or promotion), and with "off time" life changes (e.g., widowhood at 40) than with development per se. The discipline of cultural anthropology (29) may teach us more about transitions than does developmental psychology.

RELATION TO OTHER MODELS OF ADULT DEVELOPMENT

The capacity for generativity may involve a higher order of ego function that is analogous to, but very different from, the capacity for formal operations in Piagetian developmental psychology. If this is the case, it becomes important to examine differences between what Piaget, Loevinger, and Kohlberg call "ego maturation" and what is implied by the Eriksonian model. For Piaget (30) and Kohlberg (13, 31) each new

stage emerges in an invariant sequence and reflects "better" cognition; its development is only modestly dependent on the social environment. Piagetian stages reflect a change in both intellectual capacity and world view and is not affected by commitment. In the Eriksonian model stages are functional, not structural. Preordered biological development plays only a facilitative role at best. One stage is not necessarily "better" than another, and in individual cases the sequence of stages may be altered. These stages reflect affective more than cognitive integration, and performance rather than capacity is most indicative of maturation. Finally, Eriksonian stages are enormously vulnerable to social and interpersonal influences and depend on identification and commitment rather than on perception and cognition.

Perhaps the most intriguing source of discrepancy between the Piaget-Kohlberg model of moral development and the Eriksonian model of psychosocial development is their different emphases on capacity and commitment. An adolescent may be cognitively aware of all the ethical issues of the generative individual while lacking the capacity to translate such understanding into sustained behavior (13).

Erikson's platonic term "care" includes the capacity for empathy and the willingness to accept responsibility for others while respecting their autonomy. As such it is analogous to Loevinger's mature "autonomous stage" of ego development (4), so named because the person at this point recognizes the other person's need for autonomy. In Erikson's model the maturing individual becomes involved in increasingly complex social matrices. For Kohlberg and Loevinger maturity is "the process of reappropriating reality at progressively more complex levels of apprehension as the structures of thinking expand to encompass the increasing richness and intricacies of experience" (32).

So far, so good. However, to characterize maturity by Erikson's "virtue" of care runs counter to the view that maturity is the capacity to be one's own self-actualizing man or woman. Thus, Loevinger (4) and Kohlberg (13) have both proposed a maturational model characterized by an increasing differentiation of self from others and a progressive freeing of thought from contextual (and social) constraint. However, Kohlberg and Loevinger have derived their findings from pencil and paper tests, an essentially impersonal context in which the hallmark of cognitive maturity becomes, as it does for Piaget, the capacity to divorce intellectual principle from context. In challenging Kohlberg's conclusion that moral development tends to be more advanced in men than women because in women morality remains bound to social context, Gilligan (32) substituted for Kohlberg's hypothetical moral dilemmas a real-life test to abort a pregnancy. She, like Erikson, concludes "care then becomes a universal obligation, the self-chosen ethic of a post-conventional (mature) judgment."

To put it another way, at the heart of the adolescent's quest for identity and the young adult's quest for career consolidation is the ethic of responsibility to self and separation of one's own efforts at self-determination from the seeming constraints of intimacy and cloying interdependence. But the pendulum swings to and fro. The capacity for intimate interdependence comes after the adolescent has pulled free from dependence on the family of origin. Similarly, not until the young adult has consolidated his own career identity is he willing or able once again to subordinate his autonomy to responsibility for others. Indeed, Klerman and Levinson (33) suggest that the good administrator must first come to terms with the quest for autonomy of his or her adolescent children.

EMPIRICAL TESTS

Obviously, the model depicted in figure 1 is only heuristic. The development of human personality and behavior is too plastic, complex, and discontinuous to be represented adequately by any model. The purpose of this model is to provide a framework for examining Erikson's ideas in the context of empirically derived data. This paper will make three tests of the model.

1. If the model of adult development is conceived as open-ended, people will reach a given stage at different ages.
2. If the model is to be useful, it should be relatively independent of social class and education.
3. If the model is truly developmental, its stages should be sequential. It is not surprising that in our culture men are unlikely to achieve stable marriages until they have separated themselves from their parents and are self-supporting. However, the supposition in the model that mastery of intimacy is a prerequisite of career specialization, which in turn is a prerequisite of the capacity to care, would seem to contradict modern folk wisdom that career success interferes with both intimacy and social responsibility.

The paper will also address a fourth issue: What relation, if any, does this model of psychosocial development have to ego maturation as reflected by choice of defense mechanisms?

THE STUDIES

Method

The college sample consisted of 94 sophomore men selected between 1940 and 1942 for a study of psychological and physical health at a Northeastern liberal arts university (12, 34). The men were studied in detail in college and have been followed prospectively since then with biennial questionnaires. The men were reinterviewed at age 30 (circa 1951) and at age 47 (circa 1968).

The core-city sample consisted of 392 inner-city youths chosen by the Gluecks (35) as control subjects in their prospective study of juvenile delinquents. Between 1940 and 1945 these men were selected in junior high school as nondelinquent. They were matched with severely delinquent youths in terms of residence in high-crime neighborhoods, ethnicity, and intelligence. At that time these youths and their families were studied intensively. The sample was reinterviewed at ages 25 (circa 1955), 32 (circa 1962), and 47 (circa 1977).

Early life ratings. Clinicians blind to all post-adolescence information rated the men on the basis of a search of social service records and interviews with the subject, his parents, and his teacher. Raters used the scales listed below:

1. Childhood environmental strengths. This 20-point scale, described in detail elsewhere (36), rates the men by a clinical judgment of childhood environmental strengths. Points were assigned for the absence of childhood problems with physical, social, and mental health and for the presence of parental relationships and home atmospheres conducive to the "development of basic trust, autonomy and initiative." Ratings were obtained for both core-city and college samples. Interrater reliabilities (3 raters) ranged from .70 to .89.

2. Childhood environmental weaknesses. This is a 50-point scale based on 25 concrete criteria that encompasses the Gluecks' more subjectively defined delinquency prediction scale (35). The scale includes 5 items reflecting gross lack of family cohesion (e.g., 9 or more social agency contacts, raised for more than 6 months apart from both parents); 10 items reflecting lack of maternal affection and supervision (e.g., mother delinquent, mother mentally retarded, mother failed to provide supervision when absent from home, multiple observers described maternal affection as inadequate); and 10 items reflecting inadequate paternal supervision and affection (e.g., father alcoholic, delinquent, or cruel to boy, multiple observers described paternal affection as inadequate). The scale was used only for the core-city sample. Interrater reliability was .91 to .94.

3. IQ. Each core-city subject was given the Wechsler-Bellevue Intelligence Test upon entrance to the study. College men received the Army Alpha Scale and the Scholastic Aptitude Test.

4. Stage 4 tasks. This 8-point scale assessed the extent to which the core-city subjects at entrance into the study had mastered Erikson's fourth stage of industry—"doing things beside and with others." The boys received points for doing regular home chores (1 point), adjusting well to school academically (2 points, controlled for IQ) and socially (1 point), participating in extracurricular jobs (1 point) and recreation (1 point), and making the best of problems at home (2 points). Interrater reliability was .70 to .91.

Adulthood ratings. Clinicians blind to all information collected before subjects reached age 30 rated the men, primarily on the basis of the recent semistructured 2-hour interview. They used the following six scales:

1. Adult life stage. This rating was made on a 5-point scale based on the Eriksonian model (1=less than stage 5; 2=stage 5; 3=stage 6; 4=stage 6a; 5=stage 7). In the clinical assignment of a 47-year-old subject to a given stage, raters adhered to the spirit, not the letter, of Erikson's model in that the exceptional circumstances of a man's full life were taken into account. For example, if a man now functioned at a lower stage than previously, because of alcoholism or physical illness, he was assigned to the highest stage achieved. Similarly, a deeply committed Catholic priest who became a successful parochial high school principal was rated as generative, although he had never married. For the college sample this judgment was made on the basis of the write-up of a 2-hour interview with the men at age 47 that was conducted by a clinician blind to all other ratings (interrater reliability=.61). For the core-city sample, the rating was the consensus judgment of two clinicians who were familiar with all data about subjects after age 30 (interrater reliability not determined).

2. Health-sickness rating scale (HSRS). This scale is described in detail elsewhere (37, 38). The raters used 34 case illustrations as guides to place each subject on a 100-point continuum that ranged from total institutional dependency (rating of 0–25) to no psychological dysfunctions and multiple manifestations of positive mental health (rating of 90–100). Interrater reliability for this scale was .89.

3. Object relations. For the core-city sample, this was a 25-point scale summing each individual's relative success in accomplishing eight different tasks of adult object relations *excluding* marriage: one task each reflected enjoyment of children, family of origin, and workmates; three tasks reflected friendship network; and two tasks reflected participation in group activities. A similar 6-point scale for the college sample is described elsewhere (34).

4. Maturity of defenses. This 9-point scale was based, for the college sample, on ratings of coping behavior over subjects' adult lives from ages 20 to 35 (12, 39). For the core-city sample, the assessment was based only on a 2-hour interview at age 47. To quantify clinical impressions, the following procedure was observed: 1–5 points were assigned for the tendency to deploy each of three groups of defenses—mature (sublimation, suppression, anticipation, altruism, and humor), neurotic or intermediate (displacement, repression, isolation, obsession, and reaction formation), and immature (projection, schizoid fantasy, masochism, acting-out, hypochondriasis, and neurotic denial/dissociation). It was required that the three ratings sum to 8. Relative maturity of overall defensive style was estimated by subtracting the rating for immature

TABLE 1
Attrition in Two Studies of Male Psychological Health

Status	Sample: Core City N	Core City %	College N	College %
Original sample[a]	456	100	102	100
Available for life stage rating	392	86	94	92
Dead prior to life stage rating	30	7	6	6
Alive but withdrew from study before interview	19	4	2	2
Alive but insufficient data for rating	15	3	0	

[a]For the core-city sample this number was reduced from 500 by the Gluecks' decision not to reinterview men with birthdays after July 1932; for the college sample, the number was reduced from 268 by our decision to reinterview a randomly selected 50% of the subjects from the college classes (1942–1944). Subjects from prior classes had been studied less uniformly. Neither procedure should introduce bias.

TABLE 2
Comparison of Men Given Life Stage Ratings

Variable	Sample: Core City (N=392)	College (N=94)
Parents attended high school (%)	33	94
Parents in social classes I–III (%)	10	93
Age when childhood assessed (years)		
Mean	14	19
SD	2	1
Year of birth		
Mean	1929	1921
SD	2	1
Score on childhood strengths scale (units)		
Mean	9.2	9.4
SD	4.8	4.6
IQ		
Mean	95	~125–140
SD	12	
High school graduate (%)	48	100
Attended graduate school (%)	1	76
Social class I or II at age 46 (%)	9	98
HSRS score less than 70 (%)[a]	28	20

[a]Scores below 70 indicate psychopathology.

defenses (rating of 1–5) from the rating for mature defenses (rating of 1–5), giving a 9-point range.

5. Social class. This 5-point classification, devised by Hollingshead and Redlich (40), assigns weights to education, residence, and occupation (I=managers and professionals with a college degree and living in a good suburb; V=unskilled laborers with less than a high school education living in a single room). The rating was assigned when the men were 47 years old.

6. Education. This was a 7-point scale (1=some graduate school, 2=college graduate, 6=7–9 years of schooling, 7=did not reach 7th grade).

Results

Attrition and sample characteristics. Although 3 decades had elapsed between the time the men were first studied in adolescence and their interviews in middle life, attrition was modest and unlikely to affect the findings seriously. As table 1 illustrates, enough data were obtained on 92% of the original college sample and 86% of the original core-city sample to assign them adult life stage ratings at age 47.

TABLE 3
Adult Life Stage at Age 47

Adult Life Stage	Sample: College (N=94) N	College %	Core City (N=392) N	Core City %
Never fully self-supporting or still not living independent of institutional setting or family of origin (stage 4)	0		32	8
Identity: self-supporting, independent of family, but unable to achieve intimacy (stage 5)	11	12	38	10
Intimacy: long-lasting interdependent relationship but no stable career specialization and skill (stage 6)	13	14	75	19
Career consolidation: stable career specialization but little responsibility for others (stage 6a)	31	33	126	32
Generativity: clear responsibility for others (stage 7)	39	41	121	31

Table 2 underscores the fact that the two samples were skewed toward opposite ends of the socioeconomic spectrum and had enormous differences in education and social opportunity. In terms of childhood strengths, the two samples received almost identical scores. However, the average core-city youth was much more likely to have experienced a disorganized multiproblem family and/or at least one grossly neglectful or abusive parent. For the college sample, the 25 items in the childhood weakness scale were rarely applicable. Although the college sample was selected for mental health at age 18, at age 47 there was only slightly more psychopathology in the core-city sample, as reflected by HSRS scores.

Empirical test of the first two hypotheses. The data in table 3 support our first hypothesis, i.e., adult life stages were relatively independent of chronological age. Only 30%–40% of these middle-aged men could be called generative, and in many respects 1 man in 6 was still struggling with adolescent issues.

The results shown in tables 3 and 4 support the second hypothesis, i.e., social class and education were only weakly associated with and had an even weaker causal relationship to adult maturation as defined by this model. The small difference between the core-city and the college sample in adult life stage probably can be accounted for by the fact that the college sample had been preselected in late adolescence for mental health and maturity.

Table 4 suggests that for the core-city men the best predictors of maturity (11% of the variance explained) were the presence of environmental strengths of childhood, success at stage 4 tasks (at age 14), and educational attainment. When scores for childhood

TABLE 4
Selected Psychosocial Variables in Core-City Men (percents)

Variable	Adult Life Stage (Age 47)				
	Failed at Intimacy (N=70)	Intimacy (N=75)	Career Consolidation (N=126)	Generativity (N=121)	Strength of Association[a]
Important adolescent antecedents					
High stage 4 task rating at age 14 (±2)	13	23	24	37	.27
Absence of childhood environmental strengths (lowest quarter)	50	25	21	20	.24
Not a high school graduate	66	56	53	36	.26
Less important adolescent antecedents					
IQ in top third (>100)	23	28	26	44	.17
IQ in bottom third (<90)	43	23	30	25	
Multiple childhood weaknesses (lowest tenth)	20	11	10	10	.11
Important correlates at age 47					
Uses predominantly "immature" defenses	76[b]	55	30	8	.63
Ever assigned a psychiatric diagnosis	71[b]	31	18	14	.47
Social class IV or V	87	72	40	19	.54
HSRS score less than 61	57[b]	11	0	1	.78
HSRS greater than 80	2	6	29	80	
Subjectively content	32[b]	48	58	88	.47

[a]Pearson product-moment correlation coefficients of adult life stage rating, with psychosocial variable treated as a continuous rather than a dichotomous variable. Correlations greater than .17 are significant ($p<.001$).
[b]Several (5%–20%) subjects could not be rated on this variable. Therefore, the N is divided by a denominator less than 70.

TABLE 5
Variables Related to Intimacy in Core-City Men (percents)

Variable	Adult Life Stage (Age 47)				
	Failed at Intimacy (N=70)	Intimacy (N=75)	Career Consolidation (N=126)	Generativity (N=121)	Strength of Association[a]
Object relations score in bottom fifth	66	30	17	3	.59
No children or not economically responsible for those sired	86	28	19	8	.36
Income less than $10,000/year (bottom fifth)	57	25	8	3	.53
Married less than 10 years	73	12	4	4	.62
Married 20 years or more	16	68	75	86	

[a]Pearson product-moment correlation coefficients of adult life stage rating, with psychosocial variable treated as a continuous rather than a dichotomous variable. Correlations greater than .17 are significant ($p<.001$).

strengths and stage 4 tasks were entered first into a multiple-regression analysis, IQ and childhood weaknesses made no further contribution to the explained attained variance in adult life stage (8%). In such an analysis, amount of education (seventh grade to graduate school) explained only 3% additional variance. The number of relatives with mental illness, alcoholism, delinquency, and mental retardation made no independent contribution. It was both astonishing and encouraging that traumatic events as measured by the childhood weakness scale were so weakly correlated with subsequent adult development.

Table 4 also shows that most of the core-city men who failed to achieve intimacy by age 47 had at some time been called mentally ill, continued to deploy defensive styles associated with character disorder (and adolescence), and had received HSRS scores indicative of pathology.

Correlates of individual adult life stages for core-city men. Table 5 presents the variables that best distinguished the men who achieved intimacy from those who did not. Although a successful marriage served as the best empirical gauge of a clinical rating of intimacy, some men who were judged to have attained stage 4 or 5 had remained married for decades to women for whom they had no respect or affection and had sired children they failed to support. The more important finding conveyed in table 5 is that (in this culture for men born between 1925 and 1935) failure to marry was a predictor of serious impairment in subsequent object relations and occupational achievement. This observation lends support to the concept that attaining intimacy with one person reflects an important task of adult maturation. It also supports the logic of considering the mastery of intimacy a precondition for career consolidation. The men who mastered career consolidation showed even more stable marriages and greater responsibility for children than the men who had mastered intimacy but had not progressed beyond it.

Table 6 presents the variables that best distinguished the men who achieved career consolidation from those who did not. Not surprisingly, the clinical judgment of career consolidation tended to identify upwardly mobile men with highly skilled, well-paid jobs

TABLE 6
Variables Related to Career Consolidation in Core-City Men (percents)

Variable	Adult Life Stage (Age 47)				
	Failed at Intimacy (N=70)	Intimacy (N=75)	Career Consolidation (N=126)	Generativity (N=121)	Strength of Association[a]
Incapacity to channel anger into work or constructive activity	90[b]	80	49	14	.55
Four or more years of unemployment	59	27	10	4	.51
Chronic unemployment or four or more job changes in past decade	49	29	12	10	.38
Little enjoyment of job	78[b]	86	50	29	.44
Social class I-III	13	28	60	81	.54
10 years with the same firm	22	29	49	54	.40
Could identify a "mentor"	4[b]	5	21	34	.27
Professional, technician, or skilled craftsman	11	17	47	41	n.a.
Income $15,000 or more (top half)	12	33	66	79	.53
Steady promotions	13	12	30	51	.42

[a]Pearson product-moment correlation coefficients of adult life stage rating, with psychosocial variable treated as a continuous rather than a dichotomous variable. Correlations greater than .17 are significant ($p<.001$).
[b]Several (5%–20%) subjects could not be rated on this variable. Therefore, the N is divided by a denominator less than 70.

TABLE 7
Variables Related to Generativity in Core-City Men (percents)

Variable	Adult Life Stage (Age 47)				
	Failed at Intimacy (N=70)	Intimacy (N=75)	Career Consolidation (N=126)	Generativity (N=121)	Strength of Association[a]
Still enjoys first marriage	1	37	46	78	n.a.
Object relations in top two-fifths	7[b]	26	36	72	.59
Regular participation in public service	7	13	14	54	.39
Enjoys his children	4[b]	39	51	75	.45
Uses predominantly "mature" defenses	2[b]	10	19	61	.63
Manager or administrator	6	8	10	28	n.a.

[a]Pearson product-moment correlation coefficients of adult life stage rating, with psychosocial variable treated as a continuous rather than a dichotomous variable.
[b]Several (5%–20%) subjects with adult life stage ratings could not be rated on this variable. Therefore, the N is divided by a denominator less than 70.

who tended to remain with the same employer. Such men were also far more likely to enjoy their jobs and to identify a mentor who had affected their occupational lives. Although the entry of the core-city men into the labor force was not delayed by graduate school, career consolidation and the acquisition of mentors usually did not occur until these subjects were in their late 20s or early 30s.

A quite unanticipated and interesting finding was that the men who mastered career consolidation were far more likely to report constructive means of channeling their anger into work or play. Men who were still at stage 5 or 6 denied or avoided expression of anger or resorted to temper outbursts, projection, or binge drinking. (In determining this rating, belligerence directly related to alcoholism was excluded.)

Table 7 lists the criteria that best differentiated core-city generative versus nongenerative men. Since the overriding factor governing the clinical decision to place a man in this stage (stage 7) was his assumption of responsibility for other adults, it was not surprising that managers and administrators tended to be classified as generative. Of much greater interest was the variety of criteria reflecting caring, moral, or mature behaviors that distinguished generative men. The strong association of generativity with social service remained when income was held constant.

Empirical tests of the third hypothesis. In table 8 we have attempted to summarize supporting data for Erikson's contention that the developmental tasks of adult life must be mastered sequentially. The table examines the men clinically assigned to each stage in terms of their likelihood of having mastered objective tasks relevant to every other stage. We found that only 14 of the 70 men whom clinicians rated as unable to achieve intimacy with one person ever made a stable job commitment, and only 2 mastered the tasks associated with generativity. Conversely, almost all of the 121 men who were called generative on the basis of being managers or caring for others had also mastered most of the arbitrary tasks reflecting stable career and stable marriage. Most convincing, perhaps, was the fact that 94% of the 126 men who were judged to have consolidated careers had also mastered—almost always at an earlier period in their lives—the arbitrary task of staying married (in spirit as well as in letter) to another

TABLE 8
Core-City Men's Mastery of Objective Tasks Reflecting Clinical Assessment of Adult Life Stage

	Adult Life Stage (Age 47)							
	Failed at Intimacy (N=70)		Intimacy (N=75)		Career Consolidation (N=126)		Generativity (N=121)	
Tasks Achieved	N	%	N	%	N	%	N	%
Two or more stage 6[a]	12	17	69	92	119	94	116	9(
Three or more stage 6a[b]	14	20	24	32	86	68	107	8(
Two or more stage 7[c]	2	3	11	15	28	22	85	7(

[a]Tasks were 1) married 1/3 of adult life, 2) object relations not in bottom fifth, 3) financially responsible for any children sired.
[b]Tasks were 1) not more than 1 year of unemployment, 2) likes his job, 3) steady job improvement over past 2 decades, 4) same employer for 10 years, 5) has n(had four or more jobs or been chronically unemployed in last decade.
[c]Tasks were 1) enjoys adolescent children, 2) participates in volunteer activities, 3) object relations in top two-fifths.

TABLE 9
Percent of College Men Manifesting Variables Theoretically Related to Psychosocial Maturity

	Adult Life Stage (Age 47)			
Variable	Failed at Intimacy (N=11)	Intimacy or Career Consolidation (N=44)	Generativity (N=39)	Strength of Association[a]
Ego function				
Predominant use of immature defenses (age 20–35)	56	7	0	.50
Predominant use of mature defenses (age 20–25)	18	57	64	
HSRS score less than 60 (age 47)	36	2	0	.73
HSRS score above 80 (age 47)	0	11	71	
Psychosocial maturity				
Many childhood environmental strengths (age 1–18)	27	55	49	.11
Many vignettes of self-doubt, pessimism, dependence, and passivity (age 20–47)	73	18	5	.46
Many vignettes of aggressive behavior (age 20–47)	0	23	33	.31
Dominated by mother in adult life (age 20–30)	64	14	3	n.a.
Continued enjoyment of first marriage (age 47)	0	23	41	.44
Significant voluntary participation in public service (age 47)	27	43	62	.25
Subjective happiness (age 47)	0	36	41	.45
Close to his children during their adolescence (age 55)	9	68	85	.41

[a]Pearson product-moment correlation coefficients of adult life stage rating, with the psychosocial variable treated as continuous rather than dichotomous Correlations greater than .24 are significant ($p<.01$).

person for a long period of time. When we examined the exceptions in the table, we found many could be easily explained. For example, a man who was generative at age 35 became subsequently chronically unemployed because of illness, and a stage 5 man, terrified of going to jail for not paying alimony, preserved an agonizing marriage for 20 years by sleeping in a separate bedroom.

Defensive style and adult life stages (college men). Evidence has been reported elsewhere that as they grew older, the college sample used progressively more mature defenses (12, 39). Tables 4 and 7 suggest that for the core-city sample, maturity of defenses was strongly associated with adult life stage. There was a strong association between immature defenses (e.g., projection, passive aggression, and acting out) and remaining at stage 5, and between mature defenses (e.g., altruism, suppression, anticipation, and sublimation) and generativity. Since for the core-city men ratings of defensive style and adult life stage were not always made by independent raters, we have provided a more rigorous test of this association in table 9. Ratings of the college men's defensive style before age 36 were made by a rater blind to their Eriksonian rating who used the same rating scheme used for the core-city men. Ratings of adult life stage were made by a rater provided only with data from the semistructured interview conducted when subjects were 47 years old (12). In the college men maturity of defenses rated before age 35 correlated with adult life stage at age 47 ($r=.50$).

The contrast of the stage 5 with stage 7 college men (table 9) is very similar to the contrast of the core-city men in table 7. In more general terms, although the number of college men who failed to achieve intimacy was too small for extensive analysis (N=11), it was striking that the blindly assessed childhoods of the stage 5 men were less conducive to developing basic trust, autonomy, and initiative than were those of the

39 generative men. In adult life, as predicted by Erikson's model, the stage 5 men manifested more independently identified vignettes suggestive of basic mistrust and many fewer vignettes of initiative. Finally, as young adults they had not freed themselves from maternal domination.

DISCUSSION: THE STUDIES AND THE MODEL

Since the concept of stages of adult development is at best an oversimplification and at worst merely a heuristic device, several limitations must be acknowledged.

First, it is no accident that the prospective study of human development began with measurements of the most concrete variables (height and weight in children) and proceeded to other relatively objective phenomena, such as epiphyseal closure, puberty, and dentition. Only later did investigators study simple intellectual development; only now are they charting in children the more abstract processes of cognitive, moral, and ego development. The science of adult development is in the earliest stage. Like our concepts of humor, creativity, and schizophrenia, precise definition is impossible and indirect measurement is the best that we can hope for at this point. Thus, the ratings of adult life stage share the unreliability inherent in clinical diagnosis. A logical next step will be to refine definitions and increase interrater reliability.

Second, developmental stages are a biological concept most relevant to embryology. The concept of biological development remains relevant for children because the brain continues to develop in a preordered fashion over the first two decades of life, and the genital anlage continue to develop over the second decade. In postpubertal humans only a few further developmental changes in the central nervous system can be identified. Myelinization in the cortical areas of association may well progress into the fifth decade (41). As adults age, there are predictable changes in stage IV and REM sleep (42) and in slow waves in the EEG (43). At puberty and menopause predictable shifts occur in sex hormones that are known to affect cerebral function. Over the adult life span the symptoms of drug-induced parkinsonism change as does MAO concentration in the globus pallidum (44). Finally, between age 20 and age 50 measures of "crystallized" intelligence may improve and "fluid" intelligence may decline, producing predictable, if imperfectly measured, changes in cognition (22). However, there is no evidence that these endogenous changes play a greater role than culture or human relationships in determining adult development.

The more that sustained social and historical events impinge on the life of an individual, the more complex, heterogeneous, and less predictable that individual's development becomes (45). In extreme cases, culture and early childhood environment can override even the clearest examples of biological preordering. For example, in the Borana Galla tribe of Africa, a child may be retired or made an elder at birth (46). Also, as Money and Ehrhardt (47) have demonstrated, in almost every facet of adult sexual behavior the gender in which a child is reared can override his or her genotype.

Third, the Eriksonian model is culture-bound. For the model to be relevant, many variables in the environment must be held constant. The culture must be one in which individualism is highly regarded and the social order is relatively unpatterned by arranged marriage, prejudice, and economic necessity. In instances when caste and culturally dictated roles govern individual development, the model becomes less relevant (48). The best historical confirmation of this model is found in individualistic, economically favored societies, e.g., Shakespeare's description in *As You Like It* of the ages of man in Renaissance Italy (or Elizabethan England) and the Wisdom of Solon describing Greece in the 7th century B.C. (8). The model can be applied easily to women only when the culture has given them the same prerogatives as men (49).

In view of these limitations, the term "developmental task" (50) becomes more appropriate than "developmental stage." Thus in assessing adult development we have described certain tasks, the mastery of which correlated with, and perhaps facilitated, mastery of subsequent and theoretically more mature tasks. However, it is obvious that these tasks convey only the crudest approximation of adult development. Life is not painted in primary colors, nor is it mastered by task completion. Measuring intimacy by the length of an adult's married life is as preposterous as measuring a two-year-old's struggle for autonomy by his frequency of saying "no." Adult development consists of emerging capacities and commitments, and defining developmental level by a laundry list of divergent tasks rather than a psychometrically refined instrument or by precise clinical signs and symptoms is less than ideal. Alternative strategies, however, are limited. The clumsiness of diagnosis by behavior is well recognized, but after 50 years of trying to measure mental illness clinically and psychometrically we now have *DSM-III*, wherein selected behaviors are substituted for test scores and clinical judgment. A logical next step must be to chart by prospective study the sequential emergence of such tasks in the lives of men and women.

CONCLUSIONS

We have attempted to determine whether statistical ordering of prospectively gathered concrete data about human behavior over the adult life span confirms Erikson's model of adult development.

Our findings support the hypothesis that adult matu-

ration is open-ended. The development of psychosocial maturity is no more a foregone conclusion than is the development of Piagetian formal operations. In contrast to Levinson's retrospective investigation (8), we found no fixed chronological age at which interpersonal or occupational competence was established. Just as some adults never achieve postpubertal interest in the opposite sex, progression through the stages of adult development outlined in this paper can halt at any point. In both instances, environment, culture, or biology each can be a necessary and sufficient cause.

There is also the distinct possibility that the innate capacity for object relations varies on a continuum, as does musical talent or the capacity to do abstract mathematics (39), a hypothesis consistent with the concept of the schizophrenic diathesis. Indeed, schizophrenia—which spares all the developmental functions associated with Piagetian cognitive maturation and interferes with all those associated with psychosocial maturation—provides an arena in which to begin teasing apart Eriksonian and Piagetian concepts of maturation.

Our second conclusion is that adult development is not enormously influenced by education and privilege and that age-graded influences diminish as development unfolds (45). A common fear is that fools can become leaders by virtue of IQ, upper-class family background, and advanced educational opportunities—and that "poor-testing" blue-collar high school dropouts are doomed to assembly lines and anomie. Long-term follow-up demonstrates that these patterns do not always hold. Of course, entering adult life as a white American male in the 1940–1960 economic climate facilitated the chance for unfettered emotional growth in both samples. That culture can override endogenous developmental change has already been conceded; the point is simply that Erikson's model may be more socially egalitarian than is sometimes assumed.

The third empirical outcome of the study is support for Erikson's extraordinary generalization, "The human personality, in principle, develops according to steps predetermined in the growing person's readiness to be driven toward, to be aware of, and to interact with a widening social radius" (2, p. 270). In both the core-city and the college men the correlates of generativity were closeness to adolescent children, assumption of managerial responsibilities, enjoyment of one's first marriage, voluntary public service, and use of mature ego mechanisms like suppression, altruism, and anticipation. In harvesting data from the Berkeley studies of human development, Haan (25) and Block (51) both concluded that their subjects at middle age when compared to themselves at age 30 had become more sympathetic and productive, "better able and more willing to interact with others, generously and dependably." In viewing deans, politicians, and business magnates as the products of crass ambition and infantile narcissism, we ignore the ego skills that an individual must have in order to assume responsibility for groups of other adults.

The final conclusion derived from our empirical tests of Erikson's developmental model concerns ego maturation and renders our findings more analogous to those of Kohlberg and Loevinger. The fact that the adaptive style was so highly correlated with adult life stage suggests that maturation of a broader range of ego functions may be involved rather than just capacity for object relations. In an earlier study it was found that the use of immature defenses by the college men decreased with the passage of time (12). In this study when chronological age was held constant (ages 20–35 for the college men and age 47 for the core-city men), immature defenses decreased as adult life stage advanced. Mature defenses were rare in the core-city men until they mastered the tasks related to generativity. Among the 47-year-old college men, predominantly mature defenses were used by none of the stage 5 men, 31% of stage 6 men, and 67% of stage 7 men.

The key to understanding the sequential nature of adult development may lie in appreciating both the relative complexity and the inner threat of the tasks and commitments to be mastered. Twin anxieties of young adulthood are: How can I commit myself to one person or to one job without sacrificing autonomy? Older adults may watch this struggle with the same benevolent familiarity felt by young adults watching 13-year-olds wrestle with their apprehensions about the opposite sex. The developmental processes that resolve such anxieties will not be as predictable as those of embryology or as easily studied in the laboratory as those for cognition, but it is hard to conceptualize such phenomena in other than developmental terms. At the same time, there will always be exceptions to a fixed developmental sequence. Individuals with a strong religious or artistic orientation are especially likely to exhibit atypical development.

Two final questions cry out for answers. First, how does the model described in this paper pertain to women? Second, what is the distinction, if any, between maturity and mental health? Elaboration of either question would require a separate paper and fresh data.

In order to wrestle Erikson's ineffable concepts of adult growth and development into the computer we employed the strategy of viewing development as a set of task completions. However, biology is *process*, not *task completion*. Intimacy is not achieved once and for all, and the seeds of love must be eternally resown. Career consolidation without continued productivity leads to rigidity and stagnation. More than any other task, care reflects process—process that must be relinquished eventually, even as the distantiation and renunciation of advancing age become linked with the capacity for wisdom. Therefore, it seems appropriate to close with Erikson's warning,

The assumption that on each stage a goodness is achieved which is impervious to new inner conflicts and to changing conditions is, I believe, a projection . . . of that success ideology which can so dangerously pervade our private and public daydreams and can make us inept in heightened struggle for a meaningful existence continuously, even as the body's metabolism copes with decay. (2, p. 274)

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The Psychological Organization of Depression

BY SILVANO ARIETI, M.D., AND JULES R. BEMPORAD, M.D.

On the basis of the intensive psychotherapy of about 40 depressed patients over the course of two decades, the authors describe three premorbid types of depressive personality: 1) one based on a "dominant other" relationship, 2) one based on a "dominant goal," and 3) one that is a form of character structure or personality disorder. They also describe typical childhood experiences of depressive adults and discuss their theory of the nature of depression as a human experience. They characterize depression as a limitation of alternate ways of thinking and as self-inhibition from new experiences.

A large majority of current publications on depression have emphasized only the biological approach to this syndrome. Although appreciative of the contribution of biochemical hypotheses to the etiology of depression and the efficacy of organic treatments on some aspects of the depressive syndrome, we hold the view that psychological mechanisms, which have recently suffered neglect, remain important to the understanding and treatment of affective disorders. In this paper we will attempt to 1) propose a typology of different forms of depression encountered in daily practice, 2) suggest psychodynamic formulations regarding the development of depression as well as some childhood events that appear to predispose to depression in adult life, and 3) describe a psychological theory of the nature of depression as a human experience.

We have based our findings on the intensive psychotherapy of about 40 depressed patients over the course of the past two decades. We realize that our conclusions may be the result of subjective impressions because we have not used objective criteria scales or blind raters, which would have led to more rigorous data. On the other hand, we firmly believe that the knowledge gained through intensive therapeutic contact with an individual over the course of years is difficult to duplicate through more objective methods. Leff and associates (1) came to similar conclusions, stating that only after an extensive period of therapy could the depressed patients whom they studied be understood fully and that initial interviewing did not reveal the core conflicts or precipitating events involved. We have also not reported a control group because we believe that our nondepressed patients served to differentiate those elements found in depressive patients from those found in individuals who had other complaints and diagnoses.

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PREMORBID TYPES OF DEPRESSIVE PERSONALITY

Depression occurs clinically in a variety of situations and in a variety of individuals. However, despite superficial heterogeneity, a limited number of basic underlying patterns emerge from the myriad instances of depression encountered in clinical practice. As has been pointed out by Rado (2), Jacobson (3), and others, the vulnerability to acute episodes of depression appears to be a result of the individual's relying to a dangerous extent on external supports to maintain self-esteem as well as avenues of meaning and gratification. Predisposed individuals have evolved patterns of living and interpersonal modes that require for optimal functioning a continued relationship with an esteemed other, a specific role in an organization, or the pursuit of a dominant goal. During psychotherapy it is not difficult to discover that these patterns of living and interpersonal modes are based on preexisting ideology or cognitive belief systems, mostly unconscious, that have been gradually organized in the patient's life (4, 5).

These patterns culminate in a needed sense of self that the depressive person acutely requires for maintenance of well-being. When this sense of self is threatened by the irrevocable loss of another person who had pathologically supplied self-esteem, or when the individual realizes that some desired goal on which this sense of self was constructed is hopelessly unattainable, clinical depression often ensues.

The first form of predisposition to depression is based on a "dominant other" type of ideology and relationship. In individuals belonging to this group, an esteemed other is relied on to bestow meaning, allow gratification, and maintain self-esteem (6). They do not

experience satisfaction directly from effort but only through an intermediary, who either gives or withholds rewards. They have formed an imagined agreement with the important other that may be called a "bargain relationship" (7), in which the individual forgoes the independent derivation of gratification in return for the continued nurturance and support of the esteemed other. This pattern of relating was initiated by the parent during the childhood of the predepressive individual, but in later life the individual will reinstitute similar relationships in a transferential manner. Other characteristics of this type of depressive personality are clingingness, passivity, manipulativeness, and avoidance of anger. These character traits may be seen as means by which the individual attempts to extract support from the needed other as well as to ensure continuation of the relationship. An example of this type of depressive personality is Ms. A.

Case 1. Ms. A was a 28-year-old nurse who came for treatment with a moderate depressive reaction following her husband's disclosure that he had found another woman and was considering separating from her. Ms. A had been married for seven years and had been working at a job she disliked (often working nights) to put her husband through school and to earn enough for a down payment on a home. Despite her obvious self-sacrifice on her spouse's behalf, she was not angry at him but blamed herself for his infidelity, stating that her working nights had caused him to become lonely.

During the course of therapy, it became clear that she had evolved a lifestyle of diligent work and obedience to obtain praise and support from her husband. This pattern of relating to others had been established in childhood, when she assumed a caretaker role for her younger siblings in the hope of obtaining the love of her mother, which she never felt she really accomplished. As hard as she tried, her mother demanded more and rarely gave praise. Her father was a withdrawn, disillusioned man who seemed to have been psychologically absent from the family. In her teens, the patient learned that her mother's pregnancy with the patient was unexpected and forced the father to quit college and settle into a life that did not meet his aspirations. It seemed that both parents resented her presence because of her untimely birth. They never praised her creative endeavors or appearance but appeared to acknowledge only the help she provided at home. She was sent to a girl's parochial school that reinforced her family's work ethic and limited her contact with boys. In her teens she spontaneously took up the guitar and showed such talent that she was asked to play in coffee houses. Her father immediately put an end to this activity, claiming it was sinful and frivolous.

Contact with boys was infrequent and plagued with guilt and insecurity. Shortly after graduating from high school Ms. A married Mr. A, an older man whom she perceived as worldly, sophisticated, and generally superior to her. During their marriage she developed essentially no extramarital interests, having little time between working full-time and tending to household chores. She derived her satisfaction, indeed meaning, from pleasing her husband and being a model housewife and provider. She had little sense of self apart from her relationship with her husband and could not conceive of living without him. After his confession of infidelity and his threat of leaving, she felt that he was correct because she had failed him and that the other woman was probably superior to herself. She also experienced a sense of agitation and hopelessness for her life. She saw no purpose in living without her husband and contemplated suicide. She confided these destructive wishes to a coworker, who immediately referred her for therapy.

The majority of patients with this form of depressive personality are women. This is not surprising because in our culture women are trained to suppress their potential for autonomous derivation of meaning and to gain their sense of worth from reflected praise from others rather than directly by independent effort (8). A variant of the "dominant other" type that is more common in men is found in cases where the needed relationship is not to another human individual but to an organization or institution.

A second form of depressive personality organization may be called the "dominant goal" type. These individuals invest their self-esteem in the achievement of some lofty goal and shun any other activities as possibly diverting them from this quest. Originally, achievement was rewarded by the parents, and so high marks or some outstanding performance was sought as a way to ensure support and acceptance. In time, the individual selects some fantastic goal for himself which he then pursues fanatically, apparently for its own sake. However, closer scrutiny reveals that the achievement of this goal is burdened with surplus meaning. The individual believes that the goal will transform his life and, possibly, himself. Attaining his desired objective will mean that others will treat him in a special way or that he will finally be valued by others. Just as the "dominant other" type of depressive individual uses fantasies of the relationship to derive a feeling of worth, the "dominant goal" type of depressive individual obtains meaning and esteem from fantasies about obtaining his objective. Both types also use these fantasies to eschew gratification or meaning from other activities in everyday life. In contrast to the "dominant other" type, this form of depressive personality is usually seclusive, arrogant, and often obsessive. In addition, this form of personality organization is most commonly found in men. An illustration of this type of depressive personality is Mr. B.

Case 2. Mr. B was a middle-aged research scientist who came for treatment complaining of a lack of interest in his work, a sense of futility in all of his activities, difficulty sleeping, fatigue, various psychosomatic complaints of recent origin, and a subjective feeling of depression. The various symptoms had developed over the previous two years after he had not received a specific position that he greatly desired. It became clear that his depressive episode was not simply a response to missing out on the coveted job but that his failure to obtain the position signaled to him that his career plans, which were meticulous, had been thwarted forever. He spoke of great aspirations of being awarded spec-

tacular prizes and becoming director of a prestigious research institution. His being passed over for the job forced him to realize that he might never achieve his goals.

This man was obsessed with his work, putting in extraordinarily long hours (although he did not particularly enjoy what he did) and being so driven that some colleagues refused to collaborate on projects with him. His marriage was, at best, a disaster. He expected his wife to plan her life around his work and expected special treatment from her because of the alleged importance of his work. He had no hobbies or interests but was consumed with fantasies of his glorious future, when everyone would respect him and treat him with deference.

He was raised by parents who were poor and centered their hopes for upward mobility on their children. The patient's two siblings were also driven professionals. In his childhood, he had been made to understand that he had a special mission in life and that the pursuit of excellence (and prestige) was to be his way of repaying his parents for the sacrifices they endured to send him to the best schools, etc. Pursuits that were not "productive" were forbidden. The belief in this messianic role was further fostered by the local minister, who took the patient under his tutelage and, impressed by the boy's willingness to work and learn, also painted a glorious picture of his future if he applied himself. Eventually, Mr. B lived only for this great goal that, once obtained, would bring all sorts of gratification and meaning. The remote possibility that he might simply remain a respected but not extraordinary scientist so discouraged him that he no longer found meaning in his activities. The goal and its quest were his motivation for being.

The two types of depression illustrated by Ms. A and Mr. B should not be considered "reactive" in the usual sense of the word. If the external events were decisively important, it is because they acted as a total life experience and because the patient integrated them in a particular way. When a specific precipitating event exists and is likely to be of the greatest importance, we can recognize that it is so because it had a special meaning for the patient and consequently assumed pathogenetic power. Thus the environment and the patient both contribute to the transformation of the event into a cause: the environment, by offering the contingency of the event; the patient, by attributing either consciously or unconsciously a special meaning to the event.

A third type of depression occurs that cannot be recognized as episodic or recurrent but is a form of chronic character structure or personality. In patients with this disorder, depression appears to be a *constant* mode of feeling lurking in the background during everyday life. This third type differs from the previous two in that the individual is not able to ward off clinical depression by either a mode of relating or a fantasized goal. Individuals with this type of depression inhibit any form of gratification because of strongly held taboos that were instilled by their families and culture. They suffer from a chronic, mild sense of futility and hopelessness, which results from a lack of involvement in everyday activities. There is also a sense of emptiness because they do not develop deep relationships for fear of being exploited or rejected. At times, some will use alcohol or drugs to momentarily relieve their dysphoria, but usually they are too prideful or moralistic to take even these steps to ease their suffering. Other characteristics of this type include hypochondriasis, pettiness in interpersonal relationships, and a harsh critical attitude toward themselves and others. Most falsely believe that their every move is carefully watched by others.

Individuals with this type of depression experience episodes of clinical depression when some event forces them to reevaluate their mode of existence. They then perceive their lives as barren or devoid of satisfaction but also believe that they are incapable of changing. They feel caught in a way of life that has revealed itself as meaningless. This act of reevaluation may be provoked by a trivial external event that has profound subjective significance. From the point of view of an external observer, the depression appears to have been precipitated by changes in the daily life of the individual. This type is equally frequent among men and women. An example of this type of depressive personality is Ms. C.

Case 3. Ms. C was a middle-aged, single woman who sought treatment after she became increasingly depressed, tired, and anxious. She had experienced periodic episodes of depression for 10 years, occasionally seeking treatment but not sticking to it. She could describe no precipitating event but merely complained that she saw less and less purpose in living and found that nothing offered her any gratification. Furthermore, she saw no prospect of being able to alter her situation; any attempt at adding some sort of enjoyable activity was clouded by guilt and anxiety. Her one area of gratification had been her work and she had successfully risen to an important position in her field, but this also had become meaningless and empty.

This woman's weekly routine was to work late, then go home and replay the day's events in her mind, looking for things she had done for which she could criticize herself. Then she would prepare for the next day. She spent Saturdays running errands and doing household chores. On Sundays she would call her parents and listen to her family complain about how miserable and sick they were, which would leave her feeling guilty for the day. In the afternoon she would go for a walk or to a movie and in the evening prepare for the next work week.

She was afraid of men because relationships with them might lead to sexual encounters, which she perceived as sinful. She did attend a few dinner parties related to her work but always felt anxious because she believed she was spending her time in an unproductive manner. As a youngster she had shown considerable promise as an artist but never pursued painting because she considered this activity a wasteful indulgence. As an adult, the only time she felt free from guilt or anxiety was when she was working.

This woman was aware of the irrational anhedonia of her way of life, yet she felt powerless to change her mode of being. Throughout her childhood she had been indoctrinated with a stern morality that the world was a sinful place and that

work and obedience to God were the only salvation. She was no longer religious but still adhered to the dictates of her earlier years. As she herself recalled in therapy, she really never had a childhood. She went from infancy to adulthood, becoming precociously responsible and serious. The atmosphere at home while she was growing up was somber and serious. Frivolity or exuberance were never permitted; only work and effort were allowed. When she went to college and encountered a totally different set of values she experienced a sense of confusion and tormented herself over the possibility that she was losing her moral fiber, a conflict that almost led her to suicide. She resolved these difficulties by isolating herself and concentrating only on her work, a mode of life that continued until she came for therapy.

These three forms of depressive personalities differ in superficial characteristics but have basic features in common. The individuals share similar distorted beliefs about themselves and others that guide their behavior. For example, they all share anxiety over the direct attainment of pleasure, they all fear that spontaneous activity will result in rejection or criticism from others, they overvalue the opinions of others, and they overestimate their own effects on the inner lives of others. Most significantly, overwhelming inhibition prevents the development of alternate modes of attaining meaning from activity.

All three forms of depression described above are reactive to a series of life stresses that, however, are interpreted in a particular manner by the individual. To the external observer the onset of the depression may appear to be without cause or endogenous, yet if one reconstructs the patients' experiences as formulated by their inner systems of beliefs, it becomes apparent that a severe blow to their sense of esteem or meaning preceded the onset of the depression. In this regard, Leff and associates (1) looked at events that occurred before the onset of depression in 40 hospitalized patients. On the basis of symptom criteria, 13 of these patients had a diagnosis of endogenous depression and 27 had a diagnosis of reactive depression. These authors found that all of their sample had suffered adverse environmental stress regardless of their symptoms. However, the nature or existence of these stresses became apparent only after they understood the core conflicts that affected the way the patients interpreted their experiences. Other studies, such as the one by Paykel and associates (9), have also found more negative life events preceding the onset of illness in a group of depressive patients than in a control group, but they have relied mainly on objective data rather than the patient's subjective evaluation of experience. Our emphasis is that depressive episodes follow some alteration in the life of the individual and that this change can be understood only as part of the subjective interpretation of experience by the individual. Alternately, what causes someone to fall ill are not experiences per se but the personal meanings one attaches to those experiences.

CHILDHOOD EXPERIENCES OF ADULT DEPRESSIVE PATIENTS

Aside from reports that document childhood bereavement, there are surprisingly few studies in the literature documenting the childhood experiences of depressed adults. The well-known studies of Cohen and associates (10), which focused on the life histories of 12 hospitalized manic-depressive patients, may represent the only intensive and systematic investigation into the early life of patients with an affective disorder. Although Cohen's work involved patients with bipolar illness, many of our findings in unipolar individuals (to be described below) parallel those discussed by Cohen.

A possible additional source of information regarding the childhood of depressed adults may come fron anthropological studies describing childrearing practices in societies that produce an inordinate number of depressed adults. Two such exemplary studies exist: one concerns the Ojibwa Indians of northern Canada (11), and the other concerns the Hutterite societies of the Dakotas and Montana (12). Parker (11), who studied the Ojibwa Indians, reported that they believed children were empty vessels who would be easy prey to malignant spirits unless disciplined and toughened by starvation and other deprivations. The Ojibwa child was indoctrinated early into the pessimistic cultural beliefs of his tribe: he was made to feel responsible for misfortunes and taught that the deities could be propitiated only by self-induced suffering. He remained close to his family unit, which showed prideful and jealous attitudes toward others. Finally, in early adolescence, the young Ojibwa was to have a vision proclaiming the future goal he had to fulfill.

Eaton and Weil's study of the Hutterite community (12) stressed the predominance of puritanism, self-control, and duty orientation. The Hutterite child seemed to experience a free and gratified infancy but at roughly age 30 months spent most of the day in a community nursery, where he learned to conform to group values and to identify with the group. Education, which Eaton and Weil term "religious indoctrination" (p. 31), was highly moralistic. The authors were impressed by the lack of freedom, spontaneity, and privacy allowed the Hutterite child. At the same time, there was a good deal of social comforting and encouragement, but always in return for conformity and self-control. The child learned to fear the judgment of his teachers, his peers, and, ultimately, his God.

These studies show that it is difficult to separate out childrearing practices from the general beliefs of a culture. Indeed, much of the shaping of a child's later personality appears to result from the internalization of the attitudes of significant adults in his formative years. Much of what is described by Parker and by Eaton and Weil has also been reported by Cohen and associates (10) in middle-class American families that

produced manic-depressive adults. In these families there was a sense of duty orientation, punishment of spontaneity through shaming, inducing guilt, and rewarding conformity and conspicuous achievement.

Our own findings on the childhood of depressive adults are similar to those reported by the authors cited (10–12). On reviewing the childhood of depressive adults one does not find the disorganization of families of schizophrenic or psychopathic adults. Rather, the family structure may have been too stable, with little tolerance for individual deviation from an expected norm. One parent was usually dominant, and the rest of the family accepted a submissive role.

The infancy of the depressive adult appears to have been adequate, with appropriate maternal nurturance. However, with the onset of the toddler phase, a significant change occurred in the mother-child relationship (10, 13). The mother's love was no longer given unconditionally but depended on behavior that met parental expectations. Although this transition occurs in most families at this stage of development, in the family of the depressive individual the change was harsher and, sometimes, abrupt. The reason for this change in parental expectations varied in different families. In some a younger sibling was born, displacing the older child in the mother's affections. In others the child's care was directed by the more dominant father, who, without realizing it, exploited the child to fulfill his own fantasized goals. In yet others, the mother was threatened by the normal oppositional behavior of a willful toddler who was such a troublesome contrast to a helpless infant. In all cases, however, the child at first experienced a period of nurturance that was soon transformed into what was experienced as conditional love given for good behavior.

Good behavior often meant the inhibition of spontaneously gratifying behavior. The child learned to forego satisfaction needs to ensure security needs. Rightly or wrongly, in early childhood the future patient felt that willful behavior was punished with threats of abandonment and later by the inducing of guilt or shame. In later childhood the future depressive adult came to believe that he could not obtain satisfaction directly from achievement. Rather, attainment of goals was for him only a way of paying back a debt to the parent or of enhancing the honor of the family. He nourished the hope for achievement but did not learn to experience independent gratification or meaning from accomplishment. Where passivity and submission were experienced by the child as demanded by a parent or another important family member, a personality characterized by a special relation with "a dominant other" emerged. Where worldly success became the means of obtaining parental favor, the seeds were sown for a type of personality committed to the pursuit and achievement of a "dominant goal." In those families where very little support was given to the child and parental acceptance could be ensured only by the relinquishing of spontaneity and involvement with extrafamilial contacts, the third form of depression appears to have developed.

All of the families were excessively critical and moralistic, and although in many instances they did not mean to have this effect, they made the child feel essentially worthless unless he obtained the approval of the powerful parents. In our limited clinical experience, however, we have found that patients with severe depression differ from more mildly affected individuals in two historical factors. Among patients with severe depression the mother was more often the dominant other; among those with mild depression the father assumed this role. The reasons for this difference may be that when the mother is the dominant parent 1) inhibitions are instituted earlier, 2) the child is with the critical parent for more of the time, and 3) the child does not have a continued loving relationship even with the weak parent (i.e., if the mother were the weak parent rather than the dominant one, she would be more likely than the father to provide this kind of relationship). The other difference was that patients with severe depression were obliged to fulfill their inhibited, duty-oriented, and passive roles in the hope of escaping from a sense of inner evil rather than in the hope of gaining gratification. Much of the childhood of the severely depressed adult was spent in defending against terrible negative possibilities rather than learning how to maneuver himself or manipulate others to add positive elements to everyday life.

From these early experiences the depressive patient creates beliefs about himself and others that are, to a large extent, unconscious but that determine much of his behavior. A great deal of what is observed in adult depression may be understood as the retention and elaboration of these childhood beliefs applied to a state of maturity and social reality for which they are grossly inappropriate. The depressive adult still rigidly adheres to his unconscious cognitive system for structuring his social and inner world. It is this continuation of childhood social-cognitive patterns which ultimately predisposes one for depressive illness and forms the basis of depressive personality organization.

THE CONCEPT OF DEPRESSION

We conceptualize depression as a primary affect, free of complex metapsychological underpinnings. This view appears more congruent with biological approaches as well as with clinical data. However, not all affects are of identical complexity, and depression appears to require a degree of maturation and cognitive constructs that aid in structuring one's ideas of one's self and of others (14). These cognitive structures may not be fully conscious, just as Piaget's concept of schema implies an unconscious psychic structure that organizes experience and gives it meaning.

Depression is related to sadness but also implies a deviation from the normal way of experiencing sadness (15). Sadness—the normal response of the human being when he apprehends a situation that he would have preferred had not happened and that he considers adverse to his well-being—has positive values. It becomes a motivational source that induces the individual to remedy the effect of the adverse event. When a remedy is found, when what we call "sorrow-work" is completed, sadness disappears or greatly diminishes. For instance, sadness caused by the death of a loved person will eventually disappear when the survivor feels that the person has left important parts or effects of himself on earth, when the survivor comes to think that the person is still alive in some place, or spiritually, or, on the contrary, when he comes to think that the deceased is not so indispensable as he once thought but can be replaced by another relationship. In other words, the normal person is able, to a large extent, to metabolize sorrow and sadness. On the other hand, the person prone to depression in many instances lacks this capacity.

Depression also starts as something very positive. A complex situation that was totally unconscious or not yet clarified in its global effects or its implications and ramifications finally emerges partially or totally in the psyche of the patient, either as a sudden realization or a slow maturation and progressive understanding. Generally, it takes the form of the recognition that a long commitment—to another person, to a life goal, or a combination of both—will end in failure. The patient, however, cannot do "sorrow-work." Together with a personal impoverishment, he has the conviction that his unhappy state will continue indefinitely. He is trapped; he considers himself in a state of helplessness because he feels that he can do little to ameliorate his painful state. He believes that other compensatory avenues of meaning do not exist. The reasons why a realization that at first may have positive values ends up by being destructive and leads either to a temporary or prolonged feeling of helplessness are to be found in the patient's life history. The sequence of certain events and the specific psychological ways of dealing with them make some people prone to depression.

After the onset of a clinical episode, the individual will grossly distort his conceptions of himself, of others, of his future, and of his current environment in a pathological way. The extent to which an individual accepts these distortions and feels unable to fight against them defines the continuum between mild (or neurotic) and severe (or psychotic) depression. In milder forms, the individual suffers from the distortions that form the cognitive aspect of depression, but he does not accept this world view. He sees it as a painful, ego-alien state that he wishes to be rid of, although he does not know how to extricate himself from his situation. In the more severe forms, the depressive patient's distortions are wholeheartedly accepted, and the individual believes his erroneous world view. As such, the depression crosses the line into psychosis. This deterioration may occur in each of the three types of depression described above. It is less a matter of what precipitated the onset of depression than the individual's ability to avail himself of alternate avenues of cognition about himself and his ability to ward off acceptance of distortions in his world view that are permutations of the depressive experience itself.

In summary, we view the depressive adult as an individual who, as a result of childrearing practices as well as other experiences, has narrowed his sources of meaning and gratification to a dangerous extent. Furthermore, he suffers from a paucity of cognitive alternatives that would keep him from progressing into further despair following a loss of meaning from the environment. Becker (16) has described the depressive person as one who is "left in the hopeless despair of the actor who knows only one set of lines, and loses the one audience who wants to hear it." This limitation to new ways of thinking and this self-inhibition from new experiences may best characterize depression.

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Medication Use and Deaths Attributed to Asphyxia Among Psychiatric Patients

BY THOMAS J. CRAIG, M.D., M.P.H.

In a review of the charts of inpatients who died in 1969–1977 the author found 49 whose death could be attributed to asphyxia. Compared with a matched control group, 48 of the asphyxia patients represented three distinct pathologic categories: 1) older patients with a history of serious physical illness whose deaths appeared unrelated to psychotropic medication use (40%), 2) a group whose deaths were associated with seizures (31%), raising questions about subtherapeutic anticonvulsant levels in association with the use of psychotropic drugs, and 3) a group of patients who choked to death (29%). Choking has been theoretically linked to a combination of dopamine blockade plus strong anticholinergic effects leading to impairment of swallowing. The third category appears to have been virtually eliminated by the use of a drug monitoring system and the Heimlich maneuver.

Among the less common complications that have been attributed to the use of psychotropic medication, perhaps none is more enigmatic than death by asphyxiation. Although standard texts of psychopharmacology fail to even mention this entity as a potential side effect of drug therapy (1–3), sporadic anecdotal reports have appeared that have been divided in their conclusions. Some reports have linked this cause of death to the use of drugs, particularly neuroleptics. However, deaths by asphyxia have also been reported among people not receiving drugs. Table 1 summarizes the characteristics of reports listing patients whose deaths were possibly due to asphyxia (4–21).

Six reports (4, 10, 12, 13, 15, 16) found 6 or more cases of death by asphyxia, a total of 118 cases, and twelve reports (5–9, 11, 14, 17–21) found 5 or fewer cases, a total of 28 cases. Among those reports finding 6 or more cases, five (4, 10, 12, 13, 16) reported the number of patients receiving psychotropic medication at the time of death. Thirty-two of the 76 patients (42.1%) in these reports were receiving psychotropic drugs. In contrast, 25 of the 28 patients (89.2%) from the reports finding 5 or fewer cases were receiving psychotropic medication.

In their discussion of the possible mechanisms of death, three of the six reports finding 6 or more cases stated that there was only equivocal or speculative evidence of an association between psychotropic drugs and these deaths, and the authors of two studies felt that the primary mechanism was probably related to seizures. The other study (of 7 patients) postulated a combination of respiratory depression due to drugs plus esophageal reflux related to eating large amounts of food.

In contrast, several reports finding 5 or fewer cases tended to implicate drugs as a major factor. Three studies speculated on a possible adverse effect of drugs on the respiratory system. One study cited seizures as a factor. One study postulated "autonomic instability related to drug effects on the hypothalamus and brainstem." Two reports suggested an effect on cardiac function, possibly associated with either a diminished deglutative-respiratory reflex or gastric hyperacidity. However, even in this group, four reports failed to directly implicate drugs, while one report implicated a synergism between a small dose of thioridazine with an extremely high blood alcohol level.

In summary, those reports which focused on a large group of patients failed to find a strong relationship between drugs and asphyxial deaths, while the authors of some (but by no means all) of the small anecdotal studies speculated on a variety of possible drug-related mechanisms. However, none of these reports provided even circumstantial evidence to support its contention as to mechanism with the exception of the association of seizures with a substantial minority of patients in six reports and the association of poor previous physical health with more than half the cases of asphyxial death in one large series.

There are several difficulties in interpreting these reports. Most are anecdotal reports of a small number of patients with no reference to the population from which they came. Thus no assessment of the relative magnitude of this entity in terms of death rates can be

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TABLE 1
Characteristics of Reports of Possible Asphyxial Death

Study	Total Patients	Patients on Psychotropics	Speculated Mechanism of Death
Hollister (4)	19	3	Glottal spasm from a seizure and irritation from aspirated food
Wardell (5)	3	3	Possible effect of psychotropic drugs on respiratory defense system
Feldman (6)	1	1	Possible effect of psychotropic drugs on respiratory defense system
Farber (7)	1	1	Possible adverse effects of drugs on respiratory system
Zlotlow and Paganini (8)	5	5	"Masked fit" accompanied by glottal spasm and tracheal aspiration
Reinert and Hermann (9)	5	5	Autonomic instability related to drug effects on hypothalamus or brainstem
Hussar (10)	9	2	Equivocal evidence that tranquilizers may impair swallowing mechanism
Johnson and associates (11)	1	1	Fatal syncope—role of phenothiazines uncertain
Plachta (12)	7	7	Depression of respiratory tract by drugs and esophageal reflux related to eating large amount of food
Hollister and Kozek (13)	6	6	Seizure possibly associated with Adams-Stokes episode
Wendkos and Clay (14)	1	1	Abnormal eating habits
Hussar (15)[a]	42	—	Connection with tranquilizer medication speculative
Von Brauchitsch and May (16)	35	14	Poor general physical health, sudden intercurrent illness, neurological impairment, poor dental condition, and poor eating habits; no direct evidence of connection with drugs
Leetsma and Koenig (17)	2	2	Mechanism of death and etiological role of phenothiazines cannot be adequately evaluated
Moore and Book (18)	3	3	Interference with cardiac rhythm or diminished deglutative-respiratory reflex
Bland (19)	1	1	Synergism of drug and alcohol
Bort (20)	1	1	Cardiac arrest associated with gastric hyperacidity and aspiration
Koranyi (21)	4	1	No single mechanism

[a]The number of patients receiving psychotropic medication was not reported in this study.

made. Also, since medication use among patients who died of asphyxia was not compared with use of medication among the total patient population, no assessment of the relative use of these drugs is possible. The latter analysis is crucial to any attempt at implicating the use of medication in a causal way. Finally, there are conflicting data on the presence of predisposing factors in the at-risk population.

For these reasons, my colleagues and I undertook a case-control study of the occurrence of deaths in any way related to asphyxia or aspiration at Rockland Psychiatric Center. The specific questions addressed by this study are 1) What are the absolute and proportionate mortality rates for this cause of death in a defined population of psychiatric inpatients? 2) Is there a significant association between either a history of physical illness or medication use and death by asphyxia? and 3) Are there subcategories of patients within the general category of patients who died of asphyxia, and is there a significant association of previous physical illness and/or medication use with any of these subcategories?

METHOD

Case Identification

We identified the cause of death for all inpatients of a large state hospital who died during the period 1969–1977. The case group for this study consisted of all patients with a death certificate diagnosis of anoxemia or asphyxiation (*ICDA* codes E911, E972, and 783) (N=41) plus 8 patients out of a total of 31 who were diagnosed as having died of pneumonia or aspiration pneumonia (*ICDA* code 486) but who on review of the chart were judged to have died of a cause related to asphyxia.

General Population Identification

For purposes of obtaining absolute and proportionate mortality rates, the number of inpatients who had been in the hospital for a minimum of 30 days as of March 31 of each of the years 1970–1977 were used as the denominators for a calculation of annual rates. The numerator of the rates was the number of patients with a minimum 30-day hospitalization who died during each of the years 1970–1977. By limiting the study of mortality rates to the years 1970–1977, we excluded from the numerator 3 of the identified cases of patients who died during 1969. The 30-day restriction was imposed because of the relative instability of the less than 30-day inpatient population figures over time. Seven patients with a stay of less than 30 days were excluded from the calculation of mortality rates.

Case-Control Comparison

Due to substantial differences between the cases of asphyxia-related death and the total hospital popu-

TABLE 2
Demographic and Clinical Characteristics of 48 Patients Who Died of Asphyxia

Characteristic	Men (N=29)		Women (N=19)		Total (N=48)	
	N	%	N	%	N	%
Age						
16–24	3	10.3	0		3	6.3
25–34	3	10.3	2	10.5	5	10.4
35–44	7	24.1	1	5.3	8	16.7
45–54	5	17.2	4	21.1	9	18.8
55–64	8	27.6	3	15.8	11	22.9
65–74	2	6.9	5	26.3	7	14.6
75 and older	1	3.4	4	21.1	5	10.4
Diagnosis						
Schizophrenia	18	62.1	11	57.9	29	60.4
Organic brain syndrome	8	27.6	5	26.3	13	27.1
Mental retardation	1	3.4	0		1	2.1
Other	2	6.9	3	15.8	5	10.4
Duration of current hospitalization at time of death						
Less than 30 days	1	3.4	6	31.6	7	14.6
30 days to 6 months	0		0		0	
6 months to 1 year	2	6.9	0		2	4.2
1–5 years	4	13.8	2	10.5	6	12.5
More than 5 years	21	72.4	11	57.9	32	66.7
Unknown	1	3.4	0		1	2.1
Circumstances of asphyxial death						
Choking	7	24.1	7	36.8	14	29.2
Seizure	14	48.3	1	5.3	15	31.3[a]
Other	8	27.6	11	57.9	19	39.6

[a] $X^2=7.98$, $p \leq .01$.

lation, especially in regard to age, sex, and, in the case of women, duration of hospitalization, we conducted a case-control comparison. Cases and controls were compared with respect to two sets of variables that a review of the literature had identified as having a possible relationship to asphyxia death: 1) presence of a history of physical illness or certain treatments (e.g., shock therapy or lobotomy) before death and 2) medication use at the time of death. Each case patient was matched to a control drawn from the total hospital population according to the following criteria: sex, date of birth, date of admission, diagnosis, and presence of the control in the hospital at the time of the death of the matched case patient (the control may have subsequently died of other causes).

Clinical Evaluation

The charts of 48 case patients (1 chart was unavailable) and 48 control patients were reviewed. From these reviews, the following information[1] was obtained: diagnosis, medication being taken at the time of death, duration of hospitalization, associated physical illness or treatment, circumstances of death, and cause of death.

Computer Evaluation

For all 49 case patients and 49 controls, duration of medication use up to two years before the case death was compared for the following drug categories: piperidine neuroleptics, tricyclic antidepressants, more than one neuroleptic, multiple anticholinergics, and one neuroleptic plus one or more anticholinergics. The choice of these drug categories was based on clinical evaluation indicating possible association with asphyxial death. This comparison was possible due to the existence of a computerized drug monitoring system that records all psychotropic drug prescriptions for hospital inpatients (22).

Data analysis was carried out using chi-square analysis of matched pairs when appropriate. When matched pair analysis was not appropriate, chi-square analysis of pooled data was carried out.

[1] We obtained information on the control patients in reference to the date of death of the matched case.

RESULTS

Description of Patients Who Died of Asphyxia

Table 2 lists demographic and clinical characteristics of the 48 patients who died of asphyxia. Men predominated over women by a 1.5:1 ratio. Men tended to be younger (61.9% were younger than 55), and women were older (63.2% were older than 55). Psychiatric diagnoses were similar among men and women; the majority had a primary diagnosis of schizophrenia. The majority were long-term patients, although 31.6% of the women had been recently admitted. However, most of these women had long histories of psychiatric care.

Among the circumstances of death as recorded in

TABLE 3
History of Physical Illness Among 48 Patients Who Died of Asphyxia (Cases) and 48 Controls, by Circumstances of Asphyxial Death (percents)

Type of Physical Illness	Circumstance of Death					
	Seizure (N=15)		Choking (N=14)		Other (N=19)	
	Case	Control	Case	Control	Case	Control
History of seizures	80.0	40.0[a]	7.1	7.1	15.8	21.1
History of shock treatment or lobotomy	20.0	20.0	42.9	28.6	15.8	42.1
Serious medical condition or deterioration	40.0	0.0[a]	35.7	7.1	79.0	0.0[b]
Other medical condition	13.3	20.0	28.6	28.6	15.8	31.6
Neurologic condition	33.3	33.3	0.0	21.4	21.1	10.5
Mental retardation	6.7	13.3	7.1	7.1	5.3	0.0
None	0.0	13.3	7.1	42.9	0.0	26.3

[a] $\chi^2=5.2$, $p<.05$.
[b] $\chi^2=21.6$, $p<.01$.

the patient's chart, three general categories of death were identified. One group (N=14) represented patients observed to have either choked to death or to have died of apparent suffocation from food aspiration after a meal. A second group (N=15) consisted of patients who were observed to die after a seizure or who were found dead but had a history of seizures. The third group (N=19) represents a heterogeneous category of deaths associated with vomiting, infection, or sudden death not subsumed under the above categories. Almost half the men fell into the seizure category, while only 1 woman died by this mechanism. Conversely, twice as high a proportion of women fell into the "other" category, but equal numbers of men and women choked to death. The overall comparison of sex proportions for seizure deaths versus all other deaths was the only dichotomized comparison to reach statistical significance (see table 2). When these categories of death were examined by age, the patients in the "seizure" category were younger than the total case group, while the patients in the "other" category were older. Patients in the "choking" category were evenly distributed over all age groups. There was a statistically significant difference among these three patterns of age distribution ($\chi^2=9.88$, $p<.01$).

Mortality Rates

During 1970–1977 both absolute and proportionate mortality rates fluctuated considerably. For men, the absolute mortality rates ranged from .6 per 1,000 to 3 per 1,000, while for women the range was from .5 per 1,000 to 2.3 per 1,000. Proportionate mortality (i.e., proportion of total deaths due to asphyxia-aspiration) showed similar variability, averaging 3.53% for men and 1.55% for women.

Case-Control Comparison

When a history of physical illnesses was compared for the asphyxia patients and the controls, the most striking finding was the fact that the groups were quite similar on most items. The only statistically significant differences were the increased prevalence of serious medical conditions or deterioration among the asphyxia patients ($\chi^2=29.7$, $p<.01$), the absence of asphyxia patients with no preexisting medical condition ($\chi^2=10.1$, $p<.01$), and the presence among these patients of twice as many men with a history of seizures (48.3%) as compared with controls (24.1%) ($\chi^2=4.4$, $p<.05$).

Because of the differences observed between groups of asphyxia patients categorized according to circumstance of death, table 3 presents a separate case-control comparison carried out after patients were categorized by death circumstances. Again, a marked similarity between asphyxia patients and controls was found. The significant association between physical illness and asphyxia death was most closely linked to the "other" category of death; 79% had a serious medical condition or deterioration as compared with none of the matched controls (see table 3). The other two death categories ("seizure" and "choking") had about half this prevalence of serious medical conditions (40% and 35.7%, respectively). The "seizure" comparison was significant (see table 3). The "seizure" category also had significantly more asphyxia patients with a history of seizures than the controls (see table 3).

When medication use was compared among asphyxia patients and controls, the groups were also similar; statistically significant differences existed only among the use of anticonvulsants by men ($\chi^2=7.7$, $p<.01$) and the use of piperidine neuroleptics ($\chi^2=5.1$, $p<.05$), tricyclic antidepressants ($\chi^2=4.4$, $p<.05$), and anticonvulsants ($\chi^2=4.3$, $p<.05$) by the total group. In all comparisons the asphyxia patients used the drug categories significantly more than controls. In only one comparison did use of a category exceed 50% (51.7% of male asphyxia patients used anticonvulsants).

When we analyzed medication use among asphyxia patients and controls, with the former categorized as to circumstances of death, we again found similar proportions in both groups who used most drug categories (see table 4). However, each category of death circumstances showed some evidence of specificity as to medication use. The "seizure" category showed a significantly greater use of anticonvulsants among

TABLE 4
Medication Use among 48 Patients Who Died of Asphyxia (Cases) and 48 Controls, by Circumstance of Asphyxial Death (percents)

	Circumstance of Death					
	Seizure (N=15)		Choking (N=14)		Other (N=19)	
Type of Medication	Case	Control	Case	Control	Case	Control
Aliphatic	20.0	26.7	28.6	35.7	15.8	21.5
Piperidine	53.3	13.3	35.7	14.3	21.5	15.8
Piperazine	13.3	26.7	35.7	42.9	15.8	15.8
Butyrophenone	6.7	—	0.0	7.1	5.3	15.8
Thioxanthine	13.3	13.3	21.4	0.0	0.0	0.0
Tricyclic	13.3	6.7	28.6	0.0	10.5	0.0
Benzodiazepine	13.3	6.7	14.3	21.4	15.8	0.0
Anticholinergic	26.7	20.0	42.9	28.6	5.3	21.5
Anticonvulsant	80.0	20.0[a]	14.3	7.1	15.8	21.5
Multiple neuroleptic	13.3	20.0	35.7	21.4	10.5	10.5
Multiple anticholinergic	40.0	33.3	71.4	35.7	10.5	21.5
Other neuroleptic	0.0	0.0	7.1	0.0	0.0	5.3
Lithium	0.0	6.7	0.0	0.0	0.0	0.0
Reserpine	0.0	0.0	0.0	7.1	0.0	5.3
None	0.0	20.0	0.0	7.1	15.8	21.5
Any neuroleptic	93.3	60.0	100.0	78.6	47.4	63.2

[a] $\chi^2=8.5$, $p<.01$.

asphyxia patients than controls (see table 4). The only other substantial difference in this category was in the use of piperidine neuroleptics ($\chi^2=3.75$, $p<.10$). The "choking" category showed a nonsignificant ($\chi^2=2.30$, $p>.10$) excess in the use of multiple anticholinergic drugs by asphyxia patients. The "other" category demonstrated a relatively low use of all medications; the proportions were almost identical to the control group.

In examining the duration of drug use, we noted few significant associations. For piperidine neuroleptics, only the short-term use (within the month before death) was significantly more common among asphyxia patients. They showed significantly greater use of tricyclic antidepressants up to one year before death, although only 9 asphyxia patients used these drugs. These patients tended to have used multiple anticholinergics and a neuroleptic plus an anticholinergic significantly more often than controls for at least 360 days out of the 2-year span ($\chi^2=4.92$, $p<.05$), although use for shorter periods was not significantly different between the two groups. When duration of drug use was examined by specific death category, no significant associations were found. A review of chart and computer medication records revealed that in no case were any of the patients receiving psychotropic medication in excess of the recommended range.

DISCUSSION

Mortality Rates

The presence of asphyxia as a concomitant of sudden death among psychiatric inpatients appears to be associated with an absolute mortality of about 1-2 deaths per 1,000 hospital population per year, constituting a proportionate mortality of about 2.5% of the total hospital deaths. Of particular interest is the fact that both absolute and proportionate mortality rates in this population are strikingly similar to those reported in the earlier literature. Von Brauchitsch and May (16) cited a rate of 1.7 per 1,000 absolute mortality, which approximates our combined male-female rate. Hussar (15) reported a 3.3% proportionate mortality, while Von Brauchitsch and May (16) reported a minimum proportionate mortality rate of 4.2%. What is lacking in all studies, however, including our own, is the presence of data on the absolute and proportionate mortality rates for this entity in the general population. A comparison of these rates is impossible in the absence of a 100% autopsy rate because for many of these deaths the cause was not suspected clinically and was discovered only at autopsy (16, 23) or after other procedures (24).

Possible Mechanisms of Asphyxial Death

Perhaps the most informative data bearing on the possible mechanisms of death among these patients were the circumstances surrounding the death itself. When these categories are compared with the history of physical illness and medication use at the time of death, striking patterns emerge. The "seizure" category is associated with a significantly greater history of serious medical illness or deterioration (40%), use of anticonvulsants (80%), and a nonsignificant excess of use of piperidine neuroleptics (53%). The "choking" category is associated with a nonsignificant excess in the use of multiple anticholinergic drugs (71.4%). The category "other," representing 39.4% of the deaths, is significantly associated with a history of serious medical illness or deterioration (79%).

Since a large number of comparisons was made between asphyxia patients and controls and a remarkably small number of significant differences was found,

the possibility remains that some of the statistical associations suggested are the result of chance alone. However, the biologic plausibility of the findings together with an analysis of earlier studies suggests that these findings cannot be dismissed in a search for possible mechanisms of asphyxial death.

Seizures

The role of seizures as a high-risk concomitant of this type of death appears solidly established (4, 25). The most parsimonious explanation would be that seizure deaths occurred due to subtherapeutic levels of anticonvulsants. However, in the absence of data on the blood levels of anticonvulsants among these patients, the thesis is speculative and based only on the finding of subtherapeutic levels in an earlier study of similar deaths among nonpsychiatrically ill epileptic patients (25). The reason for subtherapeutic anticonvulsant levels is also speculative but could include patient noncompliance or inadequate absorption of drug. Whether neuroleptics may be involved, either through a mechanism of interaction with anticonvulsants resulting in subtherapeutic anticonvulsant levels (26) or through the lowering of seizure threshold (27, 28), must remain speculative, although the latter could be implicated in the 3 patients who were not receiving anticonvulsants who died after seizures.

Further work is needed to identify the mechanisms involved in the development of subtherapeutic anticonvulsant levels, both alone and in combination with other drugs. In addition, regular monitoring of patients' anticonvulsant blood levels is indicated as a preventive for such deaths (25). In any sudden death among epileptic patients, postmortem serum anticonvulsant levels should be routinely obtained. In addition, more studies are necessary to determine more specifically the potential for given psychotropic agents to induce seizures, either through lowering the seizure threshold or through other mechanisms. Until this is known, recommendations regarding the use of specific psychotropic agents in epileptic patients must be deferred, although the present study suggests a nonsignificant excess of piperidine neuroleptic use among the cases of seizure at the time of death. Whether such deaths are more common among epileptic patients hospitalized for psychiatric reasons or receiving psychotropic drugs cannot be determined from the literature or from the present study. However, the fact that they do occur in nonhospitalized epileptic patients and those not receiving psychotropic drugs does seem firmly established (25).

Choking

The possible physiologic mechanisms invoked as being associated with impaired swallowing fall into two categories: 1) dopamine blockade, producing both central and peripheral impairment of swallowing (either complete inhibition or dyssynchrony of the components of swallowing) (29) and 2) anticholinergic effect, producing impaired esophageal mobility and possibly impairment of the gag reflex (30–34).

The neuroleptic drugs have been shown to block dopamine transmission in several areas of the brain, and it has been strongly suggested that the antipsychotic action of these drugs is related to their ability to block transmission at dopamine synapses in the limbic forebrain and cortex (1). In addition, several studies (1, 35, 36) have quantified differential antimuscarinic anticholinergic effects for these drugs. The tricyclic antidepressants possess antimuscarinic anticholinergic effects in varying degrees (1, 36). The antiparkinson drugs are more potent anticholinergics than neuroleptics or tricyclic antidepressants but are given in smaller doses.

Since all three categories of drugs possess one or both of these actions and since they are commonly combined in psychopharmacologic treatment, there is a possibility of synergy either as a result of multiplying the same effect (e.g., anticholinergic) or adding different effects (e.g., dopamine blocking plus anticholinergic) on the swallowing mechanism. All patients who choked to death were receiving at least one neuroleptic (dopamine blockade), and 71% were receiving more than one drug with anticholinergic properties. Of the other 4 patients who choked, 2 were taking single neuroleptics considered to have relatively potent anticholinergic properties (thioridazine and chlorpromazine), while the other 2 were receiving neuroleptics with less pronounced anticholinergic properties but had additional factors that may have been implicated in the death (1 patient who suddenly collapsed had a history of seizures, while the other had a history of rapid eating). Of interest is the fact that only 3 of the 14 patients had a history of choking, eating problems, or vomiting.

Although caution must be used in the interpretation of these findings, especially since the use of multiple anticholinergics among all asphyxia patients did not differ significantly from that of the controls, these findings are provocative and cannot be dismissed. The findings are consistent with the hypothesis that, in some patients in which a dopamine blockade is coupled with a strong anticholinergic effect, choking may result, with subsequent sudden death. Since these findings have theoretical logic, further work is indicated to identify whether this combination of drug effects is associated with absence of the gag or swallowing reflex in a living population. To complicate the picture, an earlier study (37) suggested that schizophrenic patients may have an impairment of the swallowing mechanism irrespective of whether they are receiving drugs. Among the patients who choked to death, 12 of the 14 were diagnosed as schizophrenic, compared with only 60% of the total cases.

Whatever the underlying pathologic mechanism, another finding with immediate therapeutic and pre-

ventive implications emerged from the data. All choking deaths occurred in the first six years of the nine-year observation period. None occurred after 1974, whereas from 1 to 4 had occurred annually during the previous six years. In attempting to explain this observation, two possible reasons emerged.

First, a drug exception system was instituted at Rockland Psychiatric Center in late 1974 (22). As part of the system's monitoring capacity, letters regarding "inappropriate polypharmacy" were sent to hospital physicians whenever one of their patients received a combination of medications deemed inadvisable on a routine basis (special exceptions to every rule were acceptable if adequately justified). The use of dopamine blocking agents in combination with anticholinergic agents and the use of multiple drugs of one class were both considered exceptions for which justification was requested from the treating physician. A report of this system (22) documented a sharp drop in polypharmacy from a high of over 20% of drug orders in October 1974 to a low of less than 5% in June 1977. Over half of this reduction occurred in 1974–1975.

In addition, the first report describing the Heimlich maneuver as treatment for acute episodes of choking was published in 1975 (38). Anecdotally, several patients who ordinarily would have died from choking have been saved in this hospital using this procedure, an observation that parallels the success of the maneuver in other populations (38, 39).

Thus although there may be theoretical support for the speculation that a combination of drugs which produces a strong dopamine blocking and anticholinergic effect may impair the swallowing mechanism and, in a few cases, result in death by choking, this mechanism of death appears to have been virtually eliminated from our facility through the institution of two procedures, each of which is directed at a specific aspect of the swallowing mechanism.

Since many deaths in both the general population and hospital patients are not immediately recognized as being due to aspiration of food, the Heimlich maneuver should be routinely applied to all people found in a moribund condition regardless of whether evidence of aspiration is present. This should be undertaken before other cardiopulmonary resuscitation is attempted, since, as Heimlich (39) pointed out, traditional cardiopulmonary resuscitation will tend to cause aspirated material to become more firmly lodged and can make resuscitation less likely. Finally, work needs to be done to explore the possible presence of impairment of the swallowing mechanism among the living (both patients and nonpatients) as a first step toward identifying people at risk of choking and instituting appropriate preventive measures.

The largest category of deaths in this series, representing almost 40% of the cases, involved a heterogeneous group of circumstances, including vomiting and infection. The only common denominator among these deaths was the presence of preexisting serious medical illness and deterioration in over three-quarters of the patients. None of the drug categories was even moderately associated with these deaths, suggesting that drugs apparently play no role in this general category of deaths, although the possibility of a drug mechanism in an individual case can never be completely ruled out.

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A Comparative Study of Borderline Patients in a Psychiatric Outpatient Clinic

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The authors applied a list of operational criteria to all new patients in a large psychiatric outpatient department over a one-year period. A randomly selected group of patients clinically diagnosed as having borderline personality disorder was compared with groups with the diagnoses of schizophrenia, neurosis, and nonborderline personality disorder. The group with borderline personality disorder was significantly different from the other groups according to the criteria, especially on items reflecting impulsivity, affectivity, and overvaluation/derogation of others. Impaired reality testing proved a useful distinguishing criterion as well and should be retained as part of the borderline concept.

There is considerable interest currently in the definition and treatment of patients labeled "borderline." A voluminous literature has accumulated since Stern (1) popularized the term some 40 years ago. However, the term is still considered lacking in descriptive clarity. In a 1978 review of the literature, Perry and Klerman (2) remarked that the different criteria used for the borderline diagnosis represent virtually "the whole range of psychopathology of personality." Although few studies of borderline patients in the past have incorporated specific criteria for the selection and diagnosis of patients, reports of more systematic investigations have begun to emerge in the literature (3–6).

Grinker and Werble (7), in their work with borderline inpatients, are frequently cited as the first investigators to bridge the gap between theoretical formulations and descriptive-observational data. On the basis of empirical findings, they concluded that the borderline syndrome can be distinguished by specific clinical features, i.e., structural defects of the ego, disturbances in object relations, episodic and tenuous impulse control, and failure in the formation of a consistent self-identity. Unfortunately no control groups were used in their extensive inpatient studies, and the "border" with the neurotic domain was barely examined at all.

Other authors (8–11) have reviewed extensively the literature on borderline patients to identify commonly cited clinical characteristics. These characteristics include intense affect, a history of impulsive behavior, identity disturbance, brief psychotic experiences, and impaired interpersonal relationships. In addition, Carpenter and associates (4) conducted follow-up studies of matched groups of borderline and schizophrenic inpatients from which they concluded that the borderline group could not in any way be considered a subtype of the schizophrenic sample.

To further refine distinguishing characteristics, Gunderson and Kolb (5) identified a discrete list of variables capable of distinguishing borderline from both schizophrenic and neurotic inpatient groups. Outpatient groups, however, have not yet been used, although they were included in a survey of clinicians' practices recently completed by Spitzer and associates (6). On the basis of their survey these authors proposed that the borderline concept encompassed a heterogeneous group with at least two subgroups. They constructed two sets of items that seemed to tap the most salient features described in the current literature. One set of items represented a dimension comprising "a constellation of relatively enduring personality features of instability and vulnerability." This set was initially labeled the "unstable personality" item set and subsequently designated "borderline personality disorder." The other dimension included stable psychopathological characteristics, such as magical thinking and recurrent illusions, that were "genetically related to a spectrum of disorders which include chronic schizophrenia." The latter group was designated "schizotypal personality disorder." Although Spitzer and associates presented their proposal as a tentative operational definition of the borderline diagnosis, others (e.g., Rosenthal [12]) have objected to the distinction they drew because of the substantial overlap between the two groups.

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The present study is an attempt to further clarify and validate the borderline diagnosis among psychiatric outpatients. Both Kernberg and Gunderson have emphasized the need for further research on borderline patients in an outpatient setting. An outpatient group is clinically pertinent because most borderline patients are currently treated in this setting.

Underlying this investigation are the following propositions: 1) the use of operational criteria improves diagnostic reliability, 2) if the diagnostic criteria are effective in identifying a reasonably homogeneous population, good separation between the index population (borderline patients) and other broad diagnostic groups (schizophrenia, neurosis, nonborderline personality disorders) will be achieved, and 3) if such separations can be found, the boundaries of the borderline conditions and possible subtypes will more clearly emerge, making further validity studies possible, e.g., in terms of treatment response, course of illness, and etiological investigation.

METHOD

We first devised a symptom checklist. Following a review of the literature, we identified a configuration of clinical features in 6 broadly defined critical categories: 1) thought, 2) feelings and impulses, 3) identity, 4) neurotic symptoms, 5) sexual behavior, and 6) interpersonal relationships. On the basis of this overview we constructed a number of brief clinical descriptive items in each category and distributed these to senior attending staff at the outpatient service of the Department of Psychiatry, Albert Einstein College of Medicine, Bronx Municipal Hospital Center. The program at this center has been described in detail elsewhere (13).

The clinical relevance of the descriptive items was rated by a group of approximately 20 senior practicing clinicians. The criterion for acceptability of any single item was based on the clinicians' ability to independently verify its repeated observability in their own patients whom they considered "borderline." In the final construction of the checklist, concepts such as "splitting" appeared too abstract and were replaced by more behavioral consequences such as "tendency to overidealize and excessively derogate other people." Full concordance was reached on 16 items, and the resulting questionnaire, titled "Symptom Checklist," was incorporated into the routine evaluation completed by third-year postgraduate residents for every new patient entering the outpatient clinic over a one-year period (see appendix 1). The clinic population consisted largely of people from socioeconomic classes III and IV (Hollingshead scale) and was geographically quite stable.

The standard format at the clinic provided that every second patient evaluated by a resident was seen with a senior staff supervisor in attendance. Both clinicians discussed the findings and the diagnosis briefly. All cases were then reviewed in supervision with different senior staff who had not seen the patient before. The residents completed the symptom checklist after this review process along with other routine chart information forms, including a standard evaluation summary and other symptom indexes. Residents and clinical supervisors were not aware of the study hypotheses.

During a one-year period the symptom checklist was a routine part of the evaluation of all clinic patients. At the end of the year these forms were removed from the charts and the assigned clinical diagnosis was noted. Forty-five patients with the diagnosis of borderline personality disorder were randomly selected from the clinic population for study purposes. Thirty patients were randomly selected from each of the following other diagnostic categories: schizophrenia, nonborderline personality disorders, and neurotic disorders.

The criteria of Carpenter and associates (14) were used for the diagnosis of schizophrenia; 6 or more items were required. *DSM-II* criteria were used for nonborderline personality disorders and neurotic disorders. In the comparison group of patients with neurotic disorders, 63% of the patients had depressive neurosis, but there were no clear examples of major depressive disorders. Use of the criteria of Feighner and associates (15) for the diagnosis of affective disorders ensured against incorrectly diagnosing primary affective disorders as depressive neurosis. Patients given a diagnosis of drug habituation or alcoholism, or those manifesting organic features or retardation, were not included in the study.

As a final check on the reliability of the clinical diagnoses, we devised a brief descriptive listing of criteria for the acceptability of each diagnostic class. Using these guidelines one of us (M.S.), who was blind to the diagnosis, independently reviewed the initial intake "evaluation summary" of all 135 patients. Approximately 10% of the assigned diagnoses were rated as questionable or in conflict. On review of the complete chart information and follow-up notes of these cases, fewer than 5% of the diagnoses were still rated as questionable. Of these, only 2 were from the borderline group. Because the differences were so few, all subjects were retained.

RESULTS

The borderline group was predominantly female (78%) and more frequently divorced or separated than either of the comparison groups. Forty percent of the borderline patients were single, compared with 30% of the neurotic group, 33% of the nonborderline personality disorder group, and 67% of the schizophrenic pa-

TABLE 1
Standardized Coefficients in Comparison of 45 Borderline Patients and 30 Schizophrenic Patients[a]

Symptom (Variable)	Standardized Coefficient
Episodes of pronounced ignoring or distortion of reality	−.718
Episodes of chronic disturbance in impulse management	+.652
Excessively strong impulses and feelings	+.538
Tendency to overidealize and excessively derogate other people	+.466
Limited capacity to tolerate unpleasant affect	+.467
Periodic obsessions and compulsions	−.390
Periodic withdrawal into partial or complete isolation	−.282
Periodic use of projection in presence of impaired sense of reality	−.278
Difficulty maintaining stable sense of self in a variety of external situations and internal mood states	−.190
Periodic phobias	−.154
Unstable sexual behavior (promiscuity)	+.151

[a]The Wilks' Lambda was .360; the group centroid for the borderline patients was +.550, for the schizophrenic patients, −.404.

TABLE 2
Standardized Coefficients in Comparison of 45 Borderline Patients and 30 Neurotic Patients[a]

Symptom (Variable)	Standardized Coefficient
Excessively strong impulses and feelings	+.851
Episodic or chronic disturbance in impulse management	+.841
Limited capacity to tolerate unpleasant affect	+.633
Periodic withdrawal into partial or complete isolation	+.605
Tendency to overidealize and excessively derogate other people	+.577
Difficulty maintaining stable sense of self in a variety of external situations and internal mood states	+.562
Periodic use of projection in presence of impaired sense of reality	+.543
Periodic obsessions and compulsions	−.537
Episodes of pronounced ignoring or distortion of reality	+.408
Periodic abuse of drugs and/or alcohol	+.396
Unstable sexual behavior (promiscuity)	+.356
Episodes of dissociation	+.272

[a]The Wilks' Lambda was .45; the group centroid for the borderline patients was +1.503, for the neurotic patients, +.001.

tients. The mean age of borderline patients was slightly younger (30.5 years) but not significantly different from that of the comparison groups. There were fewer older patients in the borderline and nonborderline personality disorder groups than in either the neurotic or schizophrenic groups.

A discriminant function analysis was used to compare the sympton checklist profile of borderline patients with that of each of the three comparison groups. In addition, the borderline patients were compared with all other groups combined. The standardized coefficients, presented in table form, reflect the relative discriminant power of the individual items, including the relative order of importance of the variables in discriminating the groups (minus weights indicate items predictive of the nonborderline diagnosis). In an effort to compare our outpatient results with the inpatient data of Gunderson and Kolb (5), our findings are presented in a tabular form that is similar to theirs.

Borderline Versus Schizophrenic Patients

Table 1 presents those items which significantly ($p<.05$) discriminated the borderline from the schizophrenic sample. Problems pertaining to the management of impulses, affects, and overidealization/derogation emerged as the most powerful discriminators and were significantly more prevalent among the borderline patients. By contrast, pronounced failure of reality testing was more frequently found to characterize the schizophrenic patients and was the highest predictor of group differences. Manifestations of projection, with impairment in the sense of reality, were also more evident in the schizophrenic sample. The borderline group showed less evidence of well-delineated neurotic symptoms, use of withdrawal, and difficulty in maintaining a stable sense of self in this comparison. The variable of episodes of dissociation failed to differentiate the two groups. On the whole, these findings parallel those of Gunderson and Kolb (5), which indicated that problems in the management of affects, impulses, and interpersonal relationships were the strongest predictors. Further, our results are also consistent with Kernberg's focus on reality testing as critical in the differential diagnosis of borderline and schizophrenic groups (16).

Borderline Versus Neurotic Patients

Table 2 presents those items which significantly ($p<.05$) differentiated the borderline from the neurotic sample (the latter was composed largely of patients with depressive neurosis). The borderline sample displayed more failures in the management of impulses and affects, more unstable sexual behaviors (promiscuity), and more drug and alcohol abuse than the neurotic comparison group. Periodic withdrawal from close relationships and instability in the sense of self were also more common in the borderline group. In contrast to the previous comparison with the schizophrenic sample, where disruptions among the borderline group in reality testing and the use of projection were less prominent, the borderline group showed significantly more disruptions in these areas than the neurotic comparison group. It was only in the area of nonaffective neurotic symptoms, e.g., phobias and obsessions, that the neurotic group were significantly

TABLE 3
Standardized Coefficients in Comparison of 45 Borderline Patients and 30 Patients with Nonborderline Personality Disorders[a]

Symptom (Variable)	Standardized Coefficient
Tendency to overidealize and excessively derogate other people	+.644
Episodes of chronic disturbance in impulse management	+.630
Difficulty maintaining stable sense of self in a variety of external situations and internal mood states	+.627
Excessively strong impulses and feelings	+.556
Limited capacity to tolerate unpleasant affect	+.451
Periodic obsessions and compulsions	−.417
Periodic hypochondriasis	−.356
Periodic abuse of drugs and/or alcohol	+.442
Unstable sexual behavior (promiscuity)	+.353
Periodic use of projection in presence of impaired sense of reality	+.335
Anhedonia or difficulty experiencing pleasure	+.224

[a]The Wilks' Lambda was .314; the group centroid for the borderline patients was +1.200, for the personality disorders, +.111.

higher. Further, it was only in regard to this particular comparison that the variable of episodes of dissociation proved to be a useful discriminator and was found to be significantly more characteristic of the borderline sample. In line with Gunderson and Kolb's data, interpersonal and impulse control difficulties, as well as episodic failures of reality testing and use of projection differentiated borderline from neurotic patients.

Borderline Versus Nonborderline Personality Disorders

Problems in interpersonal relationships, affect, impulse regulation and control, and a cohesive sense of self were more prominent among the borderline patients ($p<.05$; see table 3). This comparison was considered critical because it had not been included in previous controlled studies. Kernberg's very broad concept of borderline personality organization actually includes all of the personality disorder diagnoses contained in the study sample.

Periodic abuse of drugs and alcohol was more frequently found among the borderline group. (Drug-addicted and alcoholic patients were excluded from the study.) Unstable sexual behavior (promiscuity) and episodic reality distortion with the use of projection were significantly less frequent in the group with nonborderline personality disorders than in the borderline sample.

The most prominent diagnoses in the nonborderline personality disorders group were obsessive-compulsive and immature personality disorders. No patients were diagnosed as having paranoid personality. As with the neurotic comparison, phobic, obsessive-compulsive, and hypochondriacal symptoms were less prominent in the borderline patients than in the group with nonborderline personality disorders.

TABLE 4
Standardized Coefficients in Comparison of 45 Borderline Patients and All Other Groups Combined (N=90)[a]

Symptom (Variable)	Standardized Coefficient
Episodic or chronic disturbance in impulse management	+.724
Excessively strong impulses and feelings	+.682
Tendency to overidealize and excessively derogate other people	+.629
Limited capacity to tolerate unpleasant affect	+.544
Periodic obsessions and compulsions	−.437
Difficulty maintaining stable sense of self in a variety of external situations and internal mood states	+.380
Periodic abuse of drugs and/or alcohol	+.361
Unstable sexual behavior (promiscuity)	+.326
Periodic use of projection in presence of impaired sense of reality	+.228
Periodic hypochondriasis	−.215
Periodic withdrawal into partial or complete isolation	+.196
Bizarre sexual fantasies	+.160

[a]The Wilks' Lambda was .433; the group centroid for the borderline patients was +1.170, for the combined groups, +.107.

Borderline Patients Versus the Combined Comparison Groups

Gunderson and Kolb (5) found that a comparison of borderline patients with a combined group of patients with other disorders "sharpened the focus of those characteristics of borderline patients which are the best overall discriminators for use in a variety of differential diagnostic problems." Listed in table 4 are the statement items that significantly ($p<.05$) discriminated the borderline group from all the other patients in our sample. The strongest discriminators were disturbances in the management of impulses, excessively strong feelings, overidealization/derogation of other people, and difficulty tolerating unpleasant affect. Significant but somewhat less prominent in comparison with all other study groups were identity problems, periodic alcohol or drug abuse, social withdrawal, unstable sexual behavior (promiscuity), projection, and bizarre sexual fantasies. On the other hand, indicators of traditional neurotic disturbance (e.g., obsessions, compulsions, and hypochondriasis) were significantly less prominent than in the other comparison groups. These findings are consistent with those of Gunderson and Kolb (5) but contradictory to the original predictions of Kernberg (17). Impulsivity, excessively strong feelings, and interpersonal difficulties are the most powerful predictors in distinguishing borderline patients from the comparison groups.

In a final analysis the sensitivity and specificity of the symptom checklist items in discriminating between the groups were derived at different cutoff levels. A cutoff score of 7 items was found to be most efficient in maximizing both the sensitivity (93%) and the specificity (76%) of the instrument (i.e., the ability to correctly identify borderline patients on the basis of the in-

strument and to differentiate nonborderline from borderline groups).

DISCUSSION

The results of this study, which was based on a psychiatric outpatient population, are consistent with the hypothesis that borderline patients can be significantly and systematically differentiated from other diagnostic groups by the use of refined operational criteria. The reliability of the assigned clinical diagnoses was reviewed by senior supervisors unaware of the study hypotheses in addition to an independent blind review by one of us (M.S.). The best overall discriminators between the borderline and other groups were found in the areas of heightened impulsivity, affectivity, and overvaluation/devaluation of others.

A greater degree of reality distortion, use of projection, and identity confusion was found in the negative direction in the borderline group in comparison with the schizophrenic group. Also in the negative direction was the extent of traditional nonaffective neurotic symptoms in comparison with both neurotic and nonborderline personality disorders. Although the group with neuroses and the group with personality disorders were limited in the range of diagnoses represented, the empirically derived boundaries are consistent with the findings of Gunderson and Kolb (5) and with the implications of Kernberg's theoretical formulations (16). At the operational level, however, the present study was able to demarcate the borderline diagnosis from a group of diagnoses of personality disorder that Kernberg would have included in his own broader borderline rubric. Gunderson and associates have consistently shown that items reflecting interpersonal behavior and impulse action patterns are the best discriminators for comparisons among inpatient groups. The present data extend these findings to an outpatient sample.

With respect to the data of Spitzer and associates (6), the present findings largely parallel those items grouped under the unstable personality item set. Although the development of our checklist did not generate the same item pool Spitzer and associates used to differentiate their subgroups, these authors indicated that over half of their clinically diagnosed borderline patients met the criteria for both subtypes. Futhermore, they stated that lists of descriptive criteria would contain items representative of both subtypes and recommended the use of the combined item set if one is primarily interested in defining the borders of the syndrome. They asserted that the schizotypal and unstable personality dimensions of the borderline syndrome may not necessarily reflect two mutually exclusive types of borderline patients, but they warned that the borderline phenomena would "appear unitary" if the comparison was between borderline and nonborderline patients. Our data analysis fell within this last comparison, although our results appear most consistent with the unstable personality item set, which, as Spitzer and associates (6) noted, has been traditionally referred to as borderline personality in the literature.

Our findings support the continued use of impaired reality testing as a criterion for the borderline diagnosis despite its elimination from the now official *DSM-III*. We doubt the practicality of suggesting, as Spitzer and Endicott did (18), that over half of those patients whom clinicians appear to regard as borderline should receive two simultaneous personality disorder diagnoses, i.e., borderline personality disorder and schizotypal personality disorder. We believe that our findings are more congruent with current clinical consensus and cast doubt on the wisdom of separating borderline and schizotypal personality disorders completely, as *DSM-III* has done. We suggest that further research and replication using external correlates, such as course over time, familial incidence, and treatment response, are needed before this distinction can be made or eliminated with confidence.

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APPENDIX 1
Symptom Checklist

1. Thought: a) episodes of pronounced ignoring or distortion of reality, b) episodes of dissociation, c) periodic use of projection in presence of impaired sense of reality.

2. Feelings and impulses: a) excessively strong impulses and feelings, b) anhedonia or difficulty experiencing pleasure, c) limited capacity to tolerate unpleasant affect, d) episodic or chronic disturbance in impulse management.

3. Identity: a) difficulty maintaining stable sense of self in a variety of external situations and internal mood states.

4. Neurotic symptoms: a) periodic phobias, b) periodic obsessions and compulsions, c) periodic hypochondriasis, d) periodic abuse of drugs and/or alcohol.

5. Sexual behavior: a) unstable sexual behavior (promiscuity), b) bizarre sexual fantasies.

6. Interpersonal relationships: a) tendency to overidealize and excessively derogate other people, b) periodic withdrawal into partial or complete isolation.

A Controlled Study of Self-Help Intervention for Widows

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The authors conducted a 2-year study of postbereavement adaptation in 162 widows. Sixty-eight were paired with a widow contact who provided emotional support and practical assistance. The differences between the women receiving intervention and the controls at 6, 12, and 24 months after bereavement suggested that those receiving intervention followed the same general course of adaptation as control subjects but that the rate of achieving landmark stages was accelerated for the intervention group. The Goldberg General Health Questionnaire and two indices derived from the study questionnaire confirmed the hypothesized "pathway of adaptation" through intra- and interpersonal adaptation to resolution of overall distress and the effectiveness of the intervention.

Conjugal bereavement is among the best documented of life event stressors and has been demonstrated to increase the risk of physical, psychological, and social impairment (1–4). As such, it has become a focus for interventions designed to mitigate some of the associated stress. There are two common modes of intervention: professional intervention, often with predefined high-risk populations (5, 6), and self-help programs, generally based on the widow-to-widow model developed by Silverman and associates (7–9).

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Almost no research has been done on the benefits of widow-to-widow programs (7, 9, 10). One study (10) found that of widows assigned to self-help, confidante, or consciousness-raising groups, those in the self-help groups showed the least improvement. These results are difficult to interpret because of sampling problems, including widely discrepant intervals since bereavement, and the fact that the "self-help" groups were led by professionals and thus did not follow the traditional self-help model.

In the course of a 2-year study of 162 widows conducted by the Social and Community Psychiatry Section of the Clarke Institute of Psychiatry, participants were randomly assigned to either an experimental widow-to-widow program or a control group that received no intervention other than the data-gathering interviews. This paper is a report on the efficacy of the widow-to-widow program.

METHOD

Sample Selection

Seven Toronto hospitals[1] participated in the study and contacted widows of men aged 67 years and younger who died in the hospitals between May 1974 and September 1975. A cut-off of 67 years was chosen to minimize confounding the problems typical of bereavement with those related to aging.

Three hundred and nineteen women met these criteria, but 134 (42%) were not contacted because of unavailability, language problems, or severe disability. The remaining 185 women received letters from the participating hospitals approximately 1 month after their bereavement. This letter acknowledged the great stress of the bereavement process, emphasized the need for further study of the problem, and introduced the project. The women were told that they would be telephoned by a research assistant who would provide details of the project, and, if they consented, an interview would be arranged. The letter stressed their freedom to refuse participation. One hundred sixty-two women (88% of those contacted) agreed to participate in the study.

[1]North York Branson Hospital, North York General Hospital, Princess Margaret Hospital, St. Michael's Hospital, Sunnybrook Hospital, Toronto Western Hospital, and Wellesley Hospital.

Sample Characteristics

The median age of the sample was 52 years; the range was 22 to 69 years. The sample was predominantly middle class on the Hollingshead two-factor index. The majority of the women were Canadian-born (68%) and Protestant (75%); more than half (57%) lived with someone else, usually their children. Twenty-nine percent were employed outside their homes, and 71% were housewives.

Eighty-one percent of the husbands had died of chronic diseases, and their median length of final illness was about 6 months. Forty-five percent of the men died of cancer and 38% from cardiovascular disease. The remaining 17% died from diverse causes, usually unexpectedly.

Procedures

A personal interview was conducted in each woman's home at 1, 6, and 24 months after the husband's death. In addition, an attempt was made to interview one-third of the women at 3 months and two-thirds at 12 months after bereavement. Since it was not always possible to interview the women by 1 month, the first interview took place up to 8 weeks after bereavement. Therefore, very few 3-month interviews were done. The 6-month interview time was allowed to extend to 8 months and the 12-month interview to 14 months. Interviews ranged from 30 minutes to 2 hours, with the mean being slightly more than 1 hour. These follow-up interviews were conducted with 108 women (67%) at 6 months, 76 women (71% of those scheduled for this interview) at 12 months, and 99 women (61%) at 24 months.

There is evidence that those lost to follow-up were a different group than those who remained in the study. The subjects lost to follow-up had younger husbands and were of a lower social class. They seemed to have more psychological problems and fewer social supports than the women who stayed in the study. From these observations it seems reasonable to assume that any effects due to attrition would bias the results in the direction of underreporting difficulties during bereavement. There were no statistically significant differences between intervention and control groups in attrition rates or characteristics of subjects lost to follow-up.

Following the initial interview, the widows were randomly assigned to either the intervention group (N=68) or the control group (N=94). We did contingency analyses using chi-square tests to investigate possible intervention versus control group differences on such sociodemographic and prebereavement variables as age, socioeconomic status, and length and quality of marriage. Not surprisingly, it was found that our groups reaffirmed Levy's Ninth Law, "Only God can make a random selection" (11). There were three significant differences between intervention and control subjects. Those assigned to the intervention program were more likely to identify cancer as the cause of the husband's death ($p \leq .05$), were more likely to report having at least two people they could count on to understand how they were really feeling ($p \leq .05$), and were more likely to report talking to the husband as though he were still alive ($p \leq .01$). We took these differences into account in analyzing outcome differences between intervention and control groups.

Only 7 women assigned to the intervention group refused to participate. These 7 subjects were homogeneous with respect to age, social status, type of death, and distress profiles. Although this homogeneity is quite striking, the number of subjects involved is so small that it must be treated as a coincidence. Certainly, one cannot use it to support theories on the appropriateness of the intervention model for different populations. Nevertheless, an equivalent, matched set of subjects was excluded from the control group for all comparison analyses.

Measures

Although a number of measures were used with this population, this paper will concern itself with only two. The Goldberg General Health Questionnaire (GHQ) (12) is a well-validated psychiatric screening instrument originally designed to indicate the likelihood that an individual with a given score would be considered a nonpsychotic psychiatric case if interviewed by a psychiatrist. In subsequent studies (13, 14) the GHQ was highly correlated with independent clinical assessment and with scores on an early 35-item version of the SCL-90 ($r=.70$ and $r=.78$, respectively). Scores on the 30-item version range from 0 to 30, with a score of 5 commonly used as a threshold of caseness. For the purposes of this study and in the context of bereavement, the GHQ was used as an indicator of overall disturbance (rather than caseness), with scores of 0–4 designated "low distress," and scores of 5 and above, "high distress."

We constructed a questionnaire/interview to sample factors potentially associated with reaction to a psychosocial stressor (as, for example, in references 1, 3, and 4), i.e., demographic/situational variables, social support system information, and some psychological and psychophysiological variables. Objective data with little emotional loading were obtained by a self-administered questionnaire filled out in the presence of the research assistant. A structured interview was used for more sensitive data such as stress of husband's illness, history of discussion of death with husband, and past and current stressors.

Intervention Program

Those in the intervention group received a letter from a "widow contact," 1 of 6 women who had resolved their own bereavement reactions and had participated in a training seminar that examined problems

of bereavement, provision of supportive counseling, and the spectrum of community resources likely to be helpful to new widows. Intervention was not limited to any predefined duration or phase of bereavement; rather, the widow contacts were available to the widows for as long as they were needed. Initially, they offered one-to-one support as needed, including practical help in locating community resources, supportive telephone calls, and face-to-face interviews. Eventually small group meetings were made available as well.

Unlike most professional programs of intervention, our program allowed contact to be initiated by either the widow contact or the widow. This design acknowledged the fact that newly widowed women are often quite passive and unable to reach out for help yet need to know that help is readily available when they need it most.

RESULTS

Several items on the GHQ differentiated the intervention and control group subjects at 6, 12, and 24 months (chi-square tests; $p \leq .05$ unless otherwise noted). Most of the differences at 6 months related primarily to intrapersonal adaptation. Among the 67 subjects in the high-distress group (GHQ score of 5 or more), the women who received intervention were significantly more likely to perceive their health as better than average, more likely to feel "better" than they did at the time of their husband's death, less likely to be seeing old friends as much as usual, and less likely ($p \leq .01$) to anticipate difficulty adjusting to widowhood. In the total group of subjects, the women who received intervention were significantly less likely to miss their husband as a sexual partner.

At 12 months the differences related more to interpersonal adaptation. The intervention group was significantly more likely to feel "much better" than at the time of their husband's death, to have made new friends, and to have begun new activities and less likely to feel anxious "often" or "almost always" or to need to keep up a front rather than express true feelings. At 24 months, the high-distress subjects who had received intervention were significantly more likely to have shifted to the low-distress group than high-distress subjects who had not received intervention.

As noted earlier, the intervention and control groups also differed on 3 items at 1 month after bereavement. Therefore, contingency analyses were used to determine the degree to which the subsequent differences were related to these 3 initial differences. In brief, neither "cause of husband's death" nor "talking to husband as though alive" showed any consistent interactional effects with subsequent group differences. However, for the 67 women who fell into the high-distress group at 1 month, "number of people to count on" did seem to be strongly related ($p \leq .02$) to GHQ score at 24 months. Indeed, such an effect is entirely consistent with the theory of this type of intervention, namely, that increasing the support available to a woman following bereavement will favorably influence her adjustment. However, it was necessary to control for this difference between the intervention and control groups in "number of people to count on." We did a three-way analysis, which demonstrated that the effects of this factor and participation in the widow-to-widow program were additive rather than interactional; that is, the subjects with neither tended to do worst and those with both did best ($\chi^2 = 10.54$, $p \leq .01$) (see table 1).

TABLE 1
Distress Level at 24 Months for 67 Widows Who Had High Distress Levels[a] 1 Month After Bereavement

	Low Distress		High Distress	
Social Support Level	N	%	N	%
Fewer than 2 people to count on				
Intervention group	1	2	3	5
Controls	3	5	7	10
2 or more people to count on				
Intervention group	21	31	5	8
Controls	16	24	11	16

[a]Score of 5 or above on the Goldberg General Health Questionnaire (12).

The nature of the items on which the groups differed at 6, 12, and 24 months seemed to fall into three categories, indicating that the women receiving intervention were proceeding along a hypothetical pathway of adaptation faster than the control subjects. On this pathway, the inner turmoil, withdrawal, and preoccupation with loss and the past would gradually abate, as shown by the intrapersonal nature of the items that differentiated the groups at 6 months. This resolution would proceed to the initiation or resumption of socialization patterns (interpersonal items) and finally lead to a reduction in overall psychiatric disturbance as indicated by fewer symptoms in the areas tapped by the GHQ at 24 months.

Because this hypothesis was an outgrowth of the results of the study, rather than a basis for research design, the capacity of the data to test this was limited. Nevertheless, sufficient data were available to develop the following design.

The description of a "pathway of adaptation" requires both consistent signposts along the way and a consistent group of subjects to follow. The signposts were derived by comparing the 1-, 6-, 12-, and 24-month questionnaire/interviews. We examined all the questions that had been asked every time. From these we selected two categories of questions: those unambiguously related to the first signpost—intrapersonal adaptation as described above—and those related to the second signpost—interpersonal adaptation. The

TABLE 2
Percent of Widows in Intervention and Control Groups Who Showed Positive, Negative, or No Change[a]

Index[a]	Months After Bereavement	Percent of Widows: Intervention Group (N=24)	Percent of Widows: Control Group (N=38)
Intrapersonal adaptation	6		
Negative change		8	24
No change		38	24
Positive change		54	53
Interpersonal adaptation	12		
Negative change		0	10
No change		8	24
Positive change		92	66
Goldberg General Health Questionnaire[b]	24		
High-high		17	26
Low-low		25	40
High-low		58	34

[a]Since 1 month after bereavement.
[b]Levels at 1 and 24 months.

GHQ measured the third signpost—overall disturbance.

We then tested the items in the categories of questions designating the first two signposts for internal reliability. For each category, the items yielding the best reliability tests were grouped to form an index of the degree to which the respective stages of adaptation had been attained. The intrapersonal adaptation index had an alpha level of .61 and included the following items: health in the past month; anxiety in the past month; psychiatric counseling in the past month; a sense of husband's presence in the past month; missed husband as a sexual partner in the past month; use of alcohol in the past month; use of tobacco in the past month; use of prescription sleeping pills, antidepressants, or tranquilizers in the past month; use of nonprescription drugs in the past month; and how feeling now compared to immediately after husband's death. The interpersonal adaptation index had an alpha level of .34 and included the following: difficulty carrying on social activities alone in the past month; need to keep up a front instead of expressing true feelings in the past month; made new friends since husband's death; and initiated new activities since husband's death. For both indices, the higher the score, the better the adjustment.

In order to have a consistent group of subjects to follow, only the 62 women who completed all four interviews were included in this analysis. Of these, 24 women received intervention from the widow contacts and 38 women were in the control group.

There were no significant differences between intervention and control groups at 1 month following bereavement on either index or on the GHQ. For 6, 12, and 24 months following bereavement, each woman was used as her own control by expression of the index scores as difference scores; that is, for each woman, the score at 1 month was subtracted from the follow-up scores. Three categories of scores were thus derived: negative change, no change, and positive change. For the GHQ, the same technique was used after dichotomizing the 1-month scores into "low-distress" and "high-distress" categories. Because virtually no one in the study went from low distress at 1 month to high distress at 24 months, GHQ follow-up results were expressed as "high-low" (a shift from 1-month high distress to 24-month low distress), "high-high," and "low-low."

Table 2 presents the results on the two indices and the GHQ for the times when the differences between the intervention and control groups were the greatest. For the intrapersonal adaptation index and the GHQ, the times shown (6 months and 24 months, respectively) are the only times when there was any appreciable difference between the intervention and control groups. For the interpersonal adaptation index, the results at 24 months still showed a larger positive change in the subjects who received intervention than in the controls (96% versus 68%). Because of the small numbers in each category, percentage differences are more illustrative of the overall pattern than statistical significances, even though the latter can be obtained by choosing various category groupings.

There still remained the question of what effect the differences between intervention and control groups at 1 month might have had on these outcome indices. For the 62 women who completed all 4 interviews, there was no statistically significant difference in the three items (cause of husband's death, number of people to count on, still talking to husband) between the two groups. In fact, for the item concerning type of death, the distribution of responses in intervention and control groups was virtually identical. However, the remaining two items showed the same direction and magnitude of group differentiation for the subsample as for the entire sample. Therefore, despite the lack of statistical significance, the effect of these items on the three outcome measures was examined.

For each variable, the effects on the outcome indices for this smaller group mimicked the results for the full group. That is, "still talking to husband" at 1 month showed no consistent interactional effects with any of the three indices. "Number of people to count on" maintained its additive effect for the 24-month GHQ.

To look specifically at high-distress subjects, the women with 1-month GHQ scores of 5 or above were analyzed separately. The results of these analyses suggested that intervention versus control differences were most pronounced for the high-distress subjects. We will document the extent of this effect and its implications for screening subjects for intervention in a future paper, which will incorporate extended group data.

DISCUSSION

The demonstration by epidemiological methods of a "pathway of adaptation" and of the accelerated progress of widows who participated in a self-help group is sufficiently important to warrant replication using indices designed from the beginning to test the hypotheses. In the present study, the intervention and control subjects seemed equally upset at 1 month after bereavement. The first change was a negative one in the control group. As immediate postbereavement support diminished and the reality of the loss was realized, even more widows manifested depressive symptoms and preoccupation with the past. However, the intervention group resisted this deterioration of emotional status. At 12 months the controls had caught up in intrapersonal adaptation but the intervention group was clearly ahead of them in the resocialization process—a lead that was maintained through the following year. At 24 months there were substantially more women in the intervention group who showed a drop in GHQ scores from the high- to low-distress range.

Such a model of adaptation has some support in the work and theories of C.M. Parkes. Parkes has described reaction to bereavement as ". . . a process of realization, of making real inside the self events which have already occurred in reality outside" (15). Parkes described this process as having both internal and external orientations, as both the preservation and protection of the "damaged self" and the revision of a personal working model of the world. Parkes further noted that during this process the individual experiences in turn anxiety, frustration, and depression before reaching a point where he can satisfactorily reinvest in external objects. Intrapersonal adaptation, which was more pronounced in our experimental group at 6 months, presumably facilitates the next phase of adaptation, in which the individual begins to look outward and make attempts to develop new roles and activities. This socialization process became significantly different from the control group by 12 months. In this paradigm, it is only by progressing through these stages of inward-directed and outward-directed adaptation that a significant decrease in overall disturbance and dysfunction is achieved. In this study the attainment of these stages of adaptation was accelerated by participation in the widow-to-widow program, which provided emotional, cognitive, and practical support to the individual.

What is perhaps most surprising about this progression is not the nature and sequence of the stages but the length of time over which it extends. Not until 2 years after bereavement did a difference in overall disturbance between intervention and control groups become apparent. From Lindemann's original description of adaptation (2) in terms of months to Caplan's later discussion (16) in terms of a year or more, the estimate of the duration of adjustment to conjugal bereavement has been steadily extended. This must affect theories of loss and the grieving process and, also, research on the significance of life events in the etiology of illness. Life event questionnaires investigating the last 6 months to 1 year of a respondent's life may be underestimating the continuing disturbance present as a result of a less recent major event such as conjugal bereavement.

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Predicting Which Patients Will Be Discharged Against Medical Advice: A Pilot Study

BY PETER STEINGLASS, M.D., CHARLES E. GRANTHAM, M.A., AND MARC HERTZMAN, M.D.

Studies of patients signing out against medical advice (AMA) from a psychiatric service have been retrospective and inconsistent or contradictory in their findings. The authors report findings from a prospective study in which data from a brief structured interview of patients at the time of admission were used to predict subsequent AMA behavior. They achieved 80% accuracy. Critical variables centered around the nature of the "treatment contract" as perceived by the patient: AMA patients never intended to stay in the hospital for more than a few days.

A patient's signing out of a hospital against medical advice (AMA) is not a routine event on any medical service. When an AMA occurs on a psychiatric inpatient service, it has traditionally been interpreted as a reflection of a profound breakdown in the therapeutic relationship between treatment staff and patient. The AMA event has also been viewed as having a sizable impact on the morale and behavior of other patients as well. Consequently, signing out against medical advice has received considerable attention in the psychiatric literature (1).

Our reading of this literature has led us to two general conclusions. First, retrospective designs have been largely unrewarding in studying the troubling question of psychiatric AMAs because of the inconsistency of their findings (2–5). Second, AMA behavior seems to be influenced primarily by factors occurring either at the time of admission or early in the course of hospitalization (6–10). This second conclusion suggests that AMA behavior, rather than reflecting a breakdown in the therapeutic relationship, reflects a breakdown or disagreement in the contracting process that occurs early in the course of hospitalization. These two conclusions, if correct, indicate that future studies of factors associated with AMA behavior should include, at the very least, an emphasis on prospective rather than retrospective designs and the inclusion of data about the initial contracting process at the time of hospitalization.

In this paper we report findings from a study of AMA behavior that followed both these guidelines. We carried out a prospective study on the psychiatric service of a university hospital. Systematic data were collected from all patients admitted to this service for a six-month time period. In this report we will focus on two aspects of the study: 1) the strategy used to develop an assessment instrument intended to have predictive validity regarding AMA behavior and 2) data indicating that this instrument was highly successful as a predictive discriminator of patients who will versus those who will not leave the hospital AMA.

METHOD

The basic strategy of the study was to administer within 48 hours of admission a series of data collection instruments to all patients admitted to a psychiatric inpatient service and to compare these data with outcome data obtained during the course of hospitalization. For the purposes of the study, an "AMA event" was defined by two criteria: 1) a patient leaves the hospital and 2) a physician or nurse places in the hospital record a written statement that the patient was advised to remain in the hospital.

The study was carried out on the psychiatric service of the George Washington University Hospital during July–December 1978. Of the 132 patients admitted to the unit during this period, 115 agreed to participate in the study (all subjects signed an informed consent form before being included in the study). We collected enough data on 96 of the 115 subjects to include them in some part of the final data analysis.

The Psychiatric Service

Our psychiatric service is a 33-bed unit in a general medical hospital staffed by the customary range of mental health professionals. The unit serves as a major training base for psychiatric residents and admits, on a voluntary basis, an urban population of patients agreeing to hospitalization for a variety of psychotic condi-

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tions and character disorders. The average length of stay on the unit is 17 days. Most patients are admitted to the unit through the hospital emergency room, one of the few 24-hour psychiatric emergency rooms in the District of Columbia. The unit does not knowingly accept involuntary admissions or patients lacking financial resources (Medicaid patients are accepted, however).

Each patient is assigned to an attending psychiatrist. The psychiatrist not only conducts some therapy—individual, group, or both—with the patient but also supervises a resident working with the patient. Each patient is assigned a nurse or psychiatric technician on each 8-hour shift. Ordinarily, there are three to five nurses or technicians on duty per shift. Medical, occupational, and other therapists, social workers, and other students also work with patients five days a week.

On average, each patient is treated in individual dynamically oriented therapy at least twice a week. All patients are also in various group therapies at least once a day and generally more often. Pharmacotherapy is used as required (a majority of patients receive some form of medication); ECT is seldom used.

AMA Profile Checklist

Because of our interest in studying the relationship between the contracting process at the time of hospital admission and subsequent AMA behavior, data collection initially centered around a series of structured interviews and questionnaires intended to elicit treatment goals for the hospitalization from the patient, the patient's family, the admitting physician, and a member of the nursing staff.

After an initial period of data collection, we performed a preliminary review of data collected from 6 patients who had signed out AMA. Two findings stood out: first, all AMA patients had left the hospital within the first seven days of admission; second, AMA patients, in comparison with non-AMA patients, seemed remarkably uninvolved in the treatment community. Our review also suggested that a series of attitudinal factors were associated with AMA behavior, including the patient's perception that he or she had been coerced into accepting voluntary admission, a concern over lost income, a concern about being labeled "sick," a concern over the "unduly restricted" regimen of the ward, an expressed ambivalence toward the therapeutic regimen, and requests for noninvolvement of family members in treatment.

A review of available data obtained from the admitting psychiatrist, the psychiatric staff, and family members failed to reveal any consistent patterns associating AMA behavior with such variables as type of treatment plan suggested, assessment of level of functioning, and identity of primary therapist.

On the basis of our initial review, we designed a screening instrument intended to serve as a predictor of AMA behavior. The instrument, which focused exclusively on data obtained from the patient, was a 5-item, Likert-scaled[1] AMA Profile that could be filled out by the research associate (C.E.G.) immediately following a structured admission interview of the patient, using criteria explicated in a scoring manual.

The five items were 1) part patient played in admission decision, 2) patient's desire for involvement in therapeutic process, 3) estimate by patient of length of hospital stay, 4) activities external to ward that would prevent patient from staying, and 5) amount of "help" patient felt he or she could receive from staff.

For the subsequent period of data collection all patients admitted to the psychiatric service were interviewed and scored on this profile. (A copy of the scoring manual is available from the authors on request.) Patients admitted before the development of the instrument were retrospectively scored using hospital charts edited to disguise the patient's identity. The success of the AMA Profile as a predictor of AMA behavior could then be assessed by comparing the hospital course of patients admitted during the six-month time period of data collection with the scores they were given on the basis of a structured interview carried out within 48 hours of admission.

We also administered the Rotter internal/external locus of control scale (11). This measure, it might be argued, reflects a dimension of psychological functioning that one would predict might be associated with a predilection to sign out of the hospital AMA (i.e., internal locus of control patients would be more likely to initiate such behavior).

RESULTS

Comparison of AMA and Non-AMA Patient Groups

Table 1 summarizes a comparison of our AMA and non-AMA patient groups along selected demographic and questionnaire variables that might be proposed as patient-related predictor variables of AMA potential for such a patient population.

Three findings stand out. First, there were no differences between the AMA and non-AMA groups in any of the demographic and social class variables examined, with the single exception that the non-AMA group was a more heterogeneous group in level of education (see table 1).

Second, there was no difference between the two groups in the Rotter internal/external locus of control score. Thus our hypothesis was not borne out by our data.

Third, by way of contrast, two AMA Profile variables were significantly different for the two groups, and a third AMA Profile variable showed a trend to-

[1]Likert-scaling requests the respondent to circle a number that most reflects his or her feeling about each item in the questionnaire. We used a 7-point scale with 1 defined as "most agree" and 7 defined as "most disagree."

TABLE 1
Characteristics of Psychiatric Patients Who Signed Out of the Hospital Against Medical Advice (AMA) and Those Who Did Not (Non-AMA)

Characteristic	AMA Patients			Non-AMA Patients			t	Significance
	N	Mean	SD	N	Mean	SD		
Age (years)	20	32.45	11.31	73	31.40	13.31	.35	n.s.
Sex (male=1, female=2)	20	1.55	.51	74	1.58	.50	.24	n.s.
Education (years)	20	14.15	2.74	75	13.85	7.26	.29	n.s.
Race (white=1, nonwhite=2)	20	1.60	1.23	75	1.36	.56	.85	n.s.
Number of previous hospitalizations	19	1.05	1.78	74	.78	1.40	.61	n.s.
Rotter score[a]	20	5.60	5.07	75	7.12	6.47	−1.12	n.s.
AMA Profile scores[b]								
Item 1	20	2.90	1.37	68	2.43	1.85	1.24	n.s.
Item 2	20	2.90	1.12	66	2.50	1.21	1.37	p=.178
Item 3	20	4.30	1.08	68	3.56	1.38	2.52	p=.016
Item 4	19	2.05	1.96	55	1.53	2.16	.98	n.s.
Item 5	20	3.15	1.23	64	2.20	1.30	2.97	p=.005

[a]A lower score indicates a more internal locus of control.
[b]A higher score indicates that the patient is more likely to sign out of the hospital AMA.

ward being different. The two groups differed significantly on the third item, the patient's estimate of his or her length of hospital stay (see table 1). AMA patients indicated at the time of admission that they would be staying in the hospital only a few days; non-AMA patients gave a much longer estimate of their anticipated length of stay. The groups also differed significantly on item 5, the patient's perception of the amount of help he or she would be receiving from the hospital staff (see table 1). AMA patients indicated their feeling that the staff would be of little assistance to them in helping them with their chief complaint; non-AMA patients were more optimistic on this score. This difference between the two groups in their perceptions of what help they could anticipate from the hospital staff was consistent with the trend for AMA patients to express less interest in becoming involved in a therapeutic process while on the ward (item 2 of the AMA Profile).

AMA and non-AMA groups were also compared along variables having to do with hospital structure rather than patient characteristics. For example, we asked, Did the identity of the psychiatric resident or the treatment team to which the patient was assigned influence the patient's predilection to sign out AMA? Was the AMA rate influenced by the time of year the patient was admitted (the treatment unit is a teaching service in which all house staff begin one-year rotations on July 1)? Was the identity of the admitting physician of any importance? Were some physicians producing distorted expectations of patients' hospital stays by the way they described the treatment service at the time of admission, and was this "false advertising" leading to an increased incidence of AMA behavior?

Although our sample size precluded inferential statistical analyses of these variables, a visual inspection of the data indicated no differences between the AMA and non-AMA groups regarding identity of primary therapist, date of admission, or identity of the admitting physician.

The AMA Profile as a Predictive Instrument

To answer the question of how accurate a predictive instrument the AMA Profile was as applied to the patient sample prospectively studied in this project, we subjected the data to a discriminate function analysis. This analysis is a multivariate procedure that deals simultaneously with all AMA Profile variables and produces a mathematically derived linear function that maximally discriminates between the independent variables (in our case AMA versus non-AMA groups).

Having determined the best coefficients and constants for the discriminant function, the procedure then computes a discriminant function score for each patient in the sample and uses this score as the basis for making a prediction about which group the patient belongs to. It is this last step we were particularly interested in.

We carried out a series of discriminant function analyses (12) on our patient sample with such a prediction in mind. An initial analysis was performed on the entire sample for whom complete AMA Profile scores were available (for 20 AMA patients and 64 non-AMA patients). Using this discriminant function as the basis for classifying cases, we found that the analysis correctly classified 80.4% of the 84 cases in the sample. However, most of the misclassified cases were AMA patients. Because of the uneven size of the two patient groups, the discriminant function was calculated to best predict patients in the larger patient group, the non-AMA patients. However, it is the AMA patient that we want to correctly identify at the time of admission. Therefore, a second series of analyses was performed using the same dependent variables, but this time we used matched sample sizes of 20 AMA patients and 20 non-AMA patients. The latter were randomly selected from our total sample of 64. Ten such analyses were separately performed, each time using a different random sample of non-AMA patients. Table 2 summarizes the results of the classification process us-

TABLE 2
Classification of Patients by Discriminant Function Analysis Using AMA Profile Scores

Sample	AMA Patients		Non-AMA Patients		Overall Correct Classification Rate (%)
	Correctly Classified	Incorrectly Classified	Correctly Classifed	Incorrectly Classified	
1	16	4	13	7	72.5
2	11	9	9	11	50.0
3	16	4	12	8	70.0
4	16	4	11	9	67.5
5	16	4	12	8	70.0
6	16	4	13	7	72.5
7	16	4	13	7	72.5
8	16	4	12	8	70.0
9	16	4	13	7	72.5
10	16	4	11	9	67.5

ing these 10 different discriminant functions. The correct classification rate was remarkably consistent with the sole exception of sample 2. As we hoped, when the discriminant functions were derived from AMA and non-AMA groups of equal size the resultant discriminant function did an excellent job of correctly classifying potential AMA patients from their AMA Profile scores (16 of 20 AMA patients were correctly classified for 9 of the 10 trials).

DISCUSSION

Distinguishing characteristics of the AMA patient. The findings of this study clearly indicate the feasibility of developing a simple questionnaire that has predictive validity in identifying potential AMA patients at the time of admission. The brief 15-minute interview used for this study was capable of providing data that, when translated into AMA Profile form, acquired 80% predictive accuracy for future AMA behavior.

The two items in the AMA Profile that proved to be the most significant discriminators of AMA versus non-AMA patients strongly suggest that potential AMA patients view the hospital not so much as a treatment resource capable of providing them with help in dealing with critical psychological and practical problems in their lives but, rather, as a temporary way station. Despite their willingness to sign into the hospital on a voluntary basis (often under pressure from medical staff and/or family members), potential AMA patients seem to enter the hospital with a profoundly different view of the treatment "contract" they have just signed than the view held by the treatment staff. The assumption on the part of the treatment staff that the patient's willingness to sign a voluntary hospitalization form is a reflection of his or her interest in receiving treatment, as opposed to an interest in temporary hospitalization, may be erroneous.

Most studies of AMA behavior have operated from the premise that predisposing characteristics of the patient (e.g., personality variables, diagnosis, and age) increase the likelihood of AMA behavior (1). However, consistent variables have not been identified despite an intense interest in the subject. The alternative approach we are suggesting, an attention to the nature of the contracting process, does not preclude the existence of such elements as personality variables as determinants of AMA behavior. Rather, we feel that as a starting point a more pragmatic approach might be to concentrate on concrete evidence about the nature of the treatment contract the potential AMA patient wishes to sign with the hospital. Our data suggest that for most AMA patients this contract is intended from the start to be short-term. Psychological treatments are considered irrelevant and unnecessary. The estimated length of stay is to be a few days, no more. It is therefore a misinterpretation to view such patients' agreement to hospitalization as a request for ongoing psychological treatment or pharmacotherapy.

The literature dealing with treatment dropouts in an outpatient setting has emphasized that changes resulting from psychotherapy often divide into a set of long-term changes relating to specific symptom alteration and changes in interpersonal relationships (13–15). Some authors (16, 17) have said that the high incidence of dropping out of therapy within the first four outpatient visits reflects a tendency for patients to leave psychotherapy following initial nonspecific symptom relief. If this evidence from the outpatient setting is extrapolated to our study, we might interpret our findings as suggestive evidence that the potential AMA patient enters the hospital to obtain immediate nonspecific relief of poorly articulated conflicts and symptoms. The hospitalization itself provides this relief, independent of the more formalized features of the treatment program. Having received such relief, the patient leaves. What is interesting about our findings is the indication that patients have a relatively clear-cut conceptualization of this process and can articulate their views in terms of their lack of interest in the formal aspects of the treatment program.

Corrective measures in dealing with potential AMA patients. One possible conclusion from this study is that nothing more should be done for the AMA patient than to allow him or her to leave without a potentially

onerous label. This view suggests that such patients should be allowed to use the hospitalization as an interruption or a temporary removal from factors or events in their lives that are overwhelming their coping resources and that the hospital should not insist that they partake of psychiatric treatment.

An alternative view is that the ability to identify potential AMA patients at the time of admission provides an opportunity to institute a treatment program for these patients uniquely tailored to their needs. This would substantially increase the value of the hospitalization for such a patient and in the process would effectively eliminate the AMA "problem" by establishing a short-term hospitalization program for this group of patients rather than insisting that they follow the customary treatment program established for the entire service.

At the very least, the evidence presented in this study suggests that future efforts in dealing with AMA behavior should be directed toward prospective identification of potential AMA patients in addition to retrospective analysis of why the patient left after he or she has left the hospital.

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Psychosis in a Peasant Society: Social Outcomes

BY JOSEPH WESTERMEYER, M.D., PH.D.

Several clinicians have theorized that somatic and residential treatments have an untoward effect on the eventual outcome of major mental illness. To test this hypothesis, the author studied social coping behavior of mentally ill people in Laos, a predominantly peasant society with no psychiatrists or psychiatric hospitals. The Lao folk term baa *("crazy" or insane) was used in determining cases. Social factors studied included legal problems, family contact, sociability, friendship, communal activities, sexuality, and work. Results indicated that levels of social function in this sample were quite limited. The author concludes that social disability associated with chronic psychosis cannot be ascribed totally to diagnostic labeling or institutionalization.*

Some psychiatrists and psychologists have argued that psychiatric treatment, hospitalization, and psychotropic medication have an untoward effect on the eventual outcome of psychosis (1-4). Others have disagreed, stating that the social concomitants of psychosis cannot be so easily labeled iatrogenic (5-10). Anecdotal case reports have also shed doubts on the strategy of letting psychosis "run its course" (11).

Cross-cultural studies provide a means for addressing this issue by studying groups that do not have modern psychiatric treatment resources (although they do have "folk" treatment methods). Unfortunately, anthropologists have argued on both sides of the question (12, 13), just as have psychiatrists and psychologists. Cross-cultural field reports, like clinical reports, tend to be rich in anecdote and virtually impossible to replicate.

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The current study was undertaken to collect field data on psychotic persons. Laos was used as a site because 1) there were no practicing psychiatrists or psychiatric facilities in the country; 2) previous research had demonstrated considerable social tolerance for behavioral deviance (14, 15); and 3) disabled persons could theoretically work part-time at a variety of tasks and live within an extended kinship group. Thus, if psychotic persons in fact do well without psychiatric care, it was anticipated that they might do well in rural areas of Laos.

METHOD

Cases of psychosis were obtained by surveying 27 villages for persons labeled socially as *baa* ("crazy" or insane). This method has been suggested by Beiser and associates (16) as a reliable means of obtaining cases, based on their work in rural areas of Africa. Two years of general medical work in Laos (1965-1967) and a pilot study of 20 *baa* villagers (in the course of other research during 1972, 1973, and 1974) had also revealed few false positives (persons labeled *baa* but not found to be psychotic) and false negatives (persons found to be psychotic but not labeled *baa*).

Thirty-five subjects were identified in 27 villages located in Vientiane Province, an area of rice paddy farmers. Village populations averaged about 200 people. Research assistants known to the people of each area located the cases by contacting village headmen, elders, monks, and merchants. Research assistants also provided an entrée to the subjects and their families. I interviewed subjects and their collateral informants in the Lao language. The study was conducted over an 8-week period from January to March 1975.

A social rating scale was devised for Laos using social rating scales developed in other populations (17-22). Premorbid and morbid social ratings were based primarily on reports by informants (a mean of 6.9 informants per subject). These ratings were supplemented by reports from 5 subjects who were sufficiently lucid to provide such information. Premorbid rating periods consisted of the year before the subject became *baa*. The morbid rating period was the year before the research interview, except for 6 subjects who had been *baa* for less than 1 year. These subjects were rated for the time since the onset of their *baa*

condition. One variable, legal problems, was not handled in this manner because it was an infrequent event. Premorbid legal problems were assessed from age 18 to the onset of *baa* (mean duration=7.5 years), and morbid legal problems were calculated from onset to the present time (mean duration=8.0 years).

Reliability and validity are important considerations in anamnestic studies of this kind. One study of major life events has indicated that reports of relatives in regular contact with a subject over time demonstrate good reliability with subjects' self-reports (23). Where errors occurred, they were in the direction of underreporting rather than overreporting. Test-retest studies on anamnestic data have shown good reliability for major life events, even over prolonged periods (24–27), but poor validity for mundane events after short periods. A cross-cultural study comparing North American and Asian reporting of significant life events has shown marked similarity in the subjects' ratings of over 40 events (28). Since the information sought from these Laotian subjects and informants was highly significant to them, it is likely that valid data were obtained. The use of several informants for each subject also ensured reliability.

RESULTS

Demographic Characteristics

Subjects ranged in age from 15 to 68 years, with a mean of 35.3 years (SD=14.6 years). There were 18 male and 17 female subjects. Although most subjects were in their "marriageable" years by local standards, only 5 were currently married and living with a spouse. About half of the subjects were single, and another fourth were divorced or separated—an atypical finding in rural Laos, where most adults are married and divorce is infrequent. One subject was widowed and 2 were involved in common law relationships. The high illiteracy rate (45%) was not unexpected because most Laotians received little education until recent years. Twelve subjects had first- to eighth-grade educations; only 7 had attended high school. Most of the subjects were ethnic Lao. The 4 tribal subjects and 1 Chinese man had migrated from areas outside Vientiane Province. These ethnic "immigrants" had not been expected in the sample because all communities surveyed were ethnic Lao.

Clinical Characteristics

A panel of four clinicians independently assigned psychiatric diagnoses to each subject based on a case report and photograph obtained by the author (these data are presented in reference 29). The most common disorder was functional psychosis (N=24). Most of these cases were categorized as schizophrenia, with a few categorized as affective psychosis. Nine individuals were diagnosed as having organic psychoses. Their etiologies included chronic alcoholism (N=4), trauma (N=2), meningoencephalitis (N=1), stroke syndrome associated with alcoholism (N=1), and chronic cannabis use (N=1). The 2 remaining subjects may have had transient psychoses in the past or have been in an early phase of chronic psychosis, but they were judged neurotic at the time of interview by the raters. Because of their relative youth (both were age 16) and the relative recency of their psychotic manifestations (less than 1 year), the panel could not confidently assign a diagnosis of psychosis to these 2 subjects, who will be deemed "borderline." These diagnostic subgroups were not distinguishable in terms of social outcome data, so results for all 35 subjects are reported.

Social Outcome Variables

Legal problems. Police were not stationed in any of these 27 villages. However, police in nearby towns could be summoned by villagers in the event of serious disruption. As shown in an earlier paper (30), recourse to police, while infrequent, was especially sought when violence or feuding was likely.

Two men had been arrested before the onset of their psychoses: one for violent behavior while drinking, and the other for leading a splinter religious movement. Both were arrested again for the same offenses following the onset of their psychoses.

An additional 6 subjects, all men, were arrested for the first time following onset of their psychosis, usually in the early weeks or months. Descriptions of their offenses demonstrate their relationship to psychotic delusions and hallucinations.

> Unemployed subject thought himself to be a high government official and stole a government vehicle.
>
> Plagued by accusatory auditory hallucinations, subject assaulted his blind wife with a knife.
>
> Unemployed subject thought government officials wanted to pay him large sums of money; he was arrested for loitering around their homes and offices.
>
> Army private thought himself to be an officer and refused orders from his commander.
>
> In a furor precipitated by heavy cannabis use, subject destroyed part of home and assaulted several people.
>
> Son of a police official suddenly assaulted a policeman at a religious festival.

Comparison of premorbid and morbid legal problems showed only a marginal statistical significance ($\chi^2=2.9167$, df=1, $p<.09$).

Family contact. Extended families in Laos provide many social functions performed by institutions in the United States, including nursing care, shelter and food at times of crop devastation or flood, and social security in old age. People often live their entire lives in the same village and are in daily contact with many kinsmen.

Family contact for the purposes of this study consisted of visits, assistance, or conversation—i.e., any discernible face-to-face social transaction. In assessing family contact, definitions of frequency were as follows: 1) regular—family contact within last 30 days, 2) occasional—family contact within 31 to 365 days, and 3) infrequent—no family contact within 365 days.

Before the onset of their *baa* condition 30 subjects had regular family contact and 5 had occasional contact. None had infrequent contact.

At the time of the research interview, 19 subjects were having regular family contact. Seven people with occasional contact wandered away from their homes for periods of time ranging from a few days to a few months. Nine individuals with infrequent contact were living out in the open without shelter, begging and foraging for food.

Since there were relatively few subjects in the occasional and infrequent contact groups, these two categories were combined for the purpose of statistical analyses. Chi-square analysis showed a significant difference between premorbid and morbid family contacts ($\chi^2=6.8027$, df=1, $p<.01$).

Sociability. This refers to the subject's ability to contact people outside of the extended family. The categories were as follows:

1. Initiates contact—approaches people for conversation, invites people to visit home, or arranges entertainment for guests.
2. Responds to contact—responds to greetings, participates in conversation started by others, or responds to polite inquiries and requests.
3. Neither initiates nor responds—ignores people and does not respond to greetings, conversation, or requests.

Before the onset of the *baa* condition, 14 subjects had been able to initiate contact and 21 to respond. At the time of interview only 2 subjects were initiating contact (both had recurrent mental illness and were in lucid intervals). Four other subjects did not initiate contact but were able to respond. The remaining 29 people neither initiated nor responded to casual social contacts. These differences in premorbid and morbid sociability were highly significant ($\chi^2=65.8702$, df=2, $p<.005$).

Friends. The Lao term *peuan-mu* (friend) refers to nonkin who visit often and who value one another positively. Three different categories were selected: 4 or more friends, 1-3 friends, and no friends.

Before they became psychotic, 13 subjects had 4 or more friends. Seventeen others had 1-3 friends. Most of the subjects in this group had been "loners" as children and teenagers and had a diagnosis of schizophrenia. Five people had no friends in the years before they became *baa*. Of these, 3 were "loners" who subsequently became schizophrenic, 1 was an alcoholic woman who had alienated her friends, and the fifth was a mentally retarded man.

In terms of current friendships, 3 subjects had 4 or more friends. They were the man with unipolar psychotic depressions, a "borderline" woman, and a 68-year-old man who had become psychotic suddenly two weeks before the interview. Of the 6 people with 1-3 friends, 5 were beggar-foragers whose friendships were more in the nature of a benefactor-recipient relationship. The sixth, a man with organic psychosis, had been village chief for 22 years, and people continued to visit him regularly and provide assistance. In the remaining 26 cases, no current friend could be identified.

The difference between the premorbid and current status for this variable was highly significant ($\chi^2=22.3136$, df=2, $p<.005$).

Communal activities. In Laos various ceremonies, festivals, and holidays blur into a single national-cultural-religious unity. Being Lao implies communal observation and/or participation in several annual festivals, such as one called the "Birth, Death, and Enlightenment of the Buddha," regardless of whether one prays or is concerned about an afterlife. Participation in such activities provided information regarding a person's relationship to the community at large. The three categories were as follows:

1. Active participation—donates rice to the monks, decorates the temple at festival time, belongs to committees that sponsor festivals, or engages in secular parades and annual boat races.
2. Passive observance—observes ceremonies and festivities (e.g., religious rituals, secular parades, boat races, fireworks and rocket displays, water rituals).
3. Neither—neither observes nor participates.

Before the onset of the *baa* condition, all of the subjects either participated in (N=11) or observed (N=14) religious activities. However, at the time of interview, only 2 people were participating in communal activities—a young schizophrenic man living as a novice in a Buddhist temple and a young woman with episodic psychosis. Three other people continued to observe religious functions, including the man with recurrent depressions, a man with a periodic psychosis, and a "borderline" woman whose family brought her to temple festivals. None of the remaining 30 individuals had recently attended a communal activity. This difference in premorbid and morbid communal participation was highly significant ($\chi^2=47.7712$, df=2, $p<.005$).

Sexuality. Each individual was assigned to one of three sexual behavior categories:

1. Appropriate—regular intercourse if the person were married, courting behavior if single, or no sexual contact if a Buddhist monk or preadolescent.
2. Impaired—absent or infrequent (less than monthly) intercourse if married, inability to sustain a courting relationship if single over the age of 18, complete avoidance of the opposite sex if aged 14 to 17.
3. No sexuality or problematic sexuality—absence of sexuality among those for whom some sexuality

would be appropriate (such as intercourse among the married, courting among young adults), or problematic sexuality (such as touching of others' genitalia in public, public exposure of subjects' own genitalia).

Prior to the onset of the *baa* condition, 16 subjects had appropriate sexuality for their age, sex, and marital status. These included a 10-year-old with no overt sexual behavior, a few teenagers and single young adults who participated in courting, and several married individuals with late onset of the *baa* condition. Nine had premorbid sexuality impairment of some kind, primarily inability to maintain courting or marriage. The remaining 10—most of whom subsequently developed conditions diagnosed by the panel as schizophrenia—had been unable to even initiate courting behavior of any kind. There were no instances of premorbid problematic sexuality. By contrast, only 3 subjects were rated as having appropriate sexuality during their *baa* condition. One of these was a religious leader who had resolved his earlier sexual dilemmas by committing himself to celibacy and a monastic existence. The other 2 were alcoholics who may have had sexual dysfunction, but no specific data could be obtained to support that notion. In the impaired category, there were 3 subjects—a functionally psychotic woman who had intercourse with her husband infrequently, another functionally psychotic woman who also had infrequent sex but who was pregnant at the time of interview, and a 16-year-old "borderline" woman who danced with young men at formal festivals but strictly avoided them in any other context. All of the remaining 29 subjects were adults who were neither courting nor having intercourse. Five of them had sexual problems—1 man had gotten into difficulty because he inappropriately touched women at temple festivals, and 4 women had exposed their pudendal genitalia in public. This premorbid-morbid difference was highly significant (χ^2=17.9700, df=2, p<.005).

Work. Among villagers in Laos, children become partially productive workers by the age of 7 or 8, looking after younger siblings, collecting firewood, tending water buffalo, and assisting their mothers or fathers in a myriad of other tasks. By age 12–15 the average individual carries out certain sex-appropriate tasks independently, such as gardening, rice planting, or carrying water. By the age of 15 most adolescents are as productive economically as adults, with the infrequent exception of those able to continue in school. Three categories were designated for the work variable:

1. Productive—independent work appropriate for age, sex, and social status, without direct supervision.
2. Partially productive—direct supervision or assistance in carrying out ordinary chores such as gardening or housekeeping as required.
3. Nonproductive—not capable of any productive labor whatsoever, whether supervised or not.

Before they became psychotic, 29 people were productive. Another 4 were only partially productive; they had had early onset of the *baa* condition during late childhood or early adolescence. Two schizophrenic subjects who had been abnormal since childhood did no gainful work during the premorbid period.

At the time of the research interviews, only 2 subjects were productively employed. One was a widow with a trauma-induced organic psychosis who gathered firewood from a nearby forest for barter, and the other was a young "borderline" woman who did housework and some child care. Another 7 individuals, all of whom had been psychotic for 3 years or more, were partially productive but impaired for their age, sex, and social role. The remaining 26 subjects were unable to perform any productive work. This difference in premorbid and current work was highly significant (χ^2=41.0630, df=2, p<.005).

DISCUSSION

Social Outcomes

This prevalence study of social function indicated that severe social dysfunction was associated with psychosis in a peasant society. Even in an agricultural subsistence economy, most subjects were dependent on others for food and shelter. They became alienated from family or friends, and they ceased recreational and communal activities. Sexual gratification with others was impaired in most cases. Relatively few subjects in this sample had legal problems as a consequence of their disorder, but the frequency of such problems was slightly greater in the morbid than the premorbid state.

These findings are in contrast to the social functioning of psychotic patients who are receiving psychiatric care. Follow-up studies of psychotic persons receiving psychiatric care in North America and Europe have shown that many return to economic productivity (about half of schizophrenics do so) and make a fair to good social adjustment (31, 32). However, psychiatric care is not a panacea. Social impairments with less severe disturbances (such as depression) can persist for years even with psychiatric care (33).

Labeling and Institutionalization

Labeling by psychiatrists and institutionalization have been implicated as causes for the social disability that attends major mental disorder (1–4, 34). In this survey the *baa* label was appended exclusively by village members and the kin group; medical practitioners were not involved in this social process. None of these subjects was institutionalized, but they showed the social disability associated with psychosis elsewhere. Conversely, prisoners with prolonged institutionalization have not shown deterioration in cognition or personality (35–37).

Several investigators have begun to study the social effects of labeling and psychiatric patients' responses

to labeling (38–41). Although work remains to be done, it appears that the individual's self-labeling is a more critical factor than social labeling. From a therapeutic perspective, responses to self-labeling can be nonproductive (e.g., an excuse for deviant behavior) or productive (e.g., a step toward integrating the past psychotic experience into one's life and perspective).

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The Role of Cholinergic Supersensitivity in the Medical Symptoms Associated with Withdrawal of Antipsychotic Drugs

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From a review of the clinical literature, the authors determined that the medical symptoms of neuroleptic withdrawal occurred more frequently with neuroleptics having potent anticholinergic effects than with those having weak anticholinergic actions. When antiparkinsonian agents were not simultaneously withdrawn, there was a striking difference between these two categories of neuroleptics. Experiments with mice showed that withdrawal of haloperidol, a neuroleptic with weak anticholinergic effects, produced subsensitivity (depression of locomotor activity and seizure thresholds) to the cholinergic effects of physostigmine. These findings support the theory that the medical side effects of neuroleptic withdrawal are due to rebound cholinergic hypersensitivity associated with the anticholinergic actions of these drugs, rather than being related to their dopamine-blocking activity.

In the first two weeks after withdrawal of neuroleptic drugs, patients often experience symptoms including nausea, vomiting, diaphoresis, restlessness, and insomnia (1-13). The first three of these symptoms can be duplicated by withdrawal of anticholinergic agents from patients with Parkinson's disease (14) and appear to be due to increased cholinergic activity. It has therefore been suggested that the neuroleptic withdrawal syndrome is caused by rebound cholinergic hypersensitivity (15).

Animal experiments have shown that withdrawal from chronic haloperidol treatment reduces central sensitivity to cholinomimetic drugs (16, 17). Although this finding appears to contradict the hypothesis that withdrawal of neuroleptics produces cholinergic hypersensitivity, haloperidol has minimal anticholinergic actions, while many other neuroleptics possess strong anticholinergic activity (18). Thus, withdrawal of neuroleptics with weak anticholinergic activity might induce a central subsensitivity to acetylcholine. In contrast, withdrawal of neuroleptics with strong anticholinergic actions might produce a combination of central and peripheral cholinergic hypersensitivity that overshadows this effect. To clarify the cholinergic effects of neuroleptic withdrawal, we examined two issues: 1) whether a neuroleptic withdrawal syndrome is associated primarily with the use of neuroleptics having strong anticholinergic activity, and 2) experimentally, whether a generalized cholinergic subsensitivity, rather than hypersensitivity, is observed when a neuroleptic with weak anticholinergic activity is withdrawn.

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LITERATURE REVIEW

Method

A computerized search using the MEDLARS II data bank was carried out on the subject of Neuroleptics and Drug Withdrawal Syndrome. The relevant citations were collected and their bibliographies were also examined for additional citations. All cases that described symptoms classically associated with the medical effects (1) of neuroleptic withdrawal (vomiting, nausea, restlessness, insomnia, headache, abdominal pains, and diaphoresis) were noted. These cases were then divided depending on 1) whether the neuroleptics used had potent versus weak antimuscarinic effects (all neuroleptics with a relative affinity for muscarinic receptors of less than 1 were considered weak antimuscarinic agents [18]; when data on binding were not available, aliphatic and piperidine phenothiazines were placed in the potent group and piperazine phenothiazines in the weak group), and 2) whether antiparkinsonian agents (benztropine, trihexyphenidyl) were withdrawn simultaneously with the neuroleptics. When information was available, cases with symptoms of either restlessness or insomnia were also separately noted to determine with which drugs they occurred and whether they occurred in conjunction with symptoms that can be produced by withdrawal of anticholinergic agents from Parkinsonism patients (e.g., gastrointestinal disturbance and diaphoresis).

TABLE 1
Reports of Patients Developing Medical Symptoms After Withdrawal of Neuroleptics With or Without Simultaneous Withdrawal of Antiparkinsonian Drugs

	Number of Patients Developing Medical Symptoms											
	Neuroleptics with Potent Antimuscarinic Effects						Neuroleptics with Weak Antimuscarinic Effects					
	Antiparkinsonians Not Withdrawn		Antiparkinsonians Withdrawn		Antiparkinsonian Data Not Known		Antiparkinsonians Not Withdrawn		Antiparkinsonians Withdrawn		Antiparkinsonian Data Not Known	
Study	N	Total Sample	N	Total Sample	N	Total Sample	N	Total Sample	N	Total Sample	N	Total Sample
LaCoursiere and associates (1)	12	24	2	10			1	7	3	7		
Greenberg and Roth (2)	15	21										
Simpson and Varga (3)	3	9										
Brooks (4)					20[a]	20					1	1
Judah and associates (5)					2[a]	42					0	35
Haden (6)					7	30					0	10
Melnyk and associates (7)					15	20						
Yepes and Winsberg (8)					2[a]	2						
Simpson and associates (9)							0	19	9[b]	14		
Gallant and associates (10)									25	68		
Kalman and Warner (11)									1	1		
Subtotal	30[c]	54	2	10	46	114	1	26	38	90	1	46
Total			78[d]	178					40	162		

[a]All of the patients had symptoms of either insomnia or restlessness.
[b]Five of these patients had symptoms of either insomnia or restlessness.
[c]In the subgroups in which antiparkinsonian drugs were not withdrawn there were significantly more cases of withdrawal symptoms among patients receiving neuroleptics with potent antimuscarinic effects than among those receiving neuroleptics with weak effects (Fisher exact probability test, p=.000004).
[d]There were significantly more cases of withdrawal symptoms among the total number of patients receiving neuroleptics with potent antimuscarinic effects than among the total number receiving neuroleptics with weak effects (chi-square test, p=.0002).

Results

Table 1 presents a list of 11 reports providing information on 118 cases of medical symptoms following withdrawal of neuroleptics with potent or weak antimuscarinic effects. There were more cases of withdrawal symptoms (relative to the number of patients) among patients who had received neuroleptics with potent (44%) versus weak (25%) antimuscarinic effects (chi-square, p=.0002). This effect was even more pronounced among patients for whom antiparkinsonian medication was not simultaneously stopped. In this group there was a greater frequency of withdrawal symptoms among the patients receiving neuroleptics with potent (56%) as compared to weak (4%) antimuscarinic effects (Fisher exact probability test, p=.000004).

Table 1 also shows that symptoms of restlessness or insomnia occurred among 29 patients after withdrawal of either phenothiazines with potent antimuscarinic effects or phenothiazines with weak effects and antiparkinsonian medication. In 21 of the cases the restlessness and/or insomnia occurred in conjunction with gastrointestinal symptoms and/or diaphoresis. Brooks (4) believed that those 8 cases in which it occurred without such symptoms represented less severe forms of the withdrawal syndrome. Finally, restlessness occurred, along with these other symptoms, in 2 of 9 patients who discontinued antiparkinsonian medication four weeks after they had stopped taking all neuroleptics (9).

In our literature search we also found two studies in which the neuroleptics were not specified and therefore the antimuscarinic effects could not be determined. Bennett and Kooi (12) reported that 8 of 25 patients developed medical symptoms after withdrawal of neuroleptics; it was not known whether they were receiving antiparkinsonian drugs. Battegay (13) reported that 35 of 58 patients receiving neuroleptics without antiparkinsonian drugs developed medical symptoms when neuroleptics alone were withdrawn; 20 of 23 patients developed medical symptoms when neuroleptics and antiparkinsonian drugs were withdrawn simultaneously.

ANIMAL EXPERIMENTS

Method

Haloperidol exposure. Animals were exposed to haloperidol as previously described (19). In brief, adult female mice weighing 25–40 g were given either haloperidol (1 mg/ml) or its vehicle (1 mg/ml of lactic acid

and deionized water) in their drinking water as their only source of fluid for 14 days. During this period the mice drank approximately 10 mg/kg of haloperidol daily. On the 14th day of haloperidol exposure the animals received an intraperitoneal injection of 10 mg/kg of haloperidol or its vehicle (1% lactic acid), and the haloperidol was discontinued. Five days later the animals were tested.

Activity testing. Animals from each treatment group (haloperidol or its vehicle) were given an intraperitoneal injection of .15 mg/kg of methylscopolamine to block the peripheral effects of the physostigmine. Physostigmine was injected intraperitoneally in one of three doses (.15 mg/kg, .3 mg/kg, and .6 mg/kg) 30 minutes later. Fifteen minutes after the physostigmine injection the animals were placed in activity meters (Motron Produkter Co.) and kept there for 10 minutes.

Seizure threshold. Animals from each treatment group were given an intraperitoneal injection of .5 mg/kg of methylscopolamine. After 30 minutes, physostigmine seizure thresholds were determined by infusing the tail vein with a solution of .3 mg/ml of physostigmine at a rate of .2 ml/minute. The infusion was continued until the animals developed clonic seizures. The time in seconds from the beginning of the infusion until the onset of seizures was then divided by the weight of the animal to determine the dosage per unit weight. Animals that did not develop seizures within 4 minutes were excluded from the analysis.

Results

The effect of various doses of physostigmine on locomotor activity in mice withdrawn from haloperidol or its vehicle was examined by a two-way analysis of variance. There was a significant ($F[1,30]=4.44$, $p<.05$) effect of chronic haloperidol treatment as well as a significant ($F[2,30]=9.20$, $p<.01$) physostigmine effect. A post hoc comparison using the Scheffé test showed that there was a significant ($p<.05$) difference between the haloperidol and vehicle conditions at a physostigmine dosage of .15 mg/kg (mean ± SD=182.7 ± 122.4 versus 91 ± 72.4, N=6) but no significant difference at .3 mg/kg (74.5 ± 44.6 versus 33.2 ± 26.4, N=6) or .6 mg/kg (26.3 ± 20.2 versus 14.8 ± 10.5, N=6).

The mean seizure threshold dosage (± SD) of physostigmine for the haloperidol group was 3.3 ± 1.2 mg/kg (N=11), and for the control group the threshold dosage was 2.2 ± .43 (N=13). This difference was statistically significant (Behrens-Fisher t test, $p<.05$).

DISCUSSION

Our literature survey has shown that the medical withdrawal symptoms related to cessation of neuroleptic medication occur primarily with agents having potent antimuscarinic effects. This finding is accentuated in situations in which antiparkinsonian agents are not simultaneously withdrawn. We could find only one case (1) in which the withdrawal of an antipsychotic with weak antimuscarinic effects produced symptoms without the simultaneous withdrawal of antiparkinsonian medication. Unfortunately, the authors did not describe any details about drug, dosage, or specific symptoms for this case. We also found that symptoms of insomnia and restlessness occurred in the same drug setting as other medical symptoms and usually in conjunction with them. These findings suggest that the classic symptoms occurring after withdrawal of neuroleptics represent a syndrome that is secondary to cholinergic rebound. This syndrome is therefore probably not related to the antipsychotic effect of these drugs, which is believed to be mediated by dopaminergic blockade (20). This conclusion is in keeping with that of others who have reviewed this literature (1, 15) and have suggested that the anticholinergic effects of neuroleptics and antiparkinsonian agents could be involved in the production of withdrawal symptoms.

Two recent reports (8, 11) have attempted to explain the nausea and vomiting noted after neuroleptic withdrawal as secondary to dopaminergic supersensitivity in the chemoreceptor trigger zone. In both reports the drug regimes involved withdrawal of neuroleptic and/or antiparkinsonian agents with potent antimuscarinic effects. Thus the hypothesis that dopaminergic supersensitivity is involved may be unwarranted. Haden (6) noted gastrointestinal symptoms in 7 of 30 patients who stopped taking agents with potent antimuscarinic effects (10 each were receiving chlorpromazine, thioridazine, and chlorprothixene) but none in 10 patients who discontinued perphenazine, which has weak antimuscarinic effects. Haden felt that these findings argued against the action of these drugs on the chemoreceptor trigger zone as being important in producing nausea and vomiting, since these symptoms occurred after withdrawal of drugs such as thioridazine that do not have significant antiemetic effects, but not after perphenazine, which does have strong antiemetic effects.

The animal studies demonstrated that the decreased central responsivity to cholinomimetics after withdrawal of haloperidol from mice is not specific to locomotor activity but is also observed for physostigmine seizure thresholds. This suggests that withdrawal of neuroleptics with weak antimuscarinic effects produces a generalized decrease in central sensitivity to cholinergic stimulation, which may be secondary to increased acetylcholine release caused by haloperidol (21). We therefore suggest that central cholinergic subsensitivity may be a direct concomitant of the antipsychotic action of the neuroleptics, while the clinical neuroleptic withdrawal syndrome is comprised of a combination of central and peripheral cholinergic hypersensitivity resulting from anticholinergic effects of

the drugs. Although certain symptoms such as restlessness and insomnia may indicate increased cholinergic activity in the central nervous system, others such as gastrointestinal distress and diaphoresis appear to be of peripheral origin. Our findings suggest that both categories of symptoms are seen primarily after withdrawal of neuroleptics with potent antimuscarinic actions only.

There is ample evidence that neuroleptics produce central dopaminergic supersensitivity (22), possibly including mesolimbic dopaminergic supersensitivity in man (23). It has been suggested that neuroleptic withdrawal might exacerbate psychosis through this mechanism (24) and that the decreased sleep and restlessness observed after withdrawal of neuroleptics reflect such dopaminergic supersensitivity (23). However, this may not be the case. First, restlessness might be an extrapyramidal reaction (akathisia) due to the slower excretion of neuroleptics than of antiparkinsonian agents. Like other extrapyramidal symptoms, it would be secondary to a relative decrease, not increase, of dopaminergic activity. Second, our literature review suggests that even restlessness and insomnia can be part of a generalized syndrome associated with cholinergic rebound. Clearly, in a setting in which neuroleptics with strong antimuscarinic effects or antiparkinsonian agents are withdrawn, the resulting restlessness and insomnia cannot be construed as evidence of dopaminergic supersensitivity.

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A Clinic for the Impaired Elderly and Their Families

BY BURTON V. REIFLER, M.D., M.P.H., AND CARL EISDORFER, PH.D., M.D.

The need for improving psychiatric services to the elderly and for recognizing the importance of the family in caring for the impaired elderly led to the establishment of the Geriatric and Family Services, an outpatient clinic. This clinic provides psychiatric, medical, social, nursing, and architectural evaluations and recommendations for impaired older persons and also provides support and practical advice to the family. Most of the clinic's patients suffer from a dementing illness; of this group most have dementia of the Alzheimer's type. The authors studied the appointment-keeping behavior of the patients and also surveyed the patients' families as an indication of the clinic's success. Out of 50 patients the broken-appointment rate, as well as the drop-out rate, was 2%; 90% of the families who responded to the questionnaire indicated they would recommend the clinic's services to others.

Persons older than 65 years of age, which includes 10% of the United States population, are at high risk for physical and mental problems; they occupy 90% of the long-term care beds in the United States and show the highest rate of first admissions to public mental hospitals (1). Despite estimates that 12%–40% of people 65 and older suffer from a mental disorder (2, 3), only 4% of these patients are in psychiatric outpatient treatment programs (4). Community mental health centers have specifically been found wanting in providing services to the elderly (5).

Although the epidemiology of psychiatric disorders in the elderly is not well elaborated, two particularly salient problems are organic brain syndrome and depression. The former is diagnosed in more than 75% of the geriatric patients admitted to a public psychiatric institution for the first time (6), while the latter accounts for 50% of the geriatric patients admitted to private psychiatric hospitals (7). The most common cause of chronic organic brain syndrome in the elderly is dementia of the Alzheimer's type, which accounts for at least 50% of the 10% to 20% of the elderly population who have evidence of the syndrome (8, 9).

There are numerous indications that families of impaired older persons could be a major resource in assuring that the psychiatrically impaired older adult receives a thorough evaluation and adheres to a treatment plan. The fact that almost 90% of the elderly have seen one or more relatives during the previous week and more than 75% live within 30 minutes of their nearest child (10) reflects the strong family ties of most elderly Americans. Common living arrangements for the elderly include living with a spouse, sibling, or other relative (11).

When an older member of the family develops an impairment, the family typically takes the responsibility for that person's welfare. Brody (12) points out that 80% of the support services to older people are provided by families. Furthermore, in a study of 280 admissions to a geriatric unit, Isaacs (13) noted that neglect by relatives played a negligible part in the need for admission, but he also found that the condition in the elderly most difficult for families to bear was the long-continued presence of mental disorder.

Lowther and Williamson (14) also pointed out the willingness of relatives to accept the care of their elderly family members. In a review of 1500 patients discharged from a geriatric unit, there were only 12 unreasonable refusals by relatives to provide home care. An unreasonable willingness to care for an elderly relative was five times more common in cases where the staff actually questioned the ability of the insistent family to undertake home care.

In assessing the available data we concluded that the psychiatric needs of the elderly have not been adequately addressed, particularly if we consider the use made by the elderly of outpatient settings, and that the potential role of the family in caring for such patients has not been sufficiently supported.

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DEVELOPMENT OF THE GERIATRIC AND FAMILY SERVICES CLINIC

During the course of our professional activities we received numerous unsolicited inquiries from family members of impaired elderly reflecting the issues noted above. Family members would call us requesting a consultation or complete evaluation for their relative, usually a parent or spouse who showed signs of confusion and memory loss and who may have been informed by his or her physician that "nothing can be done." Often placing the elderly relative in a nursing home appeared inevitable. In response to these inquiries and requests for help, a clinic was established that would provide such an evaluation and concurrently offer counseling to family members as they attempted to cope with the situation.

The clinic opened August 1, 1978, with three part-time staff members: a psychiatrist (the program director), a social worker, and an internist. A full-time research technician was added a month later to assist in evaluating the clinic's work. The social work position became full-time, and the part-time staff and clinical consultants grew to include another internist, three family physicians, an occupational therapist, a nurse, two psychologists, and an architect. Trainees have included medical students, psychiatrist and psychologist geriatric fellows, and social work students.

During the evaluation phase of the program the elderly person makes a minimum of three, but typically four, visits to our clinic. When feasible, a member of the staff visits the patient at home. On the first visit the psychiatrist conducts a diagnostic interview with the patient, who is usually accompanied by one or two family members. The psychiatrist meets with them all together, with the patient alone, and with the family alone and makes observations on the dynamics of the family constellation. The social worker makes a home visit to further assess social structure and family stresses and to gather information on the patient's home environment. At the next visit a physical examination is conducted by an internist or family physician with the primary goal of identifying and treating reversible causes of organic brain syndrome. The roles of the other clinic staff members are evolving: the architect assesses the congruence between the individual's abilities and desires and his or her actual living situation and the nurse and occupational therapist usually assess the patient's activities of daily living and seek ways to strengthen the patient's capabilities.

Following the patient's acceptance into the program, the home visit, and a physical evaluation, there is a staff conference to consolidate findings and develop recommendations in 11 specified areas: 1) housing and living situation, 2) food and nutrition, 3) self care, 4) physical health, 5) household tasks, 6) emotional and mental factors, 7) financial matters, 8) transportation, 9) day-to-day routine, 10) family stress caused by the patient, and 11) patient's interference with family members' work or other activities. Using the notes from this conference, the staff prepares treatment recommendations and discusses them with the patient and the family at the next session. The staff member may make specific suggestions to the patient and family before this meeting or introduce them at this time. The suggestions may include strategies such as starting medication; stopping medication; counseling for patient, family, or both; use of community resources; behavior modification; and environmental manipulation. Follow-up treatment is also offered by the clinic.

The three case examples below illustrate typical reasons why families bring patients to the clinic, and how the staff is able to assist them.

CASE REPORTS

Case 1. Ms. A, a 70-year-old woman, was referred by her daughter for an evaluation of Ms. A's memory loss and angry outbursts. Ms. A had had a gradual loss of memory over 2 years and then suffered a stroke causing right-sided weakness and expressive aphasia, but she continued to live in her own home until her daughter learned that she had arranged for home repairs with an unscrupulous contractor. The daughter then insisted that her mother move in with her.

The family stresses were severe. Ms. A's daughter felt anger, resentment, and guilt concerning her mother and anger toward her nearby brother who refused to help. Furthermore, her husband and her mother had never gotten along.

Our evaluation suggested a combined Alzheimer's and multi-infarct etiology for Ms. A's dementia. She was deficient in folic acid, but its replacement did not lead to improvement. The family was particularly stressed by Ms. A's habit of beginning lengthy and frustrating conversations while her daughter and husband were watching television.

During the next 6 months, the clinic staff suggested several approaches. Antipsychotic medication was used occasionally for Ms. A's belligerent outbursts, and simple behavioral techniques (reinforcing desired behaviors and ignoring undesirable ones) reduced the evening television disruptions. Also, Ms. A's daughter and her husband attended a six-session structured group, which was conducted by clinic staff, where they could share common experiences with other families in similar situations. Their need for respite was supported by this group.

On several occasions Ms. A's daughter had become fatigued and frustrated and ready to place her mother in a nursing home. Each time the staff offered to provide the daughter with whatever support she wished—either advice on how to select a nursing home, suggestions for coping with the immediate situation, or both. To date, Ms. A has remained in her daughter's home. Although the daughter reports that her mother's condition has continued to decline, she feels better able to cope and is more confident that she will know when she is no longer able to care for her mother.

Case 2. Ms. B, who was 73 years old, was referred by her son and daughter for an evaluation of her memory loss and belligerence. She lived alone, refusing all help except that which she grudgingly and thanklessly accepted from her

family. They had developed a support system of visiting her twice daily, bringing her meals, and doing her shopping. It was only at their insistence that she was brought in for an evaluation, and she was furious at her children for "wanting to kill me."

Although she had Alzheimer's disease, she had no tendency to wander, her bladder and bowel functions were not impaired, and she maintained reasonable personal care. During the family interview we noted her son was calm and kept a sense of humor, but the daughter was distraught and fearful, partly due to fears that she was seeing her own future.

Antipsychotic medication improved Ms. B's belligerence and paranoia, and her family decreased their visits to once daily. Staff discussions with her family provided a forum for practical information and supportive psychotherapy.

At the most recent follow-up, Ms. B still lived in her own home and was participating in a day center program. Unfortunately, a new problem has emerged; her son's wife felt abandoned by him because of the time he spent assisting his mother. The staff will attempt to help resolve this conflict.

Case 3. Mr. C, 67 years old, was referred by his wife for evaluation for a 4- to 5-year history of memory difficulty and increasing depression. On numerous evaluations he had been diagnosed as having Alzheimer's disease; they both sought consultation to help them deal more effectively with the situation.

Mr. C had evidence of Alzheimer's disease with depressive features, but he also had motor abnormalities suggestive of Parkinson's disease; this additional diagnosis was confirmed by the internist. An architectural consultant accompanied other clinic staff on the home visit and noted several problems with the design of their newly purchased condominium, including an awkward three-step exit from the back door. Mr. C called this exit the "elevator shaft."

The clinic staff made three recommendations to Mr. and Mrs. C. First, a carbidopa-levodopa combination was prescribed for his Parkinson's disease, and he had subsequent improvement in movement and affect. Although Mr. C still had minimal tremor, his spontaneity improved in conversation and initiation of activity.

Second, the staff recommended architectural and environmental modifications, including improved access to the back yard via a railed deck with wide steps to replace the "elevator shaft," a more definite path from the living room to hallway by moving furniture around, addition of a grab bar to the tub, and improved illumination from the bedroom to the bathroom.

Third, individual sessions were scheduled with Mr. C's wife to assist her in dealing with the guilt, anger, and frustration she felt. She was advised that these were normal reactions and that she would best be able to care for her husband if she had the opportunity to pursue her own activities and interests.

Six months after the initial visit, Mr. C had improved physically and mentally and his wife felt more able to continue her work without experiencing guilt and anger.

RESULTS

Of 82 consecutive patients seen at this clinic in 1979, 68 (83%) had evidence of cognitive impairment according to the scale of Kahn and associates (15), the Dementia Rating Scale (16), and the Mini-Mental State (17). Of these, 53 patients were rated as having moderate or severe cognitive impairment, but 26 were judged to have at least partially reversible impairment. Dementia of the Alzheimer's type was diagnosed in 48 patients. Although 18 of the 82 patients met research diagnostic criteria for definite or probable primary or secondary depression (18), 23 additional patients were suspected of having some element of depression based on less stringent clinical criteria. These findings suggest that our clinic is seeing the population for which it was intended, primarily older adults with dementing illness, often occurring with depressive features.

The mean age for the elderly patients we saw was 77 years; women were referred more than twice as often as men (56:26); approximately 50% of the patients owned their own homes; and their mean income was $688/month (range, $225-$2000). The referring family member was usually a daughter (N=32), spouse (N=27), or son (N=14), and approximately 50% of the referring family members lived with the patient. The average fee billed to each patient was $543, including an average of $350 paid by Medicare for each patient, and the average out-of-pocket cost to the patient was $193.

Services we provided in addition to the four-visit evaluation process have been monitored on 64 patients. Additional office visits were made by 27 patients (42%); the maximum number of additional visits made by one patient was 12, and the average number of additional visits made for this group was 1.3.

One crude estimate of the value of the clinic to the family is the appointment-keeping behavior of the patients. During the pilot year there was a 1- to 2-month waiting list. In spite of this, there was only one occurrence of a patient (2%) who failed to keep the initial appointment (less than 24 hours' notice of cancellation) among the first 50 patients we interviewed, and only one family (2%) in the first 50 cases dropped out before completing the 4-session evaluation. (In this case the family members requested that we verify their belief that certain disabilities were present in the patient, but our examination failed to confirm their belief.)

A questionnaire was mailed to the referring family member 3 months after the patient completed the evaluation phase of the clinic's program to determine his or her satisfaction with our service. Of the first 30 patients meeting this criterion, the questionnaire was returned by 21 family members (70%). Of those family members who responded, 90% said they would call if they needed more help, 90% would recommend the clinic to others, and 71% were satisfied with the overall evaluation and treatment. An improvement in the patient's physical health was reported in 38% of the responses, mental functioning was improved in 24%, and the family's ability to handle the stress of the pa-

tient's problems improved in 47%. We do not know which specific components of the clinic's program the families found most helpful.

We have begun a follow-up study to determine each patient's current living situation and to calculate the rate of institutionalization for these patients. Our only data thus far show that for a sample of 15 patients living at home at the time of the evaluation, 3 (20%) were living in nursing homes at the time the mailed questionnaire was returned.

DISCUSSION

The broken-appointment rates and drop-out rates noted above are surprising in the light of reports documenting a 25% broken-appointment rate in a pediatric clinic (another situation where a family would be referring a patient) and a 10%–50% broken-appointment rate in other settings, including psychiatric outpatient clinics (19–21). When the waiting period reaches 30 days, as ours did, patient nonattendance is as high as 75% (22).

Nearly all of the families requesting appointments had had difficulties for 1 year or more. There may have been a precipitant, such as accelerated decline in cognition, a deterioration in social skills, nocturnal wandering, paranoia, depression, or chaotic personal finances. At the time of the first visit the family members had usually begun to contemplate institutional care or guardianship; they were beset with guilt and were confused about their responsibilities and best course of action.

Families often have realistic overt goals but unrealistic hidden expectations that the clinic will provide a cure for their relative. Their resulting frustration frequently became a valuable point of departure for beginning the therapeutic process as the families dealt with their range of feelings. The structured group sessions have been particularly helpful to families and have been well received both times they were offered. Our current plans are to conduct them at least four times a year. The sessions are open to all families going through the clinic's program. At present three sessions are didactic with informal discussions—the psychiatrist discusses diagnosis and prognosis of memory loss, the internist talks about physical problems that affect mental function, and the occupational therapist speaks on time structuring. The remaining three sessions are led by the social worker and are intended to promote the family's expression of feelings and discussion of shared problems.

The availability of the clinic staff members to the patients and their families has been important in warding off crises. In our judgment a 10- to 15-minute telephone consultation between our staff and the family, which allows ventilation and provides practical suggestions, has often been sufficient to keep the situation tolerable. We encountered only one instance in 50 consecutively enrolled patients where the family abused our offer of telephone consultation.

Funding for the Geriatric and Family Services program has come from a variety of public and private sources, including the sponsoring university, a local civic-minded savings bank, and a national private foundation sponsored by retired persons. Although many problems remain, including the lack of Medicare coverage for counseling families of impaired elderly people, the demand for our service and the patients' appointment-keeping behavior suggest that such a program could easily be self-supporting in a private setting and nearly self-supporting in an academic setting.

Perhaps the most important early finding of this program is that it dispells the myth that older persons will not see a psychiatrist. Although most of our patients clearly would not have sought the initial psychiatric consultation on their own (their families made the arrangements and insisted that they attend), they frequently looked forward to subsequent visits.

The services provided by our clinic are clearly not applicable to all impaired older persons. There are many situations that are too urgent to wait 1 or 2 months (although expansion of our program has reduced the wait to 1 or 2 weeks), and many impaired older persons lack an interested family member or even family surrogate. Despite these limitations, this model appears to meet a need among the families of psychiatrically impaired elderly. It also offers a training site where points of view from several disciplines are consolidated into recommendations that can be implemented by the persons most likely to influence the patient—his or her own family.

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A Psychoanalytic Approach to a Therapeutic Impasse with an Impulsive Adolescent: Permission to Speak the Unspeakable

BY AXEL HOFFER, M.D., ROGER GOETTSCHE, M.D., AND FAY LINDEN, M.S.

The authors describe an impasse in the therapy of an impulsive, intermittently psychotic adolescent boy and demonstrate the usefulness of understanding the impulsive behavior according to the classical psychoanalytic definition of "acting out." Rather than being viewed as a random breaking through of impulses, this impulsive behavior can best be understood and clarified in the course of intensive psychotherapy as classical acting out under the influence of the therapy and the transference. With impulsive patients of this type they have observed a strong tendency to abandon this basic psychoanalytic formulation and the use of the verbal interventions that follow from them. Such patients often need encouragement to remember and express their painful feelings in words instead of repeating the entire emotional experience in action. The authors also describe the resistances within the treatment team that interfered with the development and utilization of this basic psychoanalytic formulation.

The treatment of the intermittently psychotic, impulse-ridden adolescent boy presents a difficult challenge. When such a patient recovers from an acute psychotic episode, impulsive behaviors of an aggressive, sadistic, antisocial, and/or suicidal nature often become the patient's major defenses against intolerable affects and fantasies. Unfortunately, the patient's unmanageable behavior frequently leads the treating staff to focus exclusively on attempts to control the patient's behavior. Typically, the patient's hospital stay is characterized by intense struggles between himself and the staff over restrictions, with periodic outbursts of violence directed toward the staff, other patients, property, and often himself.

Psychotropic medication, in contrast to its effectiveness in alleviating the patient's psychotic symptomatology during the acute phase, has minimal benefit in this phase of the illness. ECT, although helpful with an acutely suicidal patient, is of questionable value for a person whose mood swings are as sudden and extreme as his impulsive behavior. The devastating effect of this charged situation can be seen on the patient, his family, and, not surprisingly, on the treatment team as well. We have observed that the patient feels hopeless and deprived of a sense of personal worth and growth.

The family alternates between indulging the patient and ostracizing him. The therapist feels increasingly frustrated and helpless as schemes for behavior control end in an interminable struggle, which proves at best to be an uneasy draw. This feeling of helplessness may lead the therapist to extend covert invitations to the patient to engage in further antisocial behavior, and thus the patient expresses the helplessness and anger felt by everyone. Finally, these feelings may lead the staff to wish to be rid of the patient and, consequently, the patient is subjected to poorly planned or inappropriate referrals. In desperation the penal authorities may be sought out to provide greater external controls for the patient than those that can be obtained in an open-door community mental health center.

We would like to describe a treatment impasse with one such patient. (In order to protect the patient's identity, our case report is a composite report containing altered data in all but the essential technical and dynamic details.) The impasse in the treatment was reached when the patient's impulsive and destructive behavior became unmanageable. Our efforts to understand and approach this treatment impasse brought to our attention the far-reaching consequences of viewing a patient's behavior as "acting up" on the one hand or as "acting out" on the other. We define the colloquial term "acting up" as random characterologic impulsiveness without specific dynamic significance. "Acting out" is defined by Freud (1) as a phenomenon in the analytic setting in which ". . . the patient does not remember anything of what he has forgotten or re-

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pressed, but acts it out. He reproduces it not as a memory, but as an action; he repeats it, without of course, knowing that he is repeating it . . . under the condition of resistance." Regarding his approach to acting out, Freud is very clear: "The tactics to be adopted by the physician in this situation of acting out are easily justified. For him, remembering in the old manner—reproduction in the psychical field—is the aim to which he adheres. . . ."

We concluded after our experience in attempting to overcome the treatment impasse with our patient that impulsive behavior in severely disturbed patients receiving intensive individual psychotherapy can best be understood as acting out in the classical psychoanalytic sense. To permit and even encourage patients to bring their painful memories and affects into the "psychical field"—that is, to help them put their feelings into words within the therapeutic alliance—is as crucial with patients of this type as it is with healthier, neurotic patients in psychoanalysis.

LITERATURE REVIEW

From a psychoanalytic point of view, the literature pertaining to delinquent adolescents begins with Aichorn's classic book *Wayward Youth* (2). He describes with sensitivity his attempts to understand antisocial behaviors as symptoms with historical, dynamic, and developmental implications akin to neurotic symptoms. We found a sparse amount of literature describing patients who specifically fit into our patient's category of an adolescent boy who showed schizophrenic symptomatology followed by prominent impulsive and antisocial delinquent-like behavior (3, 4). We observed in our patient and a small number of others that an acutely schizophrenic patient can develop delinquent or psychopathic-like behavior following recovery from the psychosis. Kanzer (5) quoted Bettelheim as saying that psychopathy may mark a restitutional phase of psychosis, and we are in full agreement with this observation. This shift in ego defenses adds immeasurably to the complexity of finding and maintaining a consistent and appropriate therapeutic approach. The crucial role of the parents in unwittingly encouraging acting out behavior in the adolescent as an expression of unresolved parental conflicts is clearly described (6, 7). The unconscious condoning of the asocial impulses is shown to allow the parent vicarious gratification of forbidden impulses as well as the expression of hostile impulses toward the child. Our study confirms these observations, including the importance of the parent's role in treating the child.

Relevant literature on the origins, definition, interpretation, and meaning of acting out (5, 8) emphasizes disturbances in the oral phase of development, strong stimulation of sexual and aggressive drives, and a disproportion between motor activity and verbalization.

Acting out was originally defined as behavior induced by psychoanalytic treatment, but how does one conceptualize patients who are described as "habitually acting-out patients" before as well as during treatment? Anna Freud's lucid clarification (9) of the confusion introduced into the concept of acting out within the widening scope of psychoanalysis is of special relevance to our thesis. She states,

> Unlike the neurotic, the delinquent, the addict and the psychotic act out habitually, i.e., also without the releasing benefits of the analytic technique. With them the upsurges from the id which cause their impulsive behavior have to be understood as belonging to their pathology, not to a curative process. . . . Instead of originating within the analytic situation and affecting the environment secondarily only, the impulsive behavior of these types begins in the external world from where it has to be dislodged and drawn into the transference. The analysis of the delinquent, according to Aichorn, starts when he steals from the analyst instead of from strangers.

Our observations strongly indicate that by careful study one can meaningfully distinguish habitual impulsive behavior from impulsivity directly related to the influence of psychoanalytic psychotherapy. Only after the patient's behavior is defined as acting out in the classical sense, rather than habitual random acting up, will it be possible to encourage the patient to extract the painful memories from a reenactment of the past.

CASE REPORT

Stephen, a 15-year-old boy, was brought to the Massachusetts Mental Health Center following an attempted suicide by hanging while he was in jail. On admission he appeared gaunt and pale, and his eyes stared blankly into space. He was tearful and complained to his doctor of feeling persecuted and inadequate. Specifically, he was frightened: "My sperm is gone. My genitals are dried up. I am not the big man I thought I was." Several days later he described auditory hallucinations and claimed his father's voice said he would be killed. His associations were loose; there were no signs of organicity or a toxic state. He was fully oriented; results of physical and laboratory examinations were unremarkable. Stephen's admitting diagnosis was schizophrenic reaction, schizoaffective type.

Stephen was the oldest of seven children. His father, a blue-collar worker, began flaunting his extramarital affairs within the first week of marriage. He was a convicted rapist and an alcoholic who frequently beat his wife and children. Although Stephen's father was transient in the household, he returned home frequently enough to impregnate his wife eight times before a divorce was obtained. Stephen was ten years old when his parents divorced.

Stephen's mother, an attractive and seductive woman, said that since the age of 3 Stephen had been like his father in every way: "He is unaffectionate and selfish. He is spoiled, always wanting to be special. At dinner he demands the best and biggest of everything on the table. Stephen has a cruel streak towards small children, also like his father. He is cruel

to insects and small animals, which he often tears apart." Other adjectives used by his mother to describe her son were "cunning," "crafty," and "tricky."

Stephen's latency was highlighted by good performance in school. Although Stephen remained a loner outside the family, he behaved at home in a taunting and mean fashion toward his younger brothers and often provoked a physical confrontation with them from which he, the oldest, would emerge the victor. Outside the home he was ambitious and industrious, and he managed to save substantial amounts of money by running two paper routes. Before the onset of his present illness, Stephen's school performance had not declined.

Stephen said his present illness began when he was 14 years old and experienced his first ejaculation. He was having difficulty making friends in a new school and was also having frequent angry feuds with his mother, so he took his savings and went across the country. There he became involved with a homosexual group who, in exchange for his performing homosexual acts, housed and fed him until he was found by the police and returned home. He continued to fight bitterly with his mother and adopted an ideal of himself as a "tough guy with a chip on my shoulder." Finally, Stephen struck his mother one night, and she had him detained at a juvenile center for 2 weeks. Following Stephen's release, his mother became increasingly angry at his "entitled," aggressive, and sadistic behavior, and she decided that he would be better off at a relative's farm far from home. Consequently, he was sent there without plans for his return.

Although Stephen made a transient adjustment to the farm, he soon became disappointed and embittered. He left the farm and roamed the countryside stealing from small stores to sustain himself. Eventually he was discovered and sent to jail. He continued his aggressive behavior in jail and managed to have himself placed in solitary confinement, where he attempted to hang himself "because I knew they would send me home." Stephen's return to his mother was arranged, and upon arrival she brought him to the Center for his first admission as described above.

Stephen's thought disorder, noted on admission, remitted after 2 weeks while he was receiving 800 mg/day orally of chlorpromazine. However, his course in the hospital and in therapy remained tumultuous in spite of his receiving an additional 40 mg/day orally of trifluoperazine. Stephen's profound ambivalence toward his mother was always apparent. His mood seemed to be dependent upon her daily visits, during which time the two of them could easily have been mistaken for husband and wife. She often held his hand, and occasionally she lay next to him on a couch on the ward. This behavior was so open that he reported to a nurse during his first month in the hospital, "I love my mother as a husband rather than a son." Meanwhile, he repeated his "entitled," aggressive behavior with the staff and other patients as if they were extensions of his own family. Almost daily he taunted geriatric patients for their infirmities, spat at and struck patients and staff, made frequent escapes from the hospital, stole food and money from patients, and provoked fights between other patients.

During this prolonged period, Stephen's therapy hours could be viewed as a battle for control and dominance. The therapist initially became his "jailer" and, subsequently, the man who came between him and his mother. Therapeutic efforts during the first year centered around Stephen's concept of what one needed to do in order to be a man, and he became aware of how frightened, lost, and confused he often felt. It was called to Stephen's attention that he acted like his father as a way of feeling like a man; we hoped that this would provide an avenue for his developing alternative male role models. However, during the first 2 years of Stephen's hospitalization little, if any, progress was made.

After 3 months of treatment, Stephen developed a near-delusional notion that he would be hurt by another patient during the time his therapist was on vacation. While his doctor was away Stephen was increasingly aggressive on the ward, and following his doctor's return, he seemed more distant in the therapeutic relationship. He complained that his doctor had betrayed him. Over a 7-month period he again began a slowly escalating pattern of impulsive behavior, with frequent escapes, sadistic and aggressive behavior on the ward, and a determined effort to sabotage the therapeutic interventions. Toward the end of this 7-month period, he robbed a store during one escape "so I could get kicked out of the hospital." Indeed, at this time, the therapist and ward staff decided that Stephen could no longer be managed on an open psychiatric ward. Stephen committed the robbery in a way that invited apprehension, so he was arrested, sent to a juvenile detention center (instead of the Massachusetts Mental Health Center), and, subsequently, transferred to a psychologically oriented boys' residential school. Shortly before the end of his 30-day trial period at that school and immediately before his return to court for a final disposition, he drank a large amount of a dangerous substance. At this point another relative intervened and arranged for Stephen's hospitalization at yet another institution. There he received high doses of phenothiazines and 17 ECT sessions in 5 weeks. After running out of funds, the family delivered Stephen back to the Massachusetts Mental Health Center for readmission.

Upon Stephen's return to the Center the therapist and the ward staff made a new commitment to stay with him and treat him in spite of all obstacles. Initially, as he talked more openly about his ambivalent and seductive relationship with his mother, his alliance with the staff and therapist was improved. In a family session several months after the second admission, the following exchange took place:

Social worker: How do you feel toward your mother, Stephen?

Stephen: Like I'm her husband, like I'm the man of the house.

Mother: Stephen, you don't mean that. You never act that way.

His mother's denial was characteristic, but Stephen's statement was his first attempt at making explicit to her his distress in their relationship.

When Stephen's therapist planned another vacation, Stephen repeated his pattern of increasingly aggressive behavior. Attempts at using a behavior modification approach were ineffective. He inevitably sabotaged any movement toward increased privileges based on his good behavior by repeatedly behaving in such a way as to be restricted by the ward staff.

During this extended period of time Stephen's feelings toward his mother and therapist were obscured by his behavior. After another dangerous overdose several months later, he related to the therapist the content of a previously hidden

psychotic transference: "I thought if I got rid of myself, you could get married to my mother and be happy." Shortly thereafter, without a clear immediate precipitant, Stephen impulsively destroyed some expensive hospital property.

At this time it was clear that Stephen's treatment had reached an impasse. A consultation with another psychiatrist was arranged in the hope of finding a useful approach to this explosive situation. During the consultation interview Stephen described his aggressive and rivalrous feelings toward his mother's boyfriends and her apparent acceptance of him as the victor. With some encouragement from the consulting psychiatrist he was able, for the first time, to begin to speak explicitly about feelings of sexual excitement in his mother's presence, including arousal "below my belt." What appeared to be unmitigated aggression toward his mother could now be seen in part as an expression of sexual excitement toward her as well as a defense against those feelings.

Stephen's mother was also interviewed as part of this consultation and her seductive relationships with men in general and with Stephen in particular were discussed, although she vigorously denied the latter. The crucial ingredient in the consultation was that the consulting psychiatrist gave permission and encouraged the therapist and the social worker as well as Stephen and his mother to discuss directly the heretofore unspoken explicitly sexual aspects of the relationship between Stephen and his mother. What, then, were the consequences of this approach to the therapeutic impasse?

As we had feared, Stephen's assaultiveness and excitement increased for the rest of the day immediately following this first verbalization of sexual arousal in relation to his mother. Indeed, following one of the two consultation interviews he assaulted his therapist. Over a 2-week period, however, Stephen's high level of excitement, aggression, sadism, and impulsivity diminished and these symptoms remained at low levels during the 6-month period before Stephen's therapist was to complete his residency. During this time the following behavioral observations were made: Stephen's assaultive episodes diminished from once a week to once a month. His antisocial acts disappeared, except for one incident of stealing money from another patient. Stephen's epsiodes of meanness declined from nearly once a day to none at all, and his performance and attendance at the Center's high school improved dramatically.

Stephen's therapy evolved as follows. He experienced a gradual but definite sense of relief as he became able to acknowledge and share with his therapist his feelings of sexual excitement toward his mother and the accompanying shame and guilt. For example, he told his therapist, "I had feelings in my penis when talking with my mother on the telephone."

Perhaps of even greater significance than verbalizing his sexual excitement toward his mother was the fact that he also began expressing homosexual longings toward his doctor, although the longings were displaced to the past. "A year ago I wanted to blow you," he told the doctor. Stephen confided that he wanted to gain strength from the doctor in this way, and he recalled that at the time of admission he felt that he had "lost his sperm." After unburdening himself in words of these painful secret wishes, for the first time he began looking at the therapist and other people directly in the eye, which he had previously been unable to do. Concomitant with his ability to discuss these feelings directly, he showed a striking improvement in his behavior during the treatment hours. His affective presence was fuller. He would experience open anger, sadness, and at times laughter and joy in the therapist's presence. A therapeutic alliance had been established, and it was now possible for Stephen to discuss his feelings in a setting in which he felt accepted.

We observed Stephen's course over the 6½-year period following the intervention. During this time Stephen was treated in psychotherapy by two residents; each saw him for 3 years. Although Stephen remained in the hospital for 1½ years after the original resident therapist left the Center, Stephen made gradual but definite progress, including longer periods of psychosis-free intervals and decreasing destructive and self-destructive threats and behavior. After his discharge Stephen experienced several acute psychotic episodes requiring progressively briefer readmissions. These episodes were clearly precipitated by his therapists' vacations and the births of more siblings. During Stephen's therapy with the third resident, he did not require hospitalization for nearly 2 years. As we had come to anticipate, however, he required hospitalization in an acute psychotic state around the time of termination of therapy with this resident.

Not surprisingly, Stephen's powerful symbiotic tie to his mother did not diminish significantly with all the years of treatment. Over the 6½-year period following the intervention, Stephen was able to separate his living quarters from his mother's by moving to the apartment across the hall from her. Stephen's tie to his mother and the hospital remained deeply ambivalent, although his aggressive side was far more rarely expressed through violence.

DISCUSSION

An important aim in analytically oriented psychotherapy is for the patient to verbalize the memories and feelings that are acted out (1). A treatment impasse led us to examine why such a basic principle of analytically oriented psychotherapy is frequently overlooked when one works with an impulsive, intermittently psychotic young man. First, we noted that the patient's continued violent behavior served to distract the staff and himself and to preoccupy the staff with efforts to control the patient's behavior or send him elsewhere.

Second, although the highly sexualized relationship with his mother was always blatantly obvious to the most casual observer, we found ourselves reluctant to discuss this issue directly with Stephen and his mother. It continually appeared to us that matters were bad enough without adding any further stimulation to the situation. Indeed, Stephen became acutely assaultive immediately after the two consultation interviews in which he was encouraged to pay attention to his feelings of excitement while in his mother's presence. Thus, it seemed that encouraging Stephen to discuss these feelings of excitement in his body was the last thing one would consider doing with a young man already so excited that he seemed to be jumping out of his skin.

We attempted to gain a dynamic understanding of

our patient's behaviors by dividing them into two categories—the outwardly aggressive type and the inwardly aggressive type. The outwardly aggressive type of behavior included hitting his mother, running off the ward, breaking into a store, stealing, and being assaultive and destructive in the hospital. The inwardly aggressive type included self-destructive behaviors such as attempted hanging and swallowing dangerous substances. Both types of behavior were, of course, multidetermined. They had in common that each behavior was at the same time 1) the expression of a sexualized aggressive impulse, often experienced by Stephen around the time of separations from his mother or therapist, 2) a cry for both a protective and punitive response for this impulse, and 3) an avoidance of memory insofar as the massive motor discharge replaces memory. Typically, Stephen's behavior included a manipulative component and a genuinely self-destructive component, as expressed in his statement, "I know they would send me home if I tried to hang myself."

Because of Stephen's partial failure of repression, he experienced closeness with his mother in a highly undifferentiated, threatening, sexualized, and aggressive way. These feelings were transferred quite directly to the therapist, who was seen sometimes as father and sometimes as mother. In the psychotic transference mentioned above, one aspect of Stephen's attitude toward his therapist is dramatically expressed: "I thought if I got rid of myself, you could get married to my mother and be happy." This would be one solution to Stephen's quandary over his forbidden longings for his mother in the absence of his father. His therapist thus became the father in the transference and the childhood struggles and wishes were recreated and reenacted. Thus, childhood longings—both sexual and aggressive (as well as guilt about such longings)—were directed toward the therapist in the transference and his mother, the available primary object. His mother's erratic and seductive behavior kept alive Stephen's fantasy that his infantile longings would be gratified and thereby helped to maintain his pathological state.

Separations from his mother and therapist were experienced by Stephen with a powerful surge of the most intense and unacceptable longings and feelings. Stephen's impulsive behavior could now be understood as acting out in the transference, which revived past conflicts with both parents in the present experience with his therapist.

In summary, we make no claim to have cured Stephen's severe and chronic underlying psychosis by these verbal interventions. We do assert, however, that the explicit clarification of his preconscious sexual feelings toward his mother helped him to overcome the impasse that threatened to make further therapy impossible. The clarification allowed Stephen's ego to cope more effectively with these unacceptable impulses. From a technical point of view, it should be noted that Stephen's clarification involved preconscious impulses. By contrast, premature interpretation of unconscious impulses would lead to increased resistance toward recognition and remembering, which was clearly not the case here.

Following the consultation interview, the therapist continued discussing with Stephen in relative ease Stephen's explicit sexual impulses toward his mother and the therapist. The effect was to allay Stephen's anxiety and self-doubt. As the therapist's date of departure approached, the therapist was able to accomplish a degree of working through these issues. As noted above, Stephen did not significantly withdraw from his mother. The maternal side of the transference toward the therapist and the hospital remained rather stable following the intervention. Usually, the therapist and the hospital were experienced by Stephen as protective and nurturant. At times, of course, both were experienced as punitive and restrictive, like the cruel, retaliating father.

During therapy with the next two residents Stephen's explicit sexual clarification, which we viewed as having been crucial in overcoming the specific therapeutic impasse, moved into the background. The homosexual transference, which was at times clearly present in this late phase of therapy, was discussed by the therapist as a wish for closeness; the sexual aspects were de-emphasized. Stephen's affectionate concern for the consulting psychiatrist who first encouraged Stephen to verbalize his fantasies was typical in this phase of the therapy; he felt toward this consultant as he did toward his consistently caring grandfather. He would ask his therapist with a warm, humorous smile, "How is that crazy, dirty old man who thought I was interested in sex with my mother?"

Putting the unspeakable sexual feelings toward his mother and the therapist into words appeared to be useful to Stephen in two ways. Words permitted Stephen to express his wishes, thereby decreasing somewhat his intolerable physical excitement, and he no longer needed to provoke punishment and protection through his impulsive behavior because the therapist encouraged expression of these wishes. We have described the familiar analytic process of repeating, remembering, and working through (1). Or, as Dr. Semrad (10) stated, "The patient must acknowledge the unacceptable, intolerable feelings, bear them, and share them with the therapist, in order that he may put them in perspective for himself."

Our thesis is that impulsive behavior in patients like Stephen is most usefully viewed as classical acting out under the influence of the therapy rather than as a random, meaningless, breaking through of impulses. Both the therapist and the patient need permission to discuss these painful affects and fantasies in a therapy setting in order to overcome this type of therapeutic impasse.

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BRIEF COMMUNICATIONS

Poor Premorbid Adjustment and CT Scan Abnormalities in Chronic Schizophrenia

BY DANIEL R. WEINBERGER, M.D., ELEANOR CANNON-SPOOR, M.A., STEVEN G. POTKIN, M.D., AND RICHARD JED WYATT, M.D.

The authors evaluated the premorbid adjustment of 51 chronic schizophrenic patients by retrospective chart analysis using a novel scale. Those patients with CT evidence suggestive of brain atrophy had significantly worse premorbid scores, particularly during childhood, than did the patients with normal scans. This finding provides further support for the clinical relevance of CT findings in chronic schizophrenic patients and implicates a neuropathological process that occurs early in development.

Subtle manifestations of brain atrophy, specifically ventricular enlargement and dilatation of cortical sulci, have been observed on computed tomography (CT) scans (1–7) and pneumoencephalograms (8–10) of schizophrenic patients. What is unclear from these studies, however, is whether such abnormalities are related to the pathogenesis of the illness or to nonspecific factors to which schizophrenic patients are particularly vulnerable. If the latter possibility is the case, it might be expected that patients with more lengthy illnesses would be more likely to manifest the abnormalities. In studies of patients under age 50 (2, 3, 5, 6), this has not been found. The abnormalities also do not appear to be the result of various treatment modalities, including institutionalization, ECT, and neuroleptic drugs (1–3, 5, 6).

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To the extent that the CT findings may relate to the pathogenesis of a schizophrenic illness, they might be associated with a distinct manner of onset or premorbid history. For example, if patients with these structural brain abnormalities were found to have had a normal premorbid history, it would implicate an acute postpubertal neuropathological episode, perhaps a viral illness (11). On the other hand, if patients with the abnormalities appeared to be an impaired group from childhood, then a neuropathological process in early development that predisposed to schizophrenia could be hypothesized. In this study we examine these possibilities by evaluating the relationship between premorbid adjustment and CT abnormalities in schizophrenic patients.

METHOD

Fifty-one voluntary inpatients (12 women, 39 men; mean age, 29; age range, 20–50) on three NIMH research wards of St. Elizabeths Hospital, Washington, D.C., who fulfilled Research Diagnostic Criteria (12) for chronic schizophrenia comprised the study sample. In general, patients accepted into the research program had failed to respond to conventional forms of therapy sufficiently so that they could live independently in the community. Each patient had undergone CT scanning, and measurements from their scans were made of the size of the lateral ventricles and of various cortical fissures and sulci. These quantitative measures have been described in detail elsewhere (2, 3). Twenty-one patients had abnormal scans as defined by a ventricular or sulcal size in excess of the range of 62 similarly aged asymptomatic individuals who had been previously studied (13). Nineteen patients (6 women and 13 men) had enlarged ventricles (ventricular-brain ratio more than 10.6), and 8 patients also had dilated

sulci. Two of the patients with dilated sulci, both of whom were men, had normal ventricles.

A novel instrument that includes selected items from the Phillips (14) and Gittelman-Klein (15) premorbid adjustment scales and the Elgin prognostic scale (16) was used to assess premorbid adjustment.[1] The rationale for combining these various items into a new format was to develop a scale that would measure adjustment during several specified age periods in addition to scoring overall premorbid adjustment. The new scale has 28 items divided among 5 subscales, 4 of which focus on various aspects of social and school functioning during the following age periods: childhood (younger than age 12), early adolescence (ages 12–15), late adolescence (ages 16–18), and adulthood. In addition, it has a general item scale that attempts to assess the highest level of premorbid adjustment achieved.

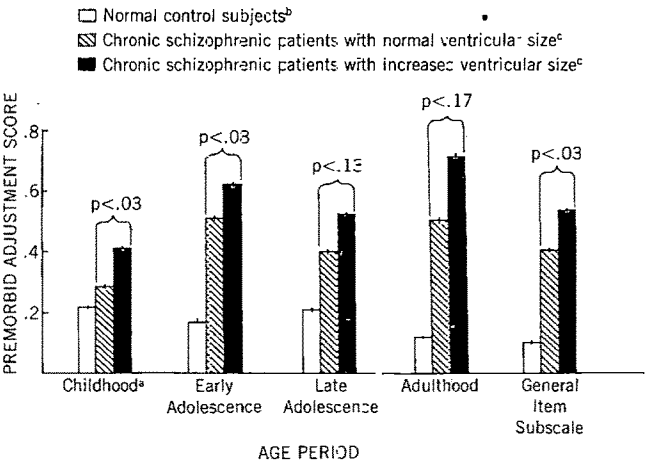
One of us (E.C.-S.) who was unaware of the CT scan findings completed the scale for each patient by reviewing the patient's chart. The research charts generally include detailed premorbid histories that are derived from family meetings and records from referral sources. If the charts did not contain sufficient information to complete a particular item, the item was left unrated. To account for unratable items, the score for each age period was expressed as a fraction of the total possible score for the ratable items. Thus the lower scores correspond to better adjustment. Every age period was not ratable in every patient due to insufficient historical data. To help standardize the ratings, the instrument was used by the same individual in an interview format to assess premorbid adjustment of 78 enlisted personnel stationed at an Air Force base in Washington, D.C. Statistical comparisons were done by a two-tailed t test.

RESULTS

To the extent that chart reviews and personal interviews are comparable, the premorbid adjustment of the group of 51 chronic schizophrenic patients was significantly worse than that of the Air Force personnel as measured on the subscales during childhood (mean scores ± SD = .33±.2 versus .23±.1, $p<.001$), early adolescence (.44±.2 versus .21±.1, $p<.0001$), late adolescence (.55±.3 versus .17±.1, $p<.0001$), and adulthood (.58±.3 versus .12±.1, $p<.0001$), as well as on the general item subscale (.44±.2 versus .09±.1, $p<.0001$). Figure 1 shows the mean scores for premorbid adjustment for the Air Force personnel and for the schizophrenic patients subdivided according to ventricular size. The patients with enlarged ventricles scored consistently worse than the other patients; the scores during childhood and on the general item subscale reached statistical significance ($p<.05$). When all the patients with CT abnormalities (the 19 patients with enlarged ventricles plus the 2 patients with only sulcal dilatation) were compared with the schizophrenic patients with normal scans (see figure 2), the differences in premorbid adjustment during early adolescence reached statistical significance as well. Of the 9 patients with the poorest adjustment during childhood (those whose rating was more than .50) all had abnormal CT scans. In contrast, of the 13 patients with good childhood adjustment (rating less than .20), only 3 had abnormal scans ($p<.001$, Fisher exact test).

[1] The instrument is available on request from Ms. Cannon-Spoor.

FIGURE 1
Comparison of Premorbid Adjustment Mean Subscale Scores of Normal Control Subjects, Chronic Schizophrenic Patients with Normal Ventricular Size, and Chronic Schizophrenic Patients with Increased Ventricular Size[a]

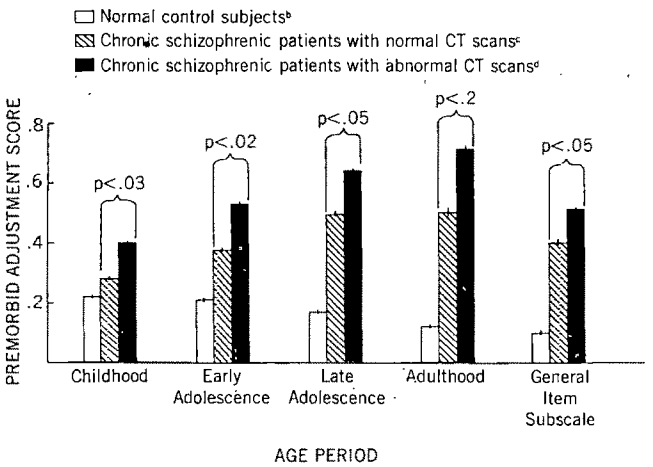


[a] The brackets indicate the comparison made to obtain p value given.
[b] Data were available for 78 control subjects for the general item subscale and for all age periods except adulthood, where N=69.
[c] Data were available for 32 patients in childhood and early adolescence, 29 in late adolescence, 10 in adulthood, and 33 for the general item subscale.
[d] Data were available for 19 patients in childhood, 18 in early adolescence, 15 in late adolescence, 6 in adulthood, and 18 for the general item subscale.

DISCUSSION

It is not uncommon in medicine for nonspecific findings to lack clinical significance in one clinical context while being meaningful in another. For example, cortical sulcal dilatation in asymptomatic elderly individuals may be clinically irrelevant while in young patients with systemic lupus erythematosis it is associated with psychosis (17, 18). Ventricular enlargement and dilated cortical sulci in schizophrenic patients are certainly nonspecific findings, since they are common in the elderly and in many neurological disorders. They have also been described in patients with chronic severe migraine headaches (19), in alcoholics (20), in patients with affective disorders (5, 10), and in autistic children (21). However, since ventricular enlargement

FIGURE 2
Comparison of Premorbid Adjustment Mean Subscale Scores of Normal Control Subjects, Chronic Schizophrenic Patients with Normal CT Scans, and Chronic Schizophrenic Patients with Abnormal CT Scans[a]



[a]The brackets indicate the comparison made to obtain p value given.
[b]Data were available for 78 control subjects for the general item subscale and for all age periods except adulthood, where N=69.
[c]Data were available for 30 patients in childhood, 31 in early adolescence, 27 in late adolescence, 10 in adulthood, and 31 for the general item subscale.
[d]Data were available for 21 patients in childhood, 20 in early adolescence, 17 in late adolescence, 6 in adulthood, and 20 for the general item subscale.

is not itself a pathological process but the result of one, it is this underlying process which may be specific.

The results of this study indicate that CT abnormalities in chronic schizophrenic patients are associated with poor premorbid adjustment. Even in a sample of generally poor premorbid patients, the CT scan criteria segregated out a significantly more impaired subgroup. This finding provides further support for the idea that these CT scan findings help define a more homogeneous subgroup of chronic schizophrenic patients. In addition to poorer premorbid adjustment, patients with these abnormalities have been shown to have greater neuropsychological deficits (1, 4, 6, 22), poorer response to neuroleptic drugs (23), more nonfocal neurological signs ("soft signs"),[2] more impaired smooth pursuit eye movements,[2] a different distribution of neuroanatomical asymmetries seen on CT scans (24), a different frequency of human leukocyte antigen (HLA) A-2 (25), and higher whole blood serotonin concentration (26). Moreover, a recent study by Kleinman and associates (27) found that certain manifestations of brain dopaminergic function may be different in these patients than in clinically similar patients with normal CT scans. Taken together, this body of research strongly suggests that although the CT scan findings are themselves nonspecific, they are biologically and clinically meaningful characteristics of these schizophrenic patients and, perhaps, markers for etiologically homogeneous populations.

The relationship of poor premorbid adjustment to CT scan abnormalities also may have implications for understanding the pathogenesis of schizophrenia in this subgroup of patients. It suggests that in such patients a neuropathological process that predisposes to or eventually causes schizophrenia in late adolescence occurs or begins early in development. However, although the results of this study seem to support this assumption, the data could also indicate that for unknown reasons schizophrenic patients with poor premorbid adjustment are more vulnerable to nonspecific factors that produce brain atrophy after the onset of their illness.

Another perspective on these findings is to consider the degree to which the clinical implications of poor premorbid adjustment correspond to the clinical implications of the CT scan findings. Like the patients with CT scan abnormalities, schizophrenic patients with poor premorbid adjustment tend to have a poor prognosis (28), to respond poorly to neuroleptic drugs (29), and to have nonfocal neurological signs (30). On the basis of these observations, Quitkin and associates (30) proposed that patients with poor premorbid adjustment are, in fact, "brain damaged." It appears that the CT scan findings partially confirm this notion. Indeed, if they are linked to the pathogenesis of some forms of schizophrenia, poor premorbid adjustment may be a manifestation of the illness rather than a predisposing or prognostic factor.

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Pharmacokinetic Study of Lorazepam Overdosage

BY MARCIA DIVOLL ALLEN, DAVID J. GREENBLATT, M.D., YVES LACASSE, M.D., AND RICHARD I. SHADER, M.D.

The authors describe three patients who attempted to commit suicide by ingesting overdoses of lorazepam, an intermediate-acting benzodiazepine that, unlike diazepam, has no active metabolites. All three patients were clinically well within 24–30 hours of ingestion, although high lorazepam concentrations persisted. The authors point out that the elimination half-life of lorazepam after extremely high doses have been ingested is approximately the same as that found after ingestion of usual therapeutic doses.

The extensive prescribing of benzodiazepines in clinical medicine has led to their frequent use for deliberate self-poisoning. Previous studies of overdosage with diazepam, a long-acting benzodiazepine with active metabolites, suggest that diazepam by itself rarely produces life-threatening intoxication, even when ingested doses and plasma concentrations are extremely high (1, 2). Furthermore, clearance of diazepam and its metabolites after massive overdosage appears similar to that observed after therapeutic doses (2). We will describe three cases of lorazepam self-poisoning with suicidal intent. Lorazepam is an intermediate-acting benzodiazepine that has no active metabolites.

CASE REPORTS

Case 1. Mr. A, a 20-year-old man, ingested an unknown amount of lorazepam with five beers approximately 2 hours before admission. His vital signs on admission were as follows: blood pressure, 120/90 mm Hg; heart rate, 92 beats/minute; and respirations, 20 per minute and spontaneous. The patient was lethargic but easily roused. Laboratory findings were within normal limits. The patient was lavaged and received activated charcoal. He was placed under observation with no specific therapy and discharged 24 hours later with no sequelae.

Case 2. Ms. B, a 23-year-old woman, ingested an estimated 120 mg of lorazepam approximately 18 hours before admission. Her clinical history also implicated ingestion of 750 mg of methotrimeprazine and an unknown amount of trihexiphenidyl. Her vital signs on admission were as follows: blood pressure, 140/90 mm Hg; heart rate, 96 beats/minute; and respirations, 20 per minute and spontaneous. The patient was responsive to light pain. Laboratory studies and arterial blood gases were normal. After gastric lavage, Ms. B received activated charcoal and intravenous fluids. She was placed under observation with no specific therapy. She was fully alert 12 hours after admission and was discharged with no sequelae.

Case 3. Mr. C, a 46-year-old man, ingested an estimated 100 mg of lorazepam 1 hour before admission. His clinical history also implicated ingestion of an unknown amount of doxepin and four glasses of wine. His vital signs on admission were as follows: blood pressure, 126/90 mm Hg; heart rate, 140 beats/minute; and respirations, 25 per minute and spontaneous. Mr. C was responsive to light pain. Laboratory results and arterial blood gas determinations were normal. Mr. C received naloxone, 50% dextrose solution, and thiamine intravenously and did not respond. He was placed under observation with no specific therapy. He was fully alert and responsive 24 hours after admission and was discharged 2 days later with no sequelae.

METHOD

Venous blood samples were drawn into heparinized tubes at multiple points in time during each patient's hospitalization. When the patient was alert and oriented, informed consent was obtained. Plasma was separated and stored at −20°C until the time of assay. Plasma concentrations of lorazepam were determined by electron-capture gas-liquid chromatography (3).

RESULTS

Figure 1 shows plasma concentrations of lorazepam for up to 60 hours after ingestion in all three patients.

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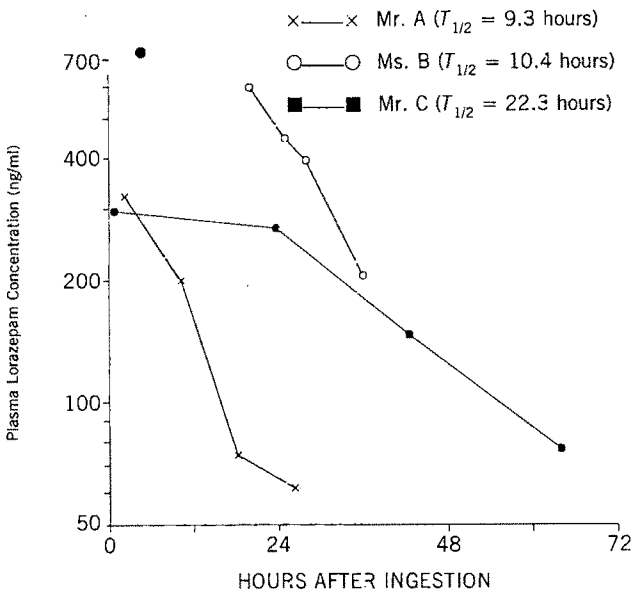
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FIGURE 1
Plasma Lorazepam Concentrations in Three Patients After Lorazepam Overdosage[a]



[a]Values of elimination half-life ($T_{1/2}$) were determined by least squares regression analysis.

Initial plasma lorazepam levels were 5 to 15 times higher than those associated with ingestion of usual therapeutic doses. Values of lorazepam elimination half-life were 22.3, 10.4, and 9.3 hours, respectively.

DISCUSSION

The use of benzodiazepines as agents of self-poisoning is increasing, consistent with the increase in their availability and therapeutic use. Little has been reported on the consequences of overdosage with lorazepam, a benzodiazepine recently introduced into clinical practice (4–6). This report describes three patients who attempted to commit suicide by ingesting overdoses of lorazepam.

On admission all three patients were responsive, although lorazepam plasma levels were as high as 600 ng/ml, greatly exceeding steady-state concentrations of lorazepam reported in normal volunteers taking usual therapeutic doses (17–50 ng/ml) (7). All three patients recovered rapidly and were clinically well within 24–30 hours of ingestion, although high lorazepam plasma concentrations persisted.

The elimination half-life of lorazepam in these individuals fell within the range observed in healthy persons after ingestion of usual therapeutic doses (8–25 hours) (7, 8). The longer half-life in patient 3 might be explained by concurrent treatment with a tricyclic antidepressant (9). In any case, the process of lorazepam biotransformation to its pharmacologically inactive glucuronide metabolite does not appear to be saturable at high doses.

Previous pharmacokinetic studies of diazepam overdosage also indicated normal elimination of the parent compound and its metabolites (2). High plasma concentrations of diazepam and its metabolites persisted long after patients were alert and clinically well. Although excessive sedation is an expected consequence of overdose with a central nervous system depressant, sedative effects may diminish or disappear long before plasma drug levels return to the therapeutic range. Tolerance or adaptation to central depressant effects may explain these observations.

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Seizures Terminable and Interminable with ECT

BY RICHARD D. WEINER, M.D., PH.D., MICHAEL R. VOLOW, M.D., DANIEL T. GIANTURCO, M.D., AND JESSE Q. CAVENAR, JR., M.D.

The authors discuss the incidence, detection, management, and significance of prolonged seizures with ECT and present a case example. They suggest that the incidence of this phenomenon may be underreported and that its occurrence may be linked to hyperoxygenation, the use of multiple-monitored ECT, and preexisting states of cerebral hyperexcitability. Since prolonged seizures may result in adverse metabolic changes, prompt detection and intervention are essential.

ECT is acknowledged as an effective treatment in psychiatry, with its therapeutic potency derived from the induction of a series of generalized seizures in susceptible individuals. One of the advantages of electricity over pharmacologic agents as a mode of inducing seizures has been that the ictal duration with ECT is relatively brief, usually less than a minute. At the same time, however, the use of modern refinements in ECT technique such as muscular relaxation, oxygenation, and, most recently, multiple-monitored ECT (in which multiple seizures, monitored electroencephalographically, are produced during a single period of anesthesia) appears to have increased the incidence of prolonged seizures, although unilateral ECT, at least with sinewave stimuli, may act to decrease seizure duration. As will be discussed later, the actual incidence of prolonged seizures may be somewhat greater than would be expected, in part due to the relatively low use of EEG monitoring. Despite the fact that prolonged seizures represent a potential risk for the development of neurologic sequelae and are not associated with increased therapeutic benefits, this phenomenon has not been adequately dealt with in the psychiatric literature, and many practitioners are not aware of its significance, detection, and management.

Accordingly, using a recent clinical case example, we will attempt to outline some of the problems presented by a prolonged seizure with ECT. In particular, this case demonstrates that a seizure of only 6–10 minutes' duration can be associated with both metabolic insufficiency and delayed return to baseline neurologic function, even in the presence of apparently adequate oxygenation. We will also discuss the possible role of an underlying state of cerebral hyperexcitability.

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CASE REPORT

The patient was a 26-year-old man with a diagnosis of schizoaffective schizophrenia, manifested by auditory hallucinations, delusions of thought control, depressed affect, and suicidal ideation. He had a history of polydrug abuse that included apparent daily intake of marijuana and intermittent use of alcoholic beverages, diazepam, oral amphetamines, cocaine, and hallucinogens. When he was admitted, his prescribed medication consisted of 1000 mg of chlorpromazine daily. There was no known personal or family history of epilepsy or other neurologic conditions and no family history of psychiatric illness.

After 15 days of imipramine, 200 mg, in addition to chlorpromazine, the level of the patient's suicidal ideation had markedly increased, and accordingly a laboratory evaluation for ECT was initiated, revealing only a borderline abnormal EEG, with brief bursts of bilateral sharply hypersynchronous 7–8 Hz theta rhythms, which were not considered significant.

As soon as the decision to use ECT had been made and informed consent for ECT obtained, imipramine and chlorpromazine therapy were discontinued. After 5 days the patient received his first electroconvulsive treatment, which was modified by atropine (.6 mg subcutaneously, 30 minutes before the treatment), methohexitol (60 mg i.v.), and oxygen (100% by Ambubag, via oral airway, beginning at the onset of anesthesia and continuing until resumption of spontaneous respirations, except for a period of a few seconds at the time of stimulation). The electrical stimulation was provided by an 800-mA bipolar pulse device (MECTA Corp.) with the following settings: 60 pulse pairs per second, .75-second pulse-width, and .75-second stimulus duration. The stimulus electrodes were in the standard bifrontotemporal location. A single channel of bifrontal EEG was monitored.

The initial stimulus (intensity of 6 joules) did not produce a seizure either behaviorally or electroencephalographically, and after approximately 15 seconds a second stimulus was applied, with the duration increased to 1.25 seconds. This stimulus (11 joules) produced a modified tonic-clonic convulsion, estimated by several observers to have lasted less

than 60 seconds. Electroencephalographically, however, the seizure continued long past the termination of the clonic movements. After several minutes clonic movements involving the rostral half of the body appeared; they lasted approximately 45 seconds and were presumably concurrent with the metabolism of the succinylcholine, since no further relaxant or anesthetic was given. At 5½ minutes, the EEG seizure activity continued to be present and diazepam, 5 mg i.v., was given. Forty seconds later, with seizure activity still present in the EEG, the paper in the chart recorder ran out. Several minutes later, while the paper was being changed, movements of the patient's face and upper extremities again occurred, but on a more disorganized basis.

Because of concern that the motor activity might represent recurrent seizure activity, a second dose of diazepam, 5 mg i.v., was given. By this time, the chart recorder was again operating, but EEG activity was obscured by muscle and movement artifact for the next 2 minutes, following which a clear period of postictal suppression was present. Because the irregular movements persisted slightly into the period of postictal suppression, it is likely that they may have been part of a postictal delirium, rather than status epilepticus, and that the actual length of the seizure had been 6–10 minutes. The EEG was continuously monitored for the next 15 minutes and revealed an unusually prolonged period of postictal suppression, with activity consisting only of low amplitude delta rhythms.

During this postictal period, approximately 15–20 minutes after seizure onset, while the patient was still unconscious on 100% oxygen but breathing spontaneously (the respiratory rate was not recorded), arterial blood gases were measured; they revealed a mild acidosis (pH=7.26), with hypercapnia (pCO_2=51), but no anoxia (pO_2=85, O_2 saturation=95%). Measurement of arterial blood gases 30 minutes later showed a resolution of the above changes (pH=7.50, pCO_2=35). It should be noted that no cyanosis was present at any time. Values for serum electrolytes, calcium, magnesium, and glucose, measured during the acute postictal period, were all normal. During this time, examination revealed hypertonia and extensor plantar reflexes. When the patient regained consciousness 25–30 minutes after seizure onset, he was markedly confused.

Within a few hours, a computerized tomographic brain scan was obtained, both with and without radionucleotide enhancement; it showed bilateral enlargement of the cerebral ventricles, without any evidence of cortical atrophy. Approximately 8 hours postictally, a lumbar puncture was carried out, with completely normal findings. An EEG done one day postictally showed an increase in the amplitude, duration, and incidence of the bilaterally synchronous paroxysmal bursts noted on the pre-ECT record; a second EEG 1 day later revealed a partial resolution of these electrographic changes. Neurological examination 1 day postictally continued to show evidence of symmetrical hypertonia and hyperreflexia with bilateral ankle clonus. The patient was also noted to be profoundly amnestic for events happening even hours after the ECT.

Behaviorally, the patient's affective disturbance began to clear somewhat over the next few days, and his psychotic ideation appeared to diminish in intensity. At the same time, however, he became uncooperative and refused further medical evaluation. He was discharged against medical advice 7 days after ECT.

DISCUSSION

A review of the literature has revealed only one previous report of a seizure lasting longer than 5 minutes when ECT was given at standard intervals of 3 times weekly (1). In that series the use of continued oxygenation before and throughout the seizure was felt to have significantly increased seizure duration. However, in the more recent technique of multiple-monitored ECT, in which two or more EEG-monitored seizures are evoked during a single period of anesthesia, prolonged seizures occur on a much more frequent basis (2–4), lasting as long as an hour (5). The only previous report of neurologic sequelae associated with prolonged seizure activity occurred with a multiple-monitored ECT session of four seizures, the last of which continued for 25 minutes and was followed by a 28-minute period of status epilepticus (6).

The fact that prolonged seizures have been reported only in the presence of EEG monitoring raises the question of whether this phenomenon actually occurs on a more frequent basis. As noted in the present case, seizure activity can continue long past the end of the convulsive motor activity. Without EEG monitoring, an undetected prolonged seizure might appear to be a prolonged postictal period of apnea or coma and would probably be ascribed to such factors as succinylcholine deficiency, idiosyncratic reactions to the anesthetic agent, or an unusually persistent and severe postictal state (7). At the same time, however, even with EEG monitoring, prolonged seizures appear to be uncommon except with multiple-monitored ECT. A recently studied series of 40 patients given ECT 3 times weekly had no seizures longer than 2 minutes, even with continuous oxygenation during the seizure (8).

Clinical and experimental data suggest that in the adequately oxygenated and relaxed patient, prolonged seizures can be tolerated without the development of ischemic changes (9). However, the case we have described indicates that ventilation with an oral airway may be insufficient to compensate for the increased metabolic demands of a prolonged seizure. This suggests that, under such circumstances, the patient be intubated and the seizure terminated with intravenous diazepam to minimize the risk of related morbidity. Bridenbaugh and associates (5) have suggested lowering ventilation rate and intensity in order to allow the seizure to be terminated by the resulting hypoxemia and hypercapnia; however, if ineffective and continued for a long time, such a procedure could actually add to the morbidity. Oxygenation only before and after the seizure or the use of room air represent potential means of minimizing the role of oxygen in prolonging the seizure, but they are not optimal from a metabolic standpoint (10).

The etiology of the prolonged seizure in the case we have described remains uncertain. The presence of preexisting paroxysmal EEG findings might suggest a

latent state of CNS hyperexcitability (11). In this regard, Bagchi and associates (12) have reported that nonepileptics with isolated paroxysmal EEG abnormalities occasionally showed increased paroxysmal activity after ECT. Furthermore, Hill and Parr (13) have reported seizures during insulin coma treatment or after catatonic episodes in nonepileptic schizophrenic patients possessing such paroxysmal abnormalities. The possibility that other exogenous factors, such as withdrawal of sedative hypnotic drugs, may have contributed to the prolongation of our patient's seizure is somewhat doubtful in view of the 20-day hospitalization preceding the ECT and the absence of clinical or EEG signs of withdrawal. However, the role of chlorpromazine and imipramine cannot be ruled out, even though they had been discontinued 5 days previously, since these agents, which increase cerebral excitability, have rather long half-lives.

The finding, in our patient, of cerebral ventricular dilation is consistent with reports (14) that some chronic schizophrenic patients may tend to have enlarged cerebral ventricles. Since the morphology and distribution of our patient's EEG abnormality could suggest a deep subcortical disturbance, a possible relationship between ventricular and EEG abnormalities may be worthy of future investigation. Studies have repeatedly shown a slightly higher incidence of various types of EEG abnormality in schizophrenic populations than in normal populations (15), although the consistency of specific findings has been poor. In most studies of abnormalities in ventricular size, correlation with EEG data has received little intensive investigation, although Haug (16) has reported that patients with definite ventricular dilation showed EEG abnormalities (which were not described) almost twice as frequently as did those without ventricular dilation.

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Modern Diagnosis Schizophrenia in Hospitalized Psychiatric Patients

BY RICHARD RIES, M.D., JOHN BOKAN, M.D., AND MARC A. SCHUCKIT, M.D.

The authors applied modern criteria to the diagnosis of 254 patients admitted to the psychiatric wards of an urban hospital. They found that only 16 (6.3%) met the St. Louis criteria for schizophrenia. Other studies using similar criteria also reported a consistently low prevalence of schizophrenia in general psychiatric inpatient populations. However, 30 "undiagnosed" patients (12%) in this study narrowly missed the definite diagnosis of schizophrenia because of the presence of affective disorder and/or insufficient duration of symptoms. The authors discuss the clinical and research implications of these findings.

The psychiatric diagnosis of schizophrenia has undergone many changes since Kraepelin defined the concept of dementia praecox. As a result, there has been variation in our definition of its etiology, prevalence, characteristics, treatment, and prognosis. The most recent definition of schizophrenia, that of *DSM-III*, uses specified criteria in diagnosis and is similar in construction and content to research criteria developed by a variety of authors, notably Feighner and associates (1), Woodruff and associates (2), Spitzer and associates (3), and Taylor and Abrams (4). Studies using these criteria have been consistent in finding that 1) the prevalence of schizophrenia is significantly lower than previously thought (4) and 2) many patients previously diagnosed as schizophrenic fit criteria for affective disorders (4–8) and should probably be treated for affective disorder rather than schizophrenia.

A primary reason for the development of rigorous criteria for schizophrenia is to define a homogeneous population of schizophrenic patients and thus be better able to study and treat this disorder. There is growing evidence that modern or "narrow" diagnosis schizophrenia is a reproducible entity in that various authors using these criteria in retrospective chart studies (9) and admission studies (4, 10–12) have found a similar low prevalence of schizophrenia in general psychiatric inpatient populations (see table 1).

Although studies have found this low prevalence of schizophrenia, little has been written about the characteristics of a schizophrenic inpatient diagnosed according to the modern criteria. The current study was designed to add to the growing body of data about prevalence as well as to examine some characteristics of this more carefully defined diagnostic group.

METHOD

This study was conducted on both the open and locked psychiatric wards of an acute-treatment urban county hospital from June 1976 to July 1977. Every third admitted patient was interviewed by a trained technician using a branching structured interview containing more than 500 items that examined history, mental status, and demographic information. Interviews were then examined by a psychiatrist (M.A.S.) and given primary, secondary, and tertiary diagnosis (if indicated) according to the St. Louis criteria of Feighner and associates (1) and Woodruff and associates (2). Patients who did not fully meet these criteria were called "undiagnosed," and a "rule out" diagnosis was made. Later, questionnaire data were computerized and analyzed.

RESULTS

Prevalence

Helzer and associates (11) showed that the diagnostic reliability of a structured interview is high. In analyzing the data of 254 patients we found only 16 patients (6.3%) who met modern criteria for a diagnosis of schizophrenia (see table 2). The largest group of diagnosed patients were those with unipolar affective disorder. The undiagnosed group comprised the largest single block of patients (37.8%) (see table 2).

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TABLE 1
Studies of Prevalence of Modern Diagnosis Schizophrenia Among Psychiatric Inpatients

Study	Criteria	Method	Number of Inpatients	Prevalence (percent)
Taylor and Abrams (4)	Taylor and Abrams (4)	Structured interview	465	6.7
Taylor and associates (10)	Taylor and Abrams (4)	Structured interview	247	4.5
Helzer and associates (11)	Feighner and associates (1)	Structured interview	101	5.0
Strauss and Gift (12)	Feighner and associates (1)	Structured interview	272	3.5
	Spitzer and associates (3)	Structured interview	272	1.5
Morrison and associates (9)	Feighner and associates (1)	Chart review	3800	5.3
This study	Woodruff and associates (2), derived from Feighner and associates (1)	Structured interview	254	6.3

TABLE 2
Primary Diagnosis, Sex, and Mean Age of 254 Psychiatric Inpatients

Primary Diagnosis	Number of Patients	Percent of Patients	Number of Men	Number of Women	Age (years)	
					Mean	SD
Alcoholism	17	6.7	9	8	37	12
Drug abuse	28	11.0	17	11	26	5
Unipolar affective disorder	59	23.2	13	46	40	14
Bipolar affective disorder	12	4.7	5	7	41	14
Schizophrenia	16	6.3	8	8	33	10
Other	26	10.2	16	10	33	13
Undiagnosed	96	37.8	42	54	36	14

Characteristics

Schizophrenic inpatients tended to be younger than those with affective disorders (see table 2). The schizophrenic patients had an equal sex distribution, but unipolar affective disorder was four times more common in women. No schizophrenic patients lived with their spouse or children; 13 (81%) lived alone or with their parental family, and 13 (81%) were single and had never married. No schizophrenic patients worked full-time, and 13 (81%) were unemployed. Only 1 schizophrenic patient reported no previous hospitalization, but 11 (70%) reported six or more. A follow-up study on these patients is in progress.

DISCUSSION

Our finding of a 6.3% prevalence of modern diagnosis schizophrenia in psychiatric inpatients in an acute care urban hospital is remarkably similar to the finding of a 6% prevalence in Taylor and associates' combined studies (4, 10). Although Overall and Hollister (13) found that the criteria of Feighner and associates (1) and the Research Diagnostic Criteria (RDC) (4) did not correlate well in the diagnosis of schizophrenia for a given patient, our findings suggest that the criteria of Taylor and Abrams and the criteria of Feighner and associates may define similar populations because of the similar prevalence figures. Further research is needed to verify this.

The data presented here underscore the importance for authors to know and specify diagnostic criteria. Although the number of patients we studied is small, our data indicate that the patients diagnosed as schizophrenic by the criteria of Feighner and associates are severely impaired socially, economically, and psychiatrically. To better define the modern concept of schizophrenia and who schizophrenic patients are, a consistent diagnostic approach must be developed because the number in any one study may be small. We are aware that a variety of definitions for schizophrenia are currently being used and caution that conclusions from studies using different diagnostic criteria should not be generalized. As we have suggested, some criteria may generate populations more similar than others; however, we hope that a consistent definition (possibly that of *DSM-III*) will be used for future research in the field.

Finally, a problem with our study and other studies that use modern research diagnostic criteria is the undiagnosed patient. Almost 40% of our patients fell into this category, and we suspect that over time some would turn out to be schizophrenic and some would have other diagnoses, as Croughan and associates (14) found. In fact, 30 of our undiagnosed patients received a "rule out schizophrenia" diagnosis. This is almost twice the number who qualified for a definite diagnosis of schizophrenia. These patients were excluded from definite schizophrenia not because they lacked schizophrenic symptoms but because of the history or presence of an affective disorder and/or the lack of sufficient duration of symptoms (criteria A of Feighner and associates). This raises two points. First, in terms of research studies, not only do diagnostic criteria need to be specified but the time sequence and method of data gathering need to be considered, especially if

studies are to be compared. Second, clinicians need to remember that modern diagnostic criteria were originally developed for research purposes (1) rather than acute clinical management. Undiagnosed patients still need to be treated for their acute symptoms while further workup proceeds.

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Combined Monoamine Oxidase Inhibitor-Tricyclic Antidepressant Treatment: A Pilot Study

BY KERRIN WHITE, M.D., TOM PISTOLE, AND JEFFREY L. BOYD, PH.D.

Thirty newly hospitalized patients with RDC major or minor depressive disorder were randomly assigned to open treatment according to fixed dosage steps with 1) amitriptyline alone, up to a maximum dose of 300 mg/day; 2) tranylcypromine alone, up to a maximum dose of 40 mg/day; or 3) the combination of amitriptyline, up to 150 mg/day, and tranylcypromine, up to 20 mg/day. For 28 patients this protocol continued for 4 weeks or until discharge. As measured by the Hamilton and Zung depression scales, patients in all three treatment groups improved equally. The combination treatment produced a nonsignificantly higher frequency of minor side effects, none of which required discontinuation of treatment. The results indicate the feasibility and safety of further controlled clinical research with combined treatment, although caution is advised.

Fear of catastrophic side effects from monoamine oxidase inhibitor (MAOI) antidepressants has discouraged clinicians from conducting controlled research with these drugs and left practitioners dependent on a folklore of uncontrolled clinical reports for guidelines to usage. The recent resurgence of interest in MAOIs has accentuated the need for more data on a number of issues, one of which is the safety and efficacy of combining MAOIs with tricyclic antidepressants.

To our knowledge virtually all authors (1–7) reviewing this area have agreed that the combination treatment, used in a specific manner, appears relatively safe. Moreover, several reports (4, 8–14) have suggested that this treatment benefits a substantial number of patients refractory to alternative treatments and results in an overall improvement rate of about 90%. Theoretical speculations also suggest that combined MAOI-tricyclic treatment might yield improvement more often, more rapidly, more completely, or across a broader spectrum of depressive subtypes than single antidepressant treatment. However, these speculations lack support from controlled clinical studies.

Davidson and associates (8) randomly assigned 19 medication-resistant, depressed inpatients to treatment with either electroconvulsive therapy or combined treatment with amitriptyline (up to 100 mg/day) and phenelzine (up to 45 mg/day) for 3–5 weeks. Despite the small sample size, they found ECT superior to the drugs; although the drugs caused no serious toxicity, they caused enough side effects to lead to dosage restriction in four cases. Actual dosage averaged 71 mg/day of amitriptyline and 34 mg/day of phenelzine, which many would consider inadequate.

In a larger study of 135 depressed outpatients not specifically selected as refractory to treatment, Young and associates (15) randomly assigned subjects on a double-blind basis to one of five 6-week treatments: 1) phenelzine only (average dose, 45 mg/day); 2) isocarboxazid only (32 mg/day); 3) trimipramine only (106 mg/day); 4) phenelzine plus trimipramine (44 and 102 mg/day, respectively); or 5) isocarboxazid plus trimipramine (30 and 96 mg/day). On several measures, trimipramine only showed superior efficacy, particularly when compared to the combined treatments. Side effects remained modest for all groups; combined treatments and MAOIs only caused more insomnia than trimipramine only, which caused more tremor. This study cast further doubt on an advantage for combined treatment while lending support for its relative safety. Methodologic problems limiting the conclusiveness of these findings included adjunctive use of both diazepam and nitrazepam, possibly inadequate dosage of phenelzine, use of an MAOI (isocarboxazid) of doubtful efficacy, and the limited monitoring of adverse effects.

Although more work in this area is needed, persistent concern over risks may deter such investigation, particularly in the United States, where the Food and Drug Administration and pharmaceutical companies have continued to warn against use of the combination.

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We designed a pilot study to evaluate relative safety and open the way for larger scale, better controlled research. We used tranylcypromine as the MAOI, partly because of its reputation as the most efficacious MAOI, even though its reputation for adverse effects had led others to prefer alternative MAOIs for combination treatment. We used a relatively unselected group of depressive patients, who were not necessarily refractory, to enhance access to subjects and to test the idea of broad spectrum efficacy for combined treatment on various subtypes of depression. Despite the limitations of our study, i.e., its small size and open administration of medications, it is one of the first reported comparisons of combined versus single antidepressant treatment to use such basic controls as prospective design, random assignment to treatment, and specified diagnostic and outcome criteria.

METHOD

Subjects were newly admitted adult inpatients at the Los Angeles County-University of Southern California Psychiatric Hospital. All subjects met Research Diagnostic Criteria (16) for major or minor depressive disorder, and all signed informed consent to participate after the research protocol had been explained.

Subjects were randomly assigned to start open treatment with either amitriptyline, 75 mg h.s., tranylcypromine, 10 mg b.i.d., or amitriptyline, 50 mg h.s., and tranylcypromine, 10 mg every morning. At the clinician's discretion, based on the patient's progress, these dosages could be increased at the end of the first week to amitriptyline, 150 mg h.s., tranylcypromine, 10 mg t.i.d., or amitriptyline, 75 mg h.s., and tranylcypromine, 15 mg/day; at the end of the second week, dosages could be further increased to amitriptyline, 300 mg h.s., tranylcypromine, 20 mg b.i.d., or amitriptyline, 150 mg h.s., and tranylcypromine, 10 mg b.i.d. Treatment on this protocol continued until the patient improved enough to be discharged, or until 4 weeks had elapsed.

The treating psychiatrist (K.W.) evaluated the patients with the Hamilton Depression Scale, the Brief Psychiatric Rating Scale, and the Clinical Global Impression just before the patients started treatment and at the end of each successive week of treatment. At the same intervals, subjects completed the Zung Self-Rating Depression Scale and the Side Effects Checklist, a checklist devised by us that consists of 40 symptoms drawn from the *Physician's Desk Reference* descriptions for each drug and rated on 4-point scales of severity. Routine laboratory tests, including complete blood count, serum electrolytes, serum liver function tests, urinalyses, and ECGs, took place on a weekly basis. Measurement of patients' vital signs, including standing and lying blood pressure and pulse rate, respiratory rate, and oral temperature, was done daily.

RESULTS

Of the 30 patients who took part in this study, 2 were dropped from further analysis—1 insisted on leaving after only 2 days on amitriptyline, and 1 (on tranylcypromine for 1 week) should have been excluded from the study in the initial evaluation because further diagnostic interviews revealed definite nonaffective hallucinations and delusions. Of the remaining 28 patients, there were 12 women and 16 men, aged 19–61 years (median, 33 years). Clinical diagnoses included neurotic depression (N=12); manic-depressive illness, unipolar type, depressed (N=10); personality disorder, hysterical type, with depression (N=2); manic-depressive illness, bipolar type, depressed (N=1); personality disorder, sociopathic type, with depression (N=1); adjustment reaction of adult life with depression (N=1); and chronic nonpsychotic organic brain syndrome (with seizure disorder) with depression (N=1). After random assignment, 8 received amitriptyline alone, 10 tranylcypromine alone, and 10 combined amitriptyline-tranylcypromine.

Duration of treatment on the study, dependent on time until discharge became appropriate, varied from 2 to 4 weeks and averaged 3.5 weeks. Maximum dosage in the three treatment groups averaged 165 mg/day for amitriptyline only, 30 mg/day for tranylcypromine only, and 80 mg/day of amitriptyline and 13 mg/day of tranylcypromine in the combined group.

Figures 1 and 2 show the therapeutic outcome in each treatment group as measured by the change in total Hamilton and Zung scores from baseline to the end of the first week and to the end of treatment.

Analyses of covariance were performed to test for differences among treatments at the endpoint; baseline scores were treated as covariates. No statistically significant differences were found among the three treatments on either the Hamilton or Zung change scores. Overall, 82% of patients showed substantial improvement (reduction of Hamilton score by at least 15 points): 86% (7 of 8) on amitriptyline only, 80% (8 of 10) on tranylcypromine only, and 70% (7 of 10) on amitriptyline-tranylcypromine.

The incidence of the various side effects on each of the three treatments was indicated by the emergence during successive treatment weeks of patients' complaints of either not present or present to a less severe degree at pretreatment. Almost all patients indicated some side effects, most commonly dizziness (N=14), weight gain (N=13), dry mouth (N=10), and anxiety (N=10). The combined treatment produced only a slightly higher frequency of side effects (8.8 per patient) than did the single antidepressant treatments (7.8 per patient for amitriptyline alone; 7.2 per patient for tranylcypromine alone). The side effects that appeared to occur more often on the combined treatment than on the single drugs included tinnitus (N=5), fatigue (N=5), weakness (N=4), and urinary hesitance

FIGURE 1
Hamilton Depression Scores Over the Course of Treatment for Each Medication Group

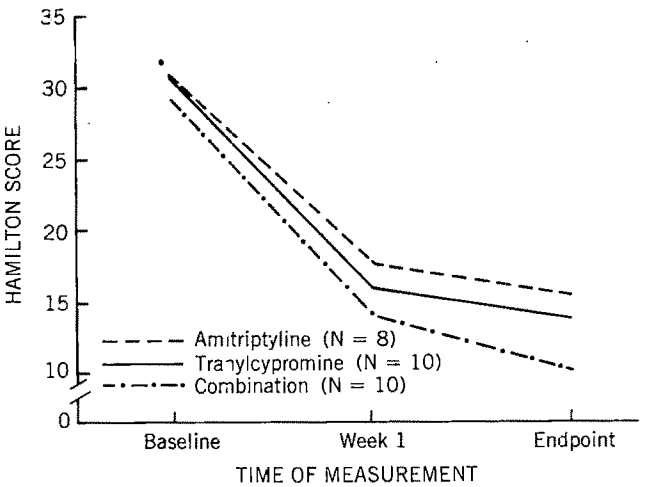
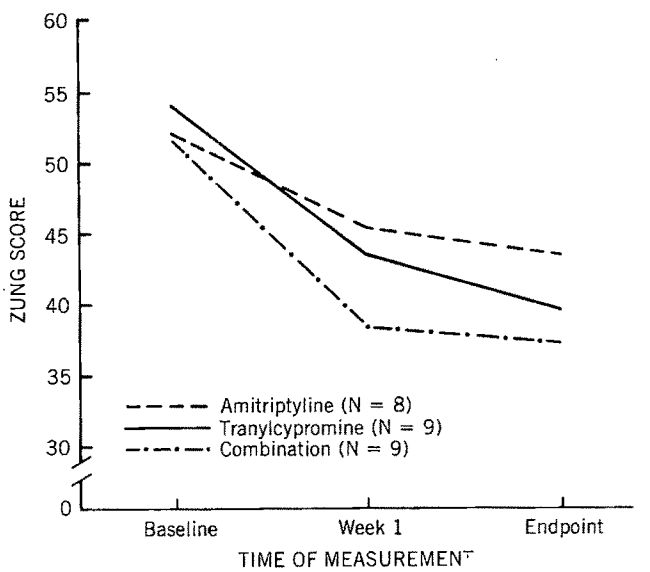


FIGURE 2
Zung Self-Rating Depression Scores Over the Course of Treatment for Each Medication Group



(N=2). Both the combined treatment and tranylcypromine alone appeared to be associated with a higher incidence than amitriptyline of headache, paresthesias, urinary frequency, muscle spasms, and insomnia.

A side effects score weighted for severity was calculated by adding the 4-point severity scores of items on the side effects checklist for each week the patient was taking medication, subtracting the pretreatment severity scores, and dividing the remainder by the number of weeks on study medications. The side effects severity rating for each patient, which took into consideration varying durations of treatment and allowed for between-treatment comparisons, yielded an index of 3.4 for the amitryptyline group, 4.2 for the tranylcypromine group, and 5.6 for the combined group. These ratings suggest that the combined treatment resulted in slightly more severe side effects than did the single drugs; however, in no case did side effects appear clinically severe enough to require discontinuation of treatment. Nor did any analysis of side effects by t tests show a statistically significant difference between the treatment groups.

Throughout the study, close monitoring of patients' physical status, including regular vital signs, uncovered no occurrence of hypertensive or hyperthermic crisis in any patient. The typical patient showed a lower blood pressure while taking medication than before treatment. Blood pressure measured in a hypertensive range (diastolic>95) in 1 patient who was taking amitriptyline only and reached 160/130 in this patient, who was slightly hypertensive (139/94) before treatment. Patients' temperatures never rose to febrile levels; the highest recorded temperature reached 100°F in 1 patient taking tranylcypromine. Routine laboratory tests and ECGs revealed no new abnormalities attributable to treatment.

DISCUSSION

The major implication of this preliminary study is that combined amitriptyline-tranylcypromine treatment, when used according to a schedule of graduated concurrent dosage increase, can be safely applied to a relatively unselected group of depressive patients. We found that the combined treatment was at least equally effective as, and at most produced slightly more minor side effects than, the single drugs used at higher dosages.

This conclusion requires caution in clinical application. Our study contains methodologic problems that should be corrected in later research: 1) possible bias in evaluation because of open rather than blind treatment; 2) the lack of data on the validity and reliability of the side effects checklist used; and 3) the small sample size. Furthermore, the clinician considering use of combination treatment should be aware that our findings apply only to the specific drugs and dosage regimen we used. The indication of relative safety excludes, for example, combinations involving the more noradrenergic tricyclics such as imipramine, often implicated in adverse reactions, and also excludes any schedule other than our approach of simultaneously beginning both drugs at a low dosage followed by graduated concurrent increase. Also, the combination treatment may pose special risks in situations (e.g., overdoses) that our study did not assess.

Moreover, the clinician should realize that the three controlled studies reported to date have failed to provide any evidence that combination MAOI-tricyclic treatment is superior to single drug treatment. The small size of this study may have prevented significant differences in efficacy between the treatments from

emerging but gave no suggestion that the combination treatment worked more often, more completely, and more rapidly than the single drugs. Since the drugs were used in higher individual dosages when given alone, the factor of dosage may have enhanced response to the single drug treatments.

As interest in and use of MAOIs revives, it is important to substitute for the folklore of the past a basis in more scientific evidence regarding both safety and efficacy. The area of combination MAOI-tricyclic antidepressant treatment requires more controlled clinical studies to determine the true indications for this approach.

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The Primal Scene: A Study of Prevalence

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Although primal scene experiences have been implicated as causal agents in innumerable psychopathologic states, no study of their actual prevalence exists. The authors conducted five separate studies, in which parents reported that 9%–41% of their children had seen parental intercourse. If the times children see parental intercourse without the parents knowing it and the times children hear parents making noises during intercourse were included, the prevalence would be much higher. In view of the frequent occurrence of primal scene experiences, factors other than the viewing itself must influence psychopathological outcomes when they occur.

Estimates of the prevalence of children viewing parental intercourse are confusing, in part because the term "primal scene" is used in diverse ways. For some authors "primal scene" refers to the actual viewing of parental coitus. To others the term includes hearing parental intercourse. Those authors who use "primal scene" most broadly even consider intrapsychic fantasies about parental intimacy as "primal scene experiences" whether or not actual intercourse has been observed or heard (1). Actual viewing of parental intercourse is sometimes said to be uniformly detrimental to a child's future sexual adjustment. However, no empirical data about prevalence exist since no study has ever explored how common these experiences are.

In contrast to the idea that great damage is invariably caused, anthropologists familiar with psychoanalytic theory have commented that in many "primitive" societies seeing parental intercourse does not appear to have a uniformly deleterious effect on later sexuality. Mead (2), for instance, found the Samoans to be "easy, casual, and without shame about sexuality," even though as children they frequently viewed parental intercourse. Similarly, Devereaux (3) noted that Mojave children were not "*too* damaged" by primal scene, ascribing this in part to the adults' freedom from guilt. Of course, none of these anthropological studies could do detailed examinations of intrapsychic life.

Esman (4) reviewed the psychoanalytic literature on primal scene. He found that in those cases in which a child interpreted the primal scene sadomasochistically, there was frequently evidence that the father had in fact beat the child or mother physically. He further commented,

> The primal scene has been indicted as the primary pathogenic agent in every form of psychopathology: mania, depression, paranoia, hebephrenia, hysteria, compulsive neurosis, character disorder, learning disturbance, asthma, headache, delinquency—all have been explained as single or multiple exposures to the primal scene. One is moved to wonder whether we are here confronted with one of those situations in which a theory, by explaining everything, succeeds in explaining nothing.

Some modern practitioners have begun to question Freud's original hypothesis, not only regarding the effects of primal scene viewing, but also what causes psychological damage if the experience is in fact traumatic and is interpreted sadomasochistically. Esman (4) stated that the sadistic conceptualization is determined not by the child's viewing of the event itself but by the amount of actual overt, violent aggression the parents demonstrate in general family life. Furthermore, the child's response to the primal scene will be

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determined in large measure by the parents' reactions, the cultural setting, and the ego resources of the child. Thus, it is not the event but the adaptive, coping mechanisms and external resources available that determine outcome.

Given the dearth of data, we decided to ascertain the prevalence of primal scene in several populations. Such an attempt is fraught with difficulties. To study children directly a researcher would have to rely on each child's almost blind trust, honesty, and ability to have access to memories that may have been repressed or denied. Furthermore, questioning children in this way raises ethical concerns.

Parents are easier to study. Since they are being asked about events that occurred during adulthood, they are less likely to repress them. In fact, anecdotal reports indicated that if children walked in while the parents were having intercourse, the parents often remembered the occasion with great clarity. Therefore, we decided to explore the prevalence of primal scene through parental reports. Since parents can be expected to be aware of only some of the times their children have observed intercourse and may be reluctant to report these honestly, the prevalence ascertained by this method must be regarded as a minimum one. To avoid confusion, we used "primal scene" to include only those experiences in which the child was physically present and awake during parental intercourse. Since we did not interview the children we cannot say exactly what they saw.

METHOD

We conducted five separate studies, three at the Naval Regional Medical Center in Portsmouth, Va., and two in Palo Alto, Calif.

The first three groups surveyed were 1) Navy enlisted men bringing their children for normal school physicals, 2) Navy physicians, and 3) parents of patients in a Navy child psychiatry clinic. All were asked to complete a questionnaire concerning affectional and sexual life in their families. It included the following questions about primal scene: "Has your child ever awakened at night and by chance come into your room while you were having sexual intercourse or preparing to have sexual intercourse?" and "Or on a special occasion has he ever awakened while sleeping in your room and happened to see sexual intercourse? Partially or fully?" From the answers (these parents usually wrote further comments), it was clear that almost all children who had experienced primal scene had walked in on actual intercourse, not on the preparation for it. Of the clinic population, 100% filled these forms in as a routine part of a child psychiatry evaluation. Of the enlisted men who were asked to complete the forms anonymously, 25% did so; of the physicians, 38%.

The low return rates in these first three studies of normative populations were overcome in the fourth and fifth studies. They were mail surveys that employed methods described in a prior communication (5) and achieved an 85% return rate. Almost all of the parents in these studies were highly educated, upper-middle-class, and white. All had children between 4 and 10 years of age and filled out a redesigned questionnaire on family affectional and sexual life. They could identify themselves or remain anonymous. The questionnaire asked, "Has your child ever by chance come into your room while you were having sexual intercourse?" and asked parents to check one of five blanks on a scale of "never" to "regularly." The next question was "If yes, how old was your child and what was your child's reaction?"

RESULTS

Among the 81 enlisted men who completed the questionnaire, 41% said their children had viewed parental intercourse, while 59% said they had not. Of the 26 Navy physicians who answered this question, 2, or 11%, said their children had viewed parental intercourse, while 16, or 89%, said they had not. The groups in these two studies differed primarily in educational level and, consequently, in socioeconomic status. Chi-square analysis revealed a significant difference between the groups ($\chi^2=5.51$, $p<.05$). Of the 38 parents of patients in the Navy child psychiatry clinic who completed the questionnaire, 3 (9%) said their children had viewed parental intercourse. Given the threatening nature of the situation, parents bringing their children to a psychiatric clinic were unlikely to be forthcoming in providing information. Therefore, the reliability or significance of the low incidence in the clinic population is difficult to assess.

In the fourth study, the question was stated more specifically; "preparing to have intercourse" was dropped. Fifty-seven, or 19%, of the children were said to have seen their parents engaged in sexual intercourse, while 240, or 81%, had not. The percentage of children said to have viewed sexual intercourse remained fairly constant for each age group. There were no significant differences in responses about male and female children, or between physicians (21%) and nonphysicians (19%). The fifth study replicated the findings of the fourth (20%).

Forty-nine of the 57 parents in study 4 who reported that their children had seen parental intercourse responded to the question concerning the child's reaction. Almost all said their children observed intercourse between the ages of 4 and 6. Most parents ascribed no particular significance to the event, stating that the child had little reaction. Of these parents, most reported that their child did not know or comprehend what was happening, that the child had little re-

action. Of these parents, most reported that their child did not know or comprehend what was happening.

> Since the bedclothes were over us, I don't believe he had any idea that we were having intercourse. Asked his question and ran out again to do his thing.
>
> I don't think she had any idea what was going on. She just stood there quietly until I realized she was there.
>
> She didn't know what was happening under the covers.

Other parents said their children noticed but had "no reaction," e.g., "child has no particular reaction" or "takes it as an ordinary occurrence," "just something adults do—neutral reaction," "he seemed uninterested."

A few parents reported that, at about age 5, their children were curious about what the parents were doing, e.g., "She asked why we were bobbing up and down." By age 6, more children were reported as reacting inquisitively: "He asked a few questions, e.g., 'Why are your clothes on the floor?' 'Why is the closet light on?' We answered his questions, and he went back to bed."

Other 6-year-olds responded with amusement or giggling. Several parents of 6-year-olds reported that their children had indicated they had some idea of the event's implication. For instance, the parent of one child who saw intercourse twice noted, "At 4, she did not understand and went out again. At 6, she calmly said we would have another baby and went back to watching TV." Another reported, "The next day at dinner she said smugly, 'I know why you want me to stay in my bed. You "hump" at night.' "

Some of the few children who saw their parents have intercourse after the age of 6 understood the events, and some were clearly embarrassed and uncomfortable at walking in on their parents: "[She] said 'Oh, no!' giggled, and left the room, closing the door." "She wanted me to know she saw—was afraid I would be mad. I was not."

DISCUSSION

These five studies determined the lowest possible prevalence of children viewing parental intercourse. Since the majority of parents in the last two studies reported that their children had not viewed primal scene, and those who said their children had usually stated it happened only once, it is probable that these parents act in ways which prevent their children from walking in on their lovemaking. In this socioeconomically homogeneous group, physicians and nonphysicians did not differ in their reports of prevalence. Thus, it is likely that if the differences between studies 1 and 2 are replicated, they are related to socioeconomic or educational status.

If the definition of "primal scene" were expanded to include those times that children see their parents have intercourse that are not known or reported by the parents and to include the number of times children hear their parents making noises during sexual intercourse one would undoubtedly come up with figures much higher than those in these studies.

Thus, even the most conservative estimate of the prevalence of primal scene viewing indicates that it is not rare, which leads us to believe that factors other than the viewing itself must influence psychopathological outcomes when they occur.

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An Indochinese Refugee Psychiatric Clinic: Culturally Accepted Treatment Approaches

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In 1978 the authors established a weekly psychiatric clinic for Indochinese refugees. During the first 20 months, 50 patients were evaluated and treated at the clinic; a Vietnamese psychiatric resident and several native Indochinese mental health counselors bridged the language and cultural barriers between patients and clinic personnel. Most of the patients seen at the beginning of the program were psychotic and severely impaired. However, patients seen later suffered from a wider variety of problems. A flexible approach to treatment was adopted that would be compatible with the cultural expectations of the refugees. This resulted in the use of different forms of therapy and special emphasis on the medical approach of the physician, a role familiar to Indochinese patients. Gradually the clinic gained acceptance by members of the local refugee community.

Since the collapse of the Saigon government in April 1975, more than 300,000 Indochinese refugees—a diverse group of Vietnamese, Cambodians, and Laotians—have come to the United States. Forced to leave their homelands because of fear of imprisonment and persecution, they suffered the consequences of an abrupt loss of jobs and possessions, and they needed to make an immediate adjustment to a new language and culture. The refugees had already endured the debilitating effects of decades of war before coming to this country. The dangerous escape from their homeland was followed by a transitional, often stressful, period in large refugee camps and finally by the settling-in process in a strange country.

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In contrast to the nearly one million Cubans who have escaped to the United States since January 1959 (1), the Indochinese refugee has faced more difficult problems. In addition to making a longer journey to a country with a very different culture, the Indochinese refugees had no similar minority groups already here to offer them support. Also, they have often been identified symbolically with the ambivalently viewed Viet Nam war.

Some refugees left Viet Nam for the United States after the fall of the Saigon government; others lived under the communists for several years, carefully plotting their successful escapes to the United States. All described the loss of relatives and friends through separation and death. All were affected by their change in social status and by the necessity of acquiring a new language and new employment. Cultural adjustments were very difficult because of the vast differences between the Indochinese and American value systems, especially pertaining to family relationships, time concepts, expression of affect, and the value of education (2).

Two careful studies among refugees in Norway and Australia concluded that there is a significant increase in psychiatric disturbances, particularly in psychoses, in refugee groups (3, 4). Long-term studies on the psychiatric problems among the Indochinese refugees are not yet complete, although there have been several early observations of adults (5, 6) and children (7) in the Vietnamese refugee camps. A recent report of 50 Vietnamese refugee families isolated in Louisiana indicated that the refugees were adjusting very well despite specific sociological stresses (8). However, a thorough 2-year community study of another group of Vietnamese refugees, using the Cornell Medical Index, found a high and continuing level of physical and mental dysfunction (9).

THE UNIVERSITY INDOCHINESE PSYCHIATRIC CLINIC

In 1977 members of the Department of Psychiatry at the University of Oregon Health Sciences Center became involved in a series of program and case consultations with the Indochinese Health Care Project organized in Portland, Ore. The project trained native

Vietnamese, Cambodian, and Laotian counselors to serve the mental health needs of Indochinese refugees. With the help of the project's staff in 1978 our Department of Psychiatry recruited a Vietnamese physician (K.A.T.) as a psychiatric resident. Later that year we established a weekly psychiatric clinic for Indochinese refugees, using the Vietnamese physician and the Indochinese mental health counselors as collaborators to bridge linguistic and cultural differences.

When we started our clinic, we were responding to the troublesome community experience of a few refugees who had psychiatric problems. Apart from these obviously disturbed people, we did not know the extent of psychiatric problems among the refugees. Our original impression was similar to that of Vignes and Hall (8)—most refugees had made good adjustments to their new country. In Oregon 90% of the refugees had learned English and 90% of their families had members who were employed.

From its inception the clinic accepted for evaluation any patient who was referred, most patients were seen within a week. The clinic received referrals from the Indochinese Health Care Project, county health clinics, other university clinics, schools, social agencies, and private practitioners.

The techniques employed have been described in reports of other treatment settings for Asian patients (10, 11). These techniques met the expectations of the patient in a culturally acceptable manner because we used a medical approach, which emphasized taking a thorough history and concentrated on the development of the patient's symptoms and problems. Psychological interpretations by the physician (psychiatrist) were infrequent, but the relationships between losses or social events and symptoms were explored. The first goal of treatment was the reduction of the patient's symptoms. Patient visits were frequent and supportive; medications were used with the clear expectation that they would be helpful. Our experience indicated that this medically oriented technique was congruent with the Indochinese patient's expectations about the behavior of physicians.

RESULTS

During the first 20 months of the clinic, 50 patients were evaluated or treated (table 1). The ethnic background of the patients, which was predominantly Vietnamese, was proportional to the refugee population in Oregon. There were 27 men and 23 women in the patient group; the age range was 15 to 59 years, with most patients in their 20s and 30s. The patients varied greatly in background—from those with prior professional training to others who had no formal education. Some had been in this country for 4 years and had established occupations. Others had arrived as recently as 2 weeks prior to being evaluated. The earliest patients referred were very impaired and primarily psychotic; almost all were schizophrenic. These patients caused severe problems for service agencies. The patients who were referred later represented broader problems, including depression, anxiety, mental retardation, adjustment reactions, and psychosomatic disorders. In the total group, 18 patients were psychotic (17 were schizophrenic and 1 was manic), 21 were depressed (4 with severe impairment), and 6 suffered from anxiety disorders. At the time the patients were first seen, 31 were unable to carry on the daily tasks of living, which included working, studying, or maintaining their households. No alcohol or drug-related problems were discovered, confirming our information that currently these problems are rare among the Indochinese refugees.

The high number of depressed patients deserves special comment. Depression seemed to be overdetermined in these patients because they had been sensitized to multiple losses and had undergone complicated economic and social setbacks. At the time they were seen in our clinic they were often very distressed, and they told their histories with great emotion. We are in the process of developing a depression rating scale in the Vietnamese language to help evaluate the prevalence of depression among the Vietnamese refugees (12).

Most patients received psychotropic medicine for psychotic disorders or depressive symptoms. These patients often had frequent, brief follow-up visits. Individual psychotherapy for support, ventilation, and further clarification of the problems was used with some patients. In a few situations spouses or other family members were brought in for brief family or marital therapy. However, this was difficult because cultural values inhibited direct expressions of problems in the presence of other family members. Education about mental illness and support of the family often proved extremely helpful.

Adjustment problems of refugees to the new environment were best handled by the Indochinese counselors. We consulted with Indochinese mental health workers to facilitate this work. At times we also functioned as advocates for patients served by multiple agencies (legal, welfare, and vocational rehabilitation).

A subjective but very important aspect of the clinic was to give refugees a safe, free environment in which to tell their stories. All had made significant decisions to leave their country, and all had doubts and conflicts about their new home. Most endured very difficult hardships traveling here; many witnessed brutality, the horrors of civil war, and piracy. Uncharacteristically, they often described their situation with a great deal of emotion. They later reported being much relieved and described how they had never been able to discuss the situation before.

From follow-up data (4 to 26 months) on all 50 patients, it was clear that there were two separate groups

TABLE 1
Characteristics of 50 Indochinese Refugee Patients Evaluated by a Psychiatric Clinic

	Patient Characteristics						
	Sex		Diagnosis				
Ethnic Group[a]	Male	Female	Psychosis	Depression	Anxiety	Other	Severely Impaired
Vietnamese (N=38)	22	16	12	17	4	5	23
Cambodian (N=6)	2	4	4	1	1	0	6
Laotian (N=6)	3	3	2	3	1	0	2

[a]The ethnic background of the patients is proportioned to the entire refugee population in Oregon. At the end of 1979 there were 4,200 Vietnamese refugees, 600 Cambodian refugees, and 1,200 Laotian refugees in the state.

of patients: those with a psychotic diagnosis who tended to have long-term contact with the clinic and those with a nonpsychotic diagnosis and short-term involvement with the clinic.

Of the 18 psychotic patients, all visited the clinic more than once, and most had extensive treatment in the hospital (N=11) or clinic. Nine of these patients continued in treatment, each with greatly reduced symptoms; 3 patients terminated treatment when they moved or developed asymptomatic behavior; and 6 patients dropped out of treatment, but if their symptoms recur, it seems likely—as has happened with others—that they will return. Accepting these severely impaired patients for treatment was important in establishing approval of the clinic by the refugee community at large.

In contrast to the psychotic patients were the patients with a nonpsychotic diagnosis (N=32); 25 (78%) of them kept their first return appointment. Of those who returned, the average number of follow-up visits was 5.5 Both these figures are slightly higher than for all nonrefugee patients who came to the University clinic. Although the nonpsychotic patients dropped out before we recommended they should, the last contact made by our clinic indicated that 21 of the 25 patients had good to marked symptomatic improvement. Clearly they responded positively to the reduction of their symptoms and regarded continuing medical treatment or psychotherapy as unnecessary.

We describe below two typical patients seen in the clinic. One represents a psychiatric disorder that developed from his changing cultural values and family relationships. The second patient illustrates the cultural and language problems encountered in making a diagnosis.

CASE REPORTS

Case 1. Mr. A was a 40-year-old married Vietnamese man who had been a school teacher in Viet Nam. He was referred by a vocational counselor for evaluation of his inability to find work despite his good qualifications. Mr. A described symptoms of sadness, insomnia, decreased appetite, loss of interest in most activities, and periodic agitation directed toward his family. When we saw him, his symptoms had been present for 1 year, but they had become worse in the last 6 months. A long-standing dermatological lesion was also exacerbated during this time. He originally had received a grant for a trade course, which he completed, but 1 year later he still was unable to find work. His wife had begun her first job 6 months previously, and she was now the only wage earner. Mr. A was suffering from moderately severe depression, which was aggravated by his loss of status; status is very important in the Vietnamese culture, particularly as family relationships are changing. He was started on antidepressant medicine, and as his symptoms improved, vocational counseling was added. Brief therapy helped him understand his status change.

Case 2. Mr. B was a 29-year-old single Vietnamese man, formerly an Army officer. He was referred because of his inability to learn English, despite opportunities in various vocational classes, and because of periodic aggressive behavior. Difficulty in communication prevented us from immediately understanding his problem. He described a bomb blast that occurred near him several years previously as having destroyed his ability to concentrate. Mr. B was unkempt and somewhat unpredictable in his behavior. Further questioning in the Vietnamese language revealed that he felt forces could control his mind and that a mysterious force from electricity encompassed his body. Findings from a neurological examination and an EEG were normal. A diagnosis of schizophrenia was made and Mr. B was begun on neuroleptics. There was marked reduction in his symptoms, and he was able to return to his regular classes.

DISCUSSION

Despite great mental distress, it is not easy for Indochinese refugees to accept a psychiatric referral. The Vietnamese have the traditional Asian attitude toward the mentally ill, which includes fear, rejection, and ridicule (13), and the presence of a mentally ill member in the family affects the family's social and economic status. Another cultural problem is the lack of a traditional counselor in Asian societies—one who listens to and advises individuals about how to cope with personal feelings and problems. Within these cultures the emotional aspect of one's life is private and shared mainly with one's family. Although traditional healers may prescribe remedies or offer advice, none attempt to question or relate to the subjective experience of the patient in the manner of Western psychotherapy. Thus, some forms of psychiatric treatment

(questions about feelings and sensitive relationships) may seem threatening to the Indochinese refugee patient. To overcome these problems it is necessary to provide services in a culturally and personally acceptable way.

The technique we used emphasized the medical approach of the physician, a role familiar to Indochinese patients. This approach proved acceptable to them, including those refugees first referred—who were severely disturbed psychotic patients. We emphasized taking the patient's history and reducing his or her symptoms, which provided a relationship with some patients that allowed for more discussion of psychological issues. Of particular importance was the newly learned ability of the refugees to open up regarding their losses and depressed feelings.

With 20 months of experience, we see that our Indochinese clinic has gone through several stages. Our first patients were severely disturbed and psychotic. Later we saw patients who were primarily impaired by depression. Now we evaluate many types of patients, whose range of problems is similar to that seen in the usual university psychiatric clinic. We now see a wider range of problems, we believe, because the clinic is currently accepted in the Indochinese community.

We found that for an Indochinese clinic to function properly, it was necessary to have mental health workers present from each ethnic group. They could interpret language and cultural norms and were available for certain case management roles. We were very fortunate to have a Vietnamese physician as a psychiatry resident who could also collaborate with the local refugee mental health training program. It became evident that our demonstrated ability and willingness to handle acutely psychotic individuals and to provide hospitalization for them further encouraged the support of the Indochinese community.

Finally, to be successful in treating the refugee patient, we found that the psychiatrist must be able to work flexibly and broadly with a wide variety of cultural, social, and psychological human problems. The clinician needs to respect the cultural background of the patient, to recognize the patient's concurrent adjustment problems, and to blend the best techniques and medical approaches of American psychiatry in order to be of service to the Indochinese refugee.

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Stress Response Syndrome Occurring After Delirium

BY THOMAS B. MACKENZIE, M.D., AND MICHAEL K. POPKIN, M.D.

Psychiatric treatment of delirium usually focuses on behavioral management during the period of cognitive disturbance. Psychological sequelae have received less attention. The authors suggest that delirium represents a stressful life event that must be assimilated after the cognitive impairment has resolved. Such assimilation may be uniquely impaired because the patient lacks clear and correct information about the event. The authors propose that the psychiatric care of delirium include facilitation of the patient's efforts to obtain and assimilate information about the delirium after its resolution.

Delirium is one of the most common reasons for psychiatric consultation. Among forms of psychopathology, it is the most readily conceptualized in terms of the medical model. Treatment focuses on determining and then reversing the physiological abnormality and on management of behavioral disturbances. The latter is accomplished by providing optimal sensory stimuli, a protective environment, cues regarding orientation, and simple explanations in combination with judicious use of psychotropic drugs. This organic focus has tended to obscure the psychological dimensions of optimal care. The following case drew our attention to an additional element in the psychiatric management of a delirious patient.

CASE REPORT

Ms. A, a 50-year-old woman, had a 35-year history of rheumatic heart disease. She was hospitalized for mitral valve replacement necessitated by worsening cardiac failure of 2 years' duration. Although Ms. A was hesitant about surgery, her son and her physician convinced her to proceed by emphasizing the improvement she could expect after the operation.

Ms. A had characteristically dealt with her illness by minimizing it and remaining active. She described herself as a private person who was cautious about relationships with others. Her husband, with whom she had raised one son, was a taciturn man whose job frequently caused him to be away from home. Ms. A had no personal or family history of psychiatric or neurological disease.

The hospital staff had considerable difficulty maintaining Ms. A's blood pressure after surgery. She required constant infusion of pressors and placement of an intra-aortic balloon. Her persistent hypotension resulted in respiratory and renal failure, which further complicated her postoperative course. She required respiratory support for 60 days, and for the first 30 days she underwent dialysis three times a week. She received multiple psychoactive medications including morphine, codeine, diazepam, flurazepam hydrochloride, hydroxyzine pamoate, diphenhydramine hydrochloride, and prochlorperazine.

Ms. A's physician sought psychiatric consultation 4 weeks after surgery to justify transferring Ms. A from the intensive care unit (ICU) to a private room despite her reliance on a respirator. This was accomplished after the consultant agreed that the noise and activity in the ICU were contributing to Ms. A's sleep deprivation, thus complicating her recovery.

When we first examined Ms. A, she was a frail, lethargic woman lying supine with a tracheostomy tube connected to a respirator. She exchanged eye contact, mouthed words, and wrote short phrases with great effort. Nurses reported that she had seemed intermittently confused and had failed to recognize her husband the previous day. We reported that Ms. A was delirious.

We arranged brief daily visits to provide support and to monitor fluctuations in Ms. A's mental state. Her affect showed abrupt transitions from smiling to tears. She demonstrated a moderate delay in responding to questions and many of her written notes revealed disorientation and perplexity. She was intermittently agitated, especially near the end of periods without respiratory assistance. She acknowledged being frightened when taken off the respirator; we noted that she was particularly apprehensive when cared for by nurses with whom she was not acquainted.

Medical management led to gradual resolution of her cardiac, respiratory, and renal failure. She was extubated 8 weeks after surgery. At that time bedside examination of her sensorium was unremarkable. Nursing staff began to notice that Ms. A was asking questions in a casual manner about her hospital course such as, "Did I know you before?" and "How long have I been here?"

One of us (T.B.M.) conducted three 1-hour interviews during the remainder of Ms. A's hospitalization. During these sessions Ms. A increasingly expressed concern about her recollections of the postoperative period. She revealed that she had initially intended to keep her concerns secret because she was afraid she would be considered a "terrible person." However, she believed that staff and family had

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become aware of her concerns through the notes she vaguely recalled writing while using the respirator. Therefore, she agreed to discuss her recollections and concerns. Ms. A reported that her first memory after surgery was of being deprived of water. She remembered thinking that the nurses were cruel and asked T.B.M., "How could they have done something like that?" She recounted that some sort of device resembling walrus tusks had been grown in her throat by the hospital staff. Ms. A's foremost concern was that she had written a note accusing one of her physicians of raping her. Although she did not believe this actually had happened, she was troubled that she could have imagined such a thing. According to Ms. A, neither she nor anyone close to her had ever been a victim of sexual assault.

We did not see Ms. A again until she was readmitted for control of congestive heart failure 10 months later. She requested that T.B.M. be reconsulted, and four 1-hour sessions took place over an interval of 2 weeks. For 2 months after her previous discharge, Ms. A said she had been afraid to fall asleep because of a recurrent nightmare. She dreamed that she was attempting to escape from the hospital but was overtaken and returned by physicians whom she could not identify. She revealed that there had been a time during her initial hospitalization when she had trusted no one. She acknowledged that this had included T.B.M. and admitted she was ashamed of having been so distrustful. When asked to give an example of her distrust, Ms. A recalled fearing that her husband would passively consent to any request from her physicians without asking them to justify their plan. Ms. A worried over how much had "escaped her mind"; she conceded that she had been skeptical about her own physician's explanations that this amnesia had resulted from medications. When asked for her opinion, she answered that she "just didn't know."

Psychotherapy was supportive, utilizing explanation and ventilation. We supplied Ms. A with information about the nature and etiology of her organic impairment. Discussion of Ms. A's distrust and guardedness led to consideration of her feelings about her marriage and her decision to undergo surgery. In view of the limited goals of treatment, we did not directly explore the explicit sexual content of Ms. A's thoughts. Rather, therapy focused on the ways in which sensations such as helplessness, confusion, and pain, in conjunction with medical-surgical procedures, could contribute to a sense of violation.

DISCUSSION

Ms. A suffered from a delirium manifested by intermittent lethargy, inattention, dysmnesia, and disorientation. It was mediated by a number of etiological factors: cardiac bypass, uremia, hypoxia, hypotension, sleep deprivation, and multiple psychoactive medications. As Ms. A's cognitive impairment resolved, it became apparent that her delirium had constituted a severely stressful life event. She attempted to conceal her concern; this was first evident when she casually sought information from ward personnel about her hospital course. Subsequently, Ms. A described frightening images of being deprived of water, having "tusks" grown in her throat, and being raped by a physician. Some of these images represented distortion of identifiable events. For instance, water deprivation was undoubtedly related to the fluid restriction that had been imposed. The "tusks" probably represented aspects of her respiratory or gastrointestinal care. The derivation of her memory of being raped is more obscure. Whether it had direct antecedents in her care, such as catheterization or rectal examination, or was a symbolic representation of her psychic experience remains unclear. However, Ms. A was certainly distressed by these thoughts, although she was inhibited in seeking help because of her concern that others would consider her a "terrible person." After discharge Ms. A had recurrent nightmares about being frustrated in her attempts to escape from the hospital. She ruminated over how so much could have "escaped her mind" during her illness and reproached herself for mistrusting her family and physicians.

Horowitz has observed a phasic response pattern in the aftermath of stressful life events (1). First comes an initial phase of outcry and incomprehension, followed by a period of denial and numbness. An oscillatory phase then emerges, characterized by fluctuation between the intrusion of traumatic information and attempts to ward off awareness of the stressful event and its implications. A phase of working through follows, marked by the reduction of emotional turmoil and acceptance of the event's meaning. As Horowitz noted, such a sequence assumes the stressed individual has access to a clear, correct perception of the event (2).

A person recovering from delirium is uniquely handicapped in terms of achieving resolution as outlined above. In contrast to most stress response states, which presume a clear sensorium at the time of the stressful event, the cognitive impairment characteristic of delirium prevents a clear and accurate perception of the delirium itself. Dysmnesia for the period of cognitive impairment may compound the problem. Delirious patients may accept fragmentary recollections of illusions, hallucinations, delusions, hypnogogic and hypnopompic phenomena, or dreams as reality. These patients may also equate dysmnesia with a loss of control and fear that they have acted out or been the victim of primitive impulses. Such distortions may increase emotional turmoil to the point that integration of the stressful life event is impaired. Some individuals may either reduce their access to information by denial and withdrawal or disguise their attempts to obtain information as casual inquiries. These responses may resemble disinterest or continued organic impairment and hence be overlooked. Fragmentary acquisition of information may result, confirming the patient's worst fears. For instance, scolding or humorous remarks might easily reinforce the patient's suspicion that something embarrassing or disgraceful had taken place. The patient may interpret statements such as, "You weren't yourself," to mean that some inexplicable change took place and may recur.

Once it is recognized that patients who have recovered from delirium may have insufficient or inaccurate information regarding the delirium itself to permit orderly resolution, the following steps should be taken. The patient and his or her family should be given a simple explanation of the nature of organic impairment. The frequent occurrence of unusual experiences during such a period should be noted. It may be useful to point out that everyone is vulnerable to organic impairment. Assurances should be provided that the patient is not losing his mind or suffering from an emotional illness. A general chronology of the disorder may be provided. Staff should be alerted to the possibility that the patient will obliquely seek information about the event. The patient should be given an opportunity to discuss concerns privately; apparently innocent and even rhetorical questions should be answered thoroughly. Discussion of specific events should be straightforward. Once the patient has expressed concern, it is likely that bland reassurance will only offer support for his or her worst fantasies. Clarifications may lead to a sufficiently clear and accurate understanding of the delirium to initiate resolution of the stress response syndrome. The therapist may then assess the indications for further psychotherapeutic intervention to facilitate the patient's assimilation of the traumatic information and its meanings, as described by Horowitz (1).

We cannot say whether the etiology and duration of a delirium are important to the appearance and resolution of stress response states. As in Ms. A's case, certain elements of the treatment may influence the nature of the disturbing recollections. Similarly, it is not clear whether preparation in situations associated with a high incidence of delirium would help minimize the nature of the posttrauma stress response state. Social supports, such as trusted family and friends, may serve to lessen severe postdelirium psychological distress. Patients such as Ms. A, with a premorbid tendency toward suspiciousness and aloofness, may be more inclined to withdraw and formulate a persecutory explanation for the delirium's occurrence.

We believe that many cases of delirium constitute a stressful life event and result in a stress response syndrome. Further, a distorted perception of the stressful event impairs the resolution of this syndrome. It follows that the psychiatric care of persons with delirium should be extended to include 1) recognition and facilitation of the patient's efforts to obtain information about the period of impairment and 2) psychotherapeutic assistance, when indicated, in assimilating the relevant information and its meanings.

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Research Directions in Child Psychiatry

BY IRVING PHILIPS, M.D.

Current research in child psychiatry emphasizes the understanding of biological mechanisms of behavior and its aberrations but neglects the transactional relationships in interpersonal phenomena. This situation may lead to an unwarranted enthusiasm for biological forms of treatment. The author calls attention to the importance of biopsychosocial elements of behavior in the understanding of growth and development. He discusses research directions to incorporate all elements of the behavioral sciences in the study of child behavior.

There are exciting developments in child psychiatry. The search for basic mechanisms is replacing the use of anecdotal impressions. Among the important investigations into basic biochemical and neurophysiological mechanisms and into classification of childhood psychopathology are those involving evoked response potentials in attention disorders (1), refined definitions of depression in childhood,[1] excretion of 3-methoxy-4-hydroxyphenylglycol in depressed children (2), the pharmacokinetics of serotonin metabolism in platelets, plasma, and urine in autistic children (3), and the breakdown products of norepinephrine in a variety of childhood disorders (4). Creative investigators are seeking biological markers of behavior. Child psychiatry is standing on a firmer base.

BIOLOGICAL EMPHASIS IN RESEARCH

Clinical studies, which are under greater scrutiny now, are still regarded as fuzzy, imprecise, intuitive, subjective, and offering little chance of comparability. The nature/nurture controversy has been reawakened, and the pendulum is swinging toward the side of nature. There is little question that basic science studies in the field are fundamental to the pursuit of ever more precise definitions of mechanisms of behavior. Whatever advances and understanding are achieved, it is important that we not lose sight of the transactional relationships in the interpersonal arena. Engel, in his essay on a new medical model, emphasized that "the physician's basic professional knowledge and skills must span the social, psychological and biological for his decision and actions on the patient's behalf involves all three" (5, p. 131).

Biology and rating scales have assumed ascendency in this time of emphasis on the biopsychosocial aspects of our specialty. There is relative neglect of both the psychological and social components within child psychiatry, although other disciplines are pursuing these lines. It was only a short time ago that the social aspects of deviant behavior were in the forefront, only to fade. Rarely today does one hear the refrain, "It is not the patient who is sick, it is society." As child psychiatry as a specialty comes under scrutiny from all quarters, we are retreating to the safe haven of the biomedical scientist. We compliantly underplay the psychosocial aspects of behavior and development as unworthy of study. We accept the derogation of others as our malady and withdraw into the study of the organism in more and more detail. The genotype, essential as it may be, has its phenotypic expression influenced by a variety of internal and external forces by means of complex interactive relationships, whether by cold, heat, or irradiation or by the quality of stress, attachment behavior, deprivation, or neglect. I am reminded of an oft-quoted remark of Seymour Kety, who noted that there is little question that in the near future we will understand the biochemistry of memory, but never of memories.

HYPERKINETIC DISORDERS

In the past decades we have been preoccupied with the hyperkinetic disorders of children. Epidemiologic studies have reported great variation in the incidence and prevalence of these disorders. The incidence of hyperkinetic disorders reported in various studies ranges from 5% to 20% of school-age children in the United States (6). Reports from England suggest

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[1] J. Puig-Antich and associates: Schedule for Affective Disorders and Schizophrenia for School Age Children (KIDDIE-SADS), unpublished.

markedly different rates. In the Isle of Wight study, Rutter and associates (7) found only .1% of the children afflicted with hyperactive attention disorders. Since the 1930s, investigators have reported the effectiveness of amphetamines and, later, methylphenidate in treating these children. The widespread use of these drugs continues[2] despite the fact that they have shown little effectiveness in improving learning (8) and that the findings in outcome studies of this population in adolescence and young adulthood are discouraging (9, 10).

The pursuit of basic mechanisms underlying this enigmatic disorder continues, as well it should. However, there is a pill and it may be effective, so physicians pursue the traditions of medicine—disease (ill-defined), a treatment (mechanisms poorly understood), and a response (questionable). Can there be that many disordered children with attention deficits? Are the English data poorly considered? Are the indications for treatment well-defined? Too many physicians proceed as if the hyperkinetic disorder had well-defined etiologies, expression, and outcome and as if there were similar findings among investigators throughout the world. At the same time, there is insufficient research on the expression of these disorders, the influence of external events, and the interactive elements of the stress factors involved. We respond to a set of clinical symptoms as if a defect is evident and the pharmacokinetics are understood.

DEPRESSION

Similarly, there has been increasing interest in the study of childhood depression. At the 1979 annual meeting of the American Academy of Child Psychiatry approximately 10% of the papers dealt with this topic; a few years ago there were few papers on the subject. In this area, too, we are confronted with the biomedical and psychosocial dichotomy. With some clarification of the genetics of manic-depressive illness, the formulation of the catecholamine hypothesis, the difference between endogenous and reactive depression, and the apparent effectiveness of antidepressant medication in adults, there is an awakening to seek similar biological antecedents of depression in children. Some studies describe a clearly delineated condition with affective, cognitive, motivational, and vasovegetative phenomena (11) similar to those of adult depressions; others describe more diffuse conditions such as masked depression or depressive equivalents, with symptoms ranging from encopresis to hyperactivity (12, 13). The incidence of the condition is reported to be as high as 30% among children who come to psychiatric clinics (14).

There has been relatively little focus on the expression of a childhood depressive disorder related to the developmental and cognitive abilities of the child (15). In the Isle of Wight study only 2 of 2,000 children were reported to have a depressive syndrome (7). Fortunately (or unfortunately) there is a drug to treat this condition, although its cardiotoxic effects limit its usefulness. The ingenuity of our commercial pharmaceutical chemists will eliminate that side effect. With a bandwagon of interest to attract ever greater numbers, who will hop on? Many children will be considered depressed and will be treated, although with somewhat limited benefit. Is childhood depression a well-defined syndrome with a well-defined etiology and outcome, and are there similar findings among investigators?

As we seek a definition, we use more and more rating scales, each idiosyncratic to the investigator who extols its virtues. We do not need more rating scales but, rather, a precise definition that is understandable by a variety of investigators.

THERAPEUTIC ENTHUSIASM

The hyperkinetic disorders and childhood depression are two examples of current research into basic mechanisms that do not sufficiently emphasize the psychosocial aspects of the disorder and therefore limit understanding of the condition. Such imprecision may lead to a therapeutic enthusiasm unwarranted by the data. Baldessarini (16) has pointed out that as we developed the phenothiazines, the diagnosis of schizophrenia was made more frequently. When lithium became available and effective for the treatment of manic-depressive illness, the disorder was reported more frequently than before in both children and adults. Suddenly we are reading reports of an increased incidence of manic-depressive disease in children (17), although it was rarely reported in the literature before the advent of lithium. Similarly, after a report describing the efficacy of haloperidol in the treatment of Gilles de la Tourette disease (18) (fewer than 500 cases had been reported in about the last 200 years), suddenly more than 500 new cases were reported in a New York City study alone during the past decade (19).

THE NEED TO QUANTIFY TRANSACTIONAL RELATIONSHIPS

It is difficult, but nevertheless essential, to quantify the transactional relationships, to enumerate clearly and concisely what we see in everyday clinical pursuits. It is equally important to develop more precise methods that quantify observations of patients about influences that affect remission and integration—ob-

[2]If we examine the scientific journals in pediatrics or child psychiatry we find pages of advertisements extolling the virtues of pills in making happier, brighter, and even more popular youngsters. The pages of advertisements alone suggest the market for the drug.

servations that lend themselves to enumeration and duplication. In a previous publication a colleague and I defined the steps in which this may be done. We recommended,

> 1) That each clinical research group undertake a painstaking study of its own clinical criteria of primary characteristics of childhood disorder. . . . Agreement among the group as to the name to be used and the specific clinical phenomena to which the name refers are essential for interobserver reliability. These agreements need to be thrashed out among the members of the group on the basis of observations of each patient by each or by several members of the group, perhaps aided by audio-visual recordings for repeated detailed review by all participants.
>
> As technology improves equipment, electronic apparatus will be cheaper and more widely utilized. Transistorized television cameras will allow the taking of pictures of the behavior of children in their everyday routine. Clinical investigators may then view, study and discuss the same clinical data and be more likely to arrive at an agreement on what is being described by specific terms and develop valid and reliable naming.
>
> 2) That clinical *differences* between patients need similar clear definition, specification of criteria and agreed upon naming of the symptoms. Here one thinks of such matters as the age, sex, . . . established age of onset of particular symptoms, their exacerbations, remissions, severity, etc., their relation to other events, the presence or absence of particular skills, traits, interests, the nature of relations towards self and others, somatic symptoms of identifiable diseases, and so on.
>
> 3) That elaboration from all this be undertaken of criteria of subgroups with similar congeries of characteristics. These subgroups may, and probably would, overlap depending upon the presence of some common attributes and the absence of others.
>
> 4) That enumeration of the various subgroups be performed for their frequency and for subsequent comparisons of the numbers of each subgroup responding, and not responding, to therapeutic maneuvers. Such data may be stored in computers for retrieval by various groups of investigators. . . .
>
> 5) That conferences be held between regional and eventually national, or even international, groups of such clinical research teams for review of the gathered data and for elaboration of agreements as to names, criteria, therapeutic outcomes, estimates of prognoses, and for the delineation of areas requiring further study. (20, pp. 288–289)

In our pursuit of the precise (this is not to be construed as an effort to negate any scientific investigation), we must not negate the interactional aspects of the biopsychosocial expression of any disorder. Although disease or defect may be one major contributing stress, the child's psychological disorder is probably not an inevitable concomitant of his or her biology but, rather, a function of the same kind of processes that give rise to emotional disorders in children who have no definable disease.

DISCUSSION

Engel emphasized that while reductionism is a powerful tool for understanding, it also creates profound misunderstanding when inversely applied. "Reductionism is particularly harmful when it neglects the impact of nonbiological circumstances upon biological processes" (5, p. 134). Any scientific statement considers all aspects of the field. *Mastery* of the psychosocial as well as *mastery* of the biophysiological comprise the behavioral sciences of child psychiatry. The neglect of either does a disservice to the basic understanding of the basic mechanisms of behavior and the expression of behavior in its social context. Child psychiatry is in a unique position to participate in the understanding of growth and development because of its scientific perspective through medical training, its developmental perspective through an understanding of the unfolding of innate patterns and social cultural influence, and its psychological perspective in the understanding of mental health and its deviations. Child psychiatrists must be vigilant not to neglect those aspects which they have been trained to observe best. The best use of clinical skills employs every scientific advance to elaborate and make vividly apparent the influence of brain and behavior as well as behavior and function.

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Documenting a Shortage of Psychiatrists: The Repair Shop Model

BY DAVID J. KNESPER, M.D.

The author presents a model for documenting a shortage of psychiatrists. The model employs efficiency, work load, and work time to determine numbers of psychiatrists needed for a hospital setting. Survey data collected from 900 psychiatrists were used to quantify these variables and to identify possible determinants of variation. Differences in residency training were associated with differences in efficiency. As presented, the model is incomplete and cannot be used to explicitly define manpower requirements. The author's discussion expands the model and focuses on the complexity of documenting a true shortage or surplus of psychiatrists.

In order to document a shortage or surplus of psychiatrists, it is necessary to assume a model that quantifies the associations between psychiatrists' work and consumer utilization of mental health services. A number of models do this (1), but they are generally used to make projections of future manpower needs and are not very useful for describing current requirements (2). This report is based on a model that is often neglected by research psychiatrists but appears to be used frequently in one form or another by policymakers concerned with allocating existing manpower resources to professional work settings. I call it the "repair shop model." For our purposes, the model in its simple form has the following five general properties: 1) there are multiple shops, and each shop has a fixed number of workbenches (e.g., 100 beds); 2) only one type of similarly broken unit needs repair (e.g., mentally ill individuals with exactly the same problem); 3) all workbenches are always occupied by a broken unit (e.g., 100 patients or 100% occupancy); 4) there are always a limited number of repair personnel (e.g., psychiatrists), who work at different degrees of efficiency during a workweek of 40 or more hours; and 5) personnel spend only a portion of their workweek doing repairs (e.g., no full-time service providers), and when a unit is repaired (e.g., patient is cured), it is immediately replaced by another broken unit.

Given this model and the examples from mental health, a question that the model can help answer is, How many psychiatrists are needed per 100 beds during a regular workweek? The answer is given by the following equation, which considers efficiency, work load, and work time: number of psychiatrists per 100 patients per week=[α hours effort per patient per workweek (median efficiency)] [100 patients (workload)] divided by [β service hours per workweek per one psychiatrist (median service time)].

The above equation has two unknowns: efficiency (α) and service hours (β). A survey research study was conducted to quantify these unknowns. The results from this study yield the number of psychiatrists needed per 100 patients for a hospital setting. The numbers vary according to the educational background of the psychiatrists and the work setting. As used in this paper, the repair shop model is of heuristic value and does not explicitly define manpower requirements. Therefore, the discussion will expand the mod-

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el and focus on the complexity of actually documenting a shortage of psychiatrists.

METHOD

During 1977 all psychiatrists graduating between 1961 and 1976 from 10 university and 17 state mental hospital residency training programs located in the state of Michigan and four similar states received a 23-item mail-return questionnaire. The states were chosen on the basis of several criteria, including a relative comparability in both total population and urban/rural distributions and the existence of established state-supported university and state mental hospital residency programs characterized in an earlier study (3). The questionnaire had been carefully designed, reviewed, pretested, and revised. This report considers the following questionnaire items: 1) the unduplicated number of inpatients and outpatients in direct treatment (face-to-face contact with patients—excluding consultation, supervision, and/or teaching), 2) the number of hours spent in different categories of professional activity (e.g., direct treatment, teaching, and administration), 3) primary professional practice setting (e.g., private practice, medical school, or public mental hospital), and 4) the respondent's medical and psychiatric residency background.

The Michigan Interactive Data Analysis System (MIDAS) was used to tabulate and analyze the results. Before analysis, the entire data set was checked for inadvertent coding errors and internal consistency.

RESULTS

Response and Respondent Population

Questionnaires were returned by 1,376 respondents (63%). Of these, 209 did not satisfy the criteria for inclusion in the survey and were therefore removed from the data set, as were 267 questionnaires from respondents who were classified as primarily child psychiatrists. Therefore, this paper reports results obtained from 900 graduates of general psychiatric residency programs.

When the 900 respondents were subdivided according to educational background, there were 182 foreign medical graduates (FMGs) from state mental hospitals, 65 FMGs from universities, 165 U.S. medical graduates (USMGs) from state mental hospitals, and 483 USMGs from universities; 5 respondents could not be classified on both dimensions. The respondents were principally employed in a private practice (53%), public practice (37%), or university (10%) setting. Slightly more FMGs than USMGs were in public practice settings, but otherwise the distribution of medical backgrounds according to practice settings was quite similar. Most respondents reported doing both inpatient and outpatient work; therefore, subsequent results probably have most utility for hospital settings that permit such a combination of duties.

Hours per Patient per Week

In order to derive effort, the unknown α (hours effort per patient per workweek), from the previously mentioned equation, the following respondent-supplied data were used: total direct-treatment hours per week was divided by total direct-treatment patients per week for each respondent. The ratio that resulted was defined as "effort" or hours per patient per week. The most remarkable differences in effort were found when residency training was considered. USMGs from state mental hospitals had a median effort of .49 hours per patient per week while USMGs from universities had a median effort of .84 hours per patient per week. Findings for FMGs from state mental hospitals and universities were similar to the USMG groups, and all values remained fairly constant regardless of year of graduation. These findings suggest that differences in residency training are associated with differences in subsequent patient practices.

Hours in Direct Treatment

In order to estimate service hours (the unknown β), data taken directly from the questionnaire were used. Both medical background and residency training were associated with median differences in service hours. Median hours in direct treatment were as follows: 30 for the USMG-state mental hospital group, 35 for the USMG-university group, 40 for the FMG-state mental hospital group, and 25 for the FMG-university group. Regardless of educational background, respondents worked a median of about 50 hours per week. Thus, educational background helps account for the distribution of hours in direct treatment as well as hours in such other professional activities as administration and teaching. FMGs spent the vast majority of their time delivering service if they had trained in a state mental hospital but divided their time among service, administration, and teaching if they had trained in a university. USMGs divided their time between service and administration if they had trained in a state mental hospital and among service, teaching, and administration if they had trained in a university. Very few respondents reported spending a major portion of their time in research.

Number of Psychiatrists Needed

The equation mentioned earlier and the data collected permit rough calculations of the number of psychiatrists needed to care for 100 patients in a hospital setting under present patterns of utilization of services and facilities. From the above findings, the number needed depends on the psychiatrist's style of effort or treatment (hours per patient per week) and the amount of work time devoted to service (hours in direct treat-

ment), which are associated with educational background and probably with other unmeasured variables. Considering educational background, the number of psychiatrists needed per 100 patients would be a minimum of 1.32 [(.53×100)÷40] if FMGs from state mental hospitals were used and a maximum of 2.48 [(.62×100)÷25] if FMGs from universities were used. Similar arithmetic finds that 1.63 USMGs trained in state mental hospitals and 2.40 trained in universities would be required per 100 patients. Rather than being definitive, these findings suggest there is a range of acceptable values that depends on such factors as who is employed, where the person was trained, and how the employed person spends his or her time.

Shortages and Surpluses According to Setting

The above calculations can be used to identify possible shortages or surpluses of psychiatrists in hospital settings offering inpatient and outpatient care. The shortage of psychiatrists in state mental hospitals appears probable: 15 states average 1 psychiatrist or fewer and 32 states average 2 psychiatrists or fewer per 100 state mental hospital inpatients; only 7 states average 4 or more psychiatrists per 100 inpatients (4). There are data to suggest possible surpluses: general hospital psychiatric units average 8.5 psychiatrists per 100 inpatients, and private mental hospitals average 9.3 psychiatrists per 100 inpatients (5).

These findings suggest what was suspected all along, namely, that there is an interaction between practice setting and effort. Within certain limits state mental hospital or university graduates adopted the style of effort imposed by the way patients are distributed among practice settings. Neurotic and other similar disorders that receive more hours of psychiatric care per episode of illness are more common in private settings than in public settings, where psychotic disorders are more common. Thus, compared with similar respondents working in a private hospital, FMG or USMG respondents trained in a university reported much less effort per patient when working in a state mental hospital. Similar patterns were found for respondents trained in state mental hospitals. These findings help document an apparent shortage of psychiatrists in state mental hospitals and a possible surplus in other settings. The previous statement may require modification when similarity of setting and the use of allied health personnel are considered.

DISCUSSION

A repair shop has an obvious manpower shortage when all available personnel are assigned to only necessary and essential tasks and are working at maximum efficiency, and when there is an increasing backlog of valued units in need of repair. The critical question that policymakers are asking is, Does mental health manpower conform to this analogy at the present time? Nobody knows the answer because descriptive data are unavailable. Left to their own devices, government and other policymakers are quick to assume a managed (mismanaged?) system and to find instances of apparently improper management practices. For example, private practice psychiatry may have sufficient manpower and possibly a surplus if, as many policymakers suggest, the work of private practitioners is inefficient (6–7). In contrast, public practice psychiatry appears to have a manpower shortage and many patients who are seriously ill but without needed treatment (8). Policymakers define this and similar situations in terms of maldistribution subject to distribution-changing public policy.

Although referring to psychiatric practice as a repair shop may offend the sensibilities of some readers, many policymakers think in these terms, and the analogy is not as outrageous as it first appears. Psychiatrists may have a predetermined and relatively stable number of hours during which they see inpatients and/or outpatients. Further, as this paper reports, efficiency varies; psychiatrists in some settings see a few patients in the same number of hours in which psychiatrists in other settings see large numbers of patients. Obviously, to make the repair shop analogy realistic a number of additional variables need to be introduced to account for variations. When this is done, a complex repair shop model results.

What complicates the model, and what therefore makes the shortage of psychiatrists difficult to document, is that psychiatrists do very different things during equivalent amounts of time spent in direct patient contact. This produces the variability in effort or efficiency. The mixture of patient types, treatment modalities, allied health personnel, and treatment outcomes undoubtedly varies enormously among practitioners and treatment settings. The number of individuals with a true need for mental health services (workload) is unknown, and whatever the number, it varies according to the definition of mental illness. In addition, such health economic factors as manpower supply and consumer demand cause variations (9). Unfortunately, there are no data about how the psychiatric profession differentially distributes its efforts across these and other relevant dimensions. In the absence of data, policymakers suspect that for similar patient problems, treatments differing in efficiency and cost are used but treatment outcomes are essentially alike. Private practice settings are considered inefficient because labor-intensive and unnecessarily elegant repairs may be attempted in situations where reduced labor, directed toward achieving less ambitious goals, would produce acceptable outcomes. If data were available, such suspicions could be properly evaluated, mechanisms that cause variations in efficiency identified, and rational interventions used to wisely alter manpower outcomes.

This report identifies one possible determinant of variations in efficiency. Differences in residency training were associated with differences in efficiency. Thus, regardless of foreign or U.S. medical education or practice in a public or private setting, psychiatrists trained in a state mental hospital were more efficient than their university-trained counterparts. The emphasis on biological treatments, the patient population, and the patient overload conditions under which many state mental hospital physicians are trained may account for this finding. Whatever the finding's origin, efficiency is only meaningful in terms of treatment outcome because some forms of seeming efficiency may turn out to be actually inefficient when outcome is considered. Consequently, it becomes increasingly important for psychiatry to investigate clinical accomplishments in outcome terms and combine these investigations with studies of manpower requirements.

Ignoring outcome, the data presented imply that policymakers have a choice between two different styles of training and treatment, with different treatments often used for similar patient problems. Because no one has documented that differences in residency training are associated with differences in patient outcome, policymakers may opt for subsidizing training emphasizing efficient treatments that are, at the same time, associated with desirable, if imperfect, clinical outcomes. Certainly, as this study suggests, such efficient treatments could reduce manpower requirements for psychiatrists.

Some methodologies for estimating manpower requirements forecast future needs rather than evaluating present manpower allocations. Projective methodologies may be an unproductive exercise in forecasting research, while utilization methodologies such as the repair shop model describe present manpower distributions with normative data in ways helpful for identifying policy options and making policy decisions. Readers interested in more sophisticated presentations of utilization methodologies are referred to the works of Navarro (10), Fries (11), and Bartholomew and Forbes (12).

"Guesstimates" and projections of manpower requirements will be found progressively less acceptable to policymakers. The profession, as well as government authorities, must begin to use more logical methods to describe and justify the present distribution of psychiatric manpower resources. A complex version of the repair shop model may be one such logical method for determining present requirements.

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From Supervisee to Supervisor: An Unexplored Aspect of the Psychiatrist's Education

BY ROGER B. GRANET, M.D., THOMAS P. KALMAN, M.D., AND MICHAEL H. SACKS, M.D.

The authors describe several problems that are unique to the beginning supervisor of psychiatry residents. These difficulties can be divided into four general areas: inexperience, competition, identity, and administration. The authors provide examples to illustrate the types of situations that the new supervisor may encounter and make recommendations for dealing effectively with them.

Individual supervision of residents' clinical work is the traditional method of teaching in psychiatry, where it occupies a more dominant position than in most other medical specialties. A great deal has been written about the purpose, function, techniques, and problems of supervision, but virtually all of this material has dealt with the process from the perspective of the mature supervisor (1–9). Little attention has been directed toward the neophyte who has recently advanced from the role of supervisee to that of supervisor, even though this transition has a powerful potential for stimulating both professional and personal growth. This paper is an attempt to address this neglected area. Two of the authors (R.B.G. and T.P.K.) are writing from the perspective of having made the transition over the past 2 years; the third author (M.H.S.) is writing from the perspective of having observed neophyte supervisors for more than 10 years.

REVIEW OF THE LITERATURE

In discussing the social context of psychoanalytic supervision, Emsch (10) has listed the components involved; they include the supervisor, student, patient, and the student's analyst. This model is representative of the trend in the analytic literature to view supervisors as finished products; they are regularly portrayed as already fully educated, analyzed, and supervised.

Potential pitfalls for the supervisor include disciple hunting, rivalry with the supervisee's analyst or therapist, and, finally, using supervision to resolve administrative or intellectual conflicts (11). Schneider (12) emphasized the last problem in a discussion of the dangers of using patients to act out professional difficulties. Similarly, supervisors may use students manipulatively to resolve conflicts with colleagues. Searles (13) pointed out the danger of the supervisor's intellectual competitiveness with the student. He also described the special supervisory problem of allowing the growth and ultimate separation of the supervisee. Supervisors must be capable of dealing with the reality that as their pupils grow, they will be needed less.

In elaborating on an analysis of the beginning supervisor's anxiety, Kaplan focused on the need to fit in with a series of supervisory styles.[1] Schuster and associates, in turn, observed, "Hopefully, the supervisor has been able to achieve his own 'integrity,' in order to be usefully adopted as a role model, the primary and essential ingredient of supervision" (1, pp. 73–74).

COMMON DIFFICULTIES OF THE NEW SUPERVISOR

The problems of a beginning supervisor can be conveniently divided into four general areas: inexperience, competition, identity, and administration. These categories are intended not as strict nosological criteria but rather as a guiding descriptive framework from which to approach the difficulties of the beginning supervisor. There also may be considerable overlap between these categories, and the boundaries between them often are not absolute.

Inexperience

Problems of inexperience include not only the realities of little prior teaching experience but also inadequate clinical and theoretical knowledge. After the

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[1] E.A. Kaplan: Psychotherapy supervision: the role of the supervisor (panel discussion). presented at the 124th annual meeting of the American Psychiatric Association, Washington, D.C., May 3–7, 1971.

completion of a four-year residency in psychiatry, new supervisors are just beginning to develop a firm clinical foundation for themselves. Understandably, there will be much that they have not yet mastered, and difficulty in accepting this will result in problems.

An example of this situation occurred during a supervisory situation with a resident who had already begun her psychoanalytic training. When she focused the discussion on the dynamics of a paranoid patient, the neophyte supervisor shifted the topic to biological management and lectured the supervisee on the side effects of psychotropic medication. The shift involved the supervisor's feelings of inadequacy about the psychodynamic material, which were intensified by the resident's own advanced work (analytic training) in dynamic psychiatry. It was not until after the session that the supervisor realized that the resident's focus on the dynamic material was an effort to avoid her own anxiety regarding the patient's potential violence. The supervisor's inexperience and anxiety prevented him from recognizing the resident's needs and interfered with the understanding of the patient.

Competition

The second problem area, competition between supervisor and resident, often goes hand in hand with problems of inexperience. The closeness in age between the new supervisor and resident often fuels comparisons, identifications, and rivalries that may trigger anxiety in the supervisor about his or her own worth, causing the supervisor to question, "Did I learn and assimilate anything during my own training?" This may lead to unconscious strategies such as giving too many facts, overquoting the literature, and focusing on areas of special knowledge when they are not relevant.

An example of this situation occurred during a supervisory session in which an outstanding resident had completed a presentation that included an organic psychosis as part of the differential diagnosis. The new supervisor had just published an article in this area and began a 15-minute discourse on the article, neglecting the more pertinent particular needs of the resident. It was only afterward that the supervisor realized that he had set aside the educational needs of the resident in favor of acting out his insecurities, his need to establish his credibility, and his competitive feelings toward the resident.

Another example of a neophyte supervisor's competitive feelings toward a resident emerged during the collaborative writing of a paper by a first-year resident and his first-year supervisor. The two had agreed to try to publish a case report of a patient whom the resident had treated and who manifested an interesting psychopharmacological problem. They agreed at the outset that the resident would be first author and the supervisor second author, but after they had exchanged several drafts the supervisor realized that they were having difficulty arriving at a final manuscript. It then emerged that the supervisor had not really been able to accept the resident's significant contribution and had become overly critical of stylistic differences. Although there was complete agreement between the two authors on the content of the paper, they exchanged rephrased drafts in a struggle by each to have the paper written in his own hand. Resolution came about only when the supervisor was able to "let go" as he realized that the differences (quite literally over punctuation and sentence structure) were related to his inappropriate competitive feelings toward the resident.

Identity

Identity problems for the neophyte supervisor can be summarized by the question, Is he or she an "old resident" or a "young professor"? This conflict underscores a major dilemma of supervision itself, namely, what is the function of a supervisor? Is the supervisor a supporter or teacher, therapist or educator, friend or role model? Confusion about roles may be manifested in the supervisor's being overly supportive to the supervisee, neglecting both the real needs of the resident and the clinical needs of the patient. The converse may also occur, i.e., the supervisor may become overly analytic in order to prove himself or herself in the new professional role.

An example of this difficulty occurred during a resident's presentation of the case of an assaultive patient. The resident expressed a realistically appropriate degree of anxiety about his patient, but the supervisor found himself assuming the role of therapist and tried to focus on possible neurotic determinants of the resident's anxiety. The supervisor thus neglected the more crucial and appropriate issue of management of the assaultive patient. If the supervisor had spent more time discussing patient management, he could have imparted new knowledge to the supervisee, providing a needed sense of direction and mastery. Instead, the focus on the resident's anxiety was infantilizing, supported the resident's sense of inadequacy, and did not help his faltering self-esteem. The situation called for the supervisor to act as an educator, and his failure to assume this appropriate role led to the mishandling of the supervision.

The difficulties related to identity may also emerge in the new supervisor's relationships with former teachers. The new supervisor may be unable to relate comfortably to former teachers as peers. This situation may lead to an inappropriate identification with the resident being supervised, resulting in a maladaptive supervisory identity and hence poor teaching. Identification with the resident may temporarily facilitate the interpersonal relationship for the young supervisor who is uncomfortable with this new position of authority, but it must be recognized that this is a maladaptive identification and is inappropriate to the teacher-student role.

Administration

The final problem area involves the supervisor's administrative responsibilities. This is a particularly tenuous area for the young supervisor who is struggling with the issues of inexperience, competition, and identity. An example of this occurred at the conclusion of the supervisory semester when the training director requested a formal evaluation of a resident. Although the neophyte supervisor had found the resident to have significant difficulties, he felt that with appropriate direction the resident would develop into a talented and responsible psychiatrist. It was apparent that the young supervisor's perception was very different from that of senior supervisors, and he feared that his judgment might reflect his immaturity and result in the loss of his seniors' respect for him. This supervisor felt considerable pressure to modify his own evaluation, since it was discrepant with the evaluations done by others.

New supervisors' uncertainties about their superiors' view of them and their wishes for approval are important causes of anxiety for many. Concerns about their evaluation as supervisors and the effect that the evaluation may have on promotion can be strong influences. These pressures may lead inexperienced supervisors to avoid conflict-laden situations that would normally involve their taking a more active administrative role. An example of this occurred when a resident indicated a wish to work with a psychotic patient without the use of medication. The supervisor actually felt that this was justified both as a reasonable treatment for the patient and as a significant step in the growth of the resident, who was now more able to tolerate the psychotic behavior of his patients. However, the supervisor's administrative chief strongly opposed psychotherapy with psychotic patients and belittled those who used such techniques. The new supervisor avoided this potential conflict by insisting that the resident obtain the necessary administrative approval before proceeding with the therapy. The supervisor thus avoided any responsibility for the treatment decision because of his own reluctance to cross swords with an administrative superior. This inappropriately left the resident to resolve the conflict himself and also provided a less than desirable role model for him to follow.

CONCLUSIONS

In this paper we have focused on the problems faced by the neophyte supervisor as he or she undertakes this new professional role. It should not be forgotten that there are a number of unique joys and gratifications available to the beginning supervisor. Teaching provides an opportunity for further personal learning while providing for the learning of others; in addition, supervision enables the supervisor to participate in the growth and expansion of his or her profession by bringing others into it. Finally, the supervisory experience provides a nidus for professional and personal growth, reinforcing the concept that being a psychiatrist is a continuous educational process.

There are several ways by which the new supervisor's experience and transition can be facilitated. One is by formal education and training in supervision, such as through the supervisory conference (as organized at the University of Rochester, N.Y., in 1970). This approach entails a meeting of supervisors, who listen to audio tapes of residents' therapy sessions and then discuss the different approaches that could be taken toward effective supervision and therapy. Faculty members are then able to reflect on their differing styles and approaches to supervision (1, pp. 166–325).

Another approach is that found at the Payne Whitney Clinic of Cornell University Medical College, which uses videotapes to record actual supervisory hours. The tapes are then available for "supervision of the supervision." Other supervisors can view the tapes and discuss the process in addition to suggesting alternatives that might improve the supervision. At times, the supervisee may join the conference and provide his or her own perspective. An additional use is the viewing of the taped supervisory session by senior residents and junior faculty psychiatrists, who themselves are beginning to supervise others in training. These tapes can thus be used for the supervision of the supervisor in the same way that tapes of resident-conducted interviews are used for the education of the resident in clinical skills.

An additional way for neophyte supervisors to adjust to their new responsibilities is to continue to receive supervision on their own patients. This will increase their knowledge and expertise while allowing them to continue the experience of being supervised by a senior colleague. Another method for enhancing the role of supervisor is to have joint discussions with other supervisors about the individual supervisees. This allows for a sharing of supervisory experiences and facilitates the identification of problem areas.

Beginning supervisors and the special problems they face have been neglected topics of study and discussion. We feel that we have made potentially helpful suggestions for dealing with common difficulties of new supervisors. Clearly, however, further investigation into this half of the supervisory dyad is called for.

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Importance of Akinesia: Plasma Chlorpromazine and Prolactin Levels

BY THEODORE VAN PUTTEN, M.D., PHILIP R.A. MAY, M.D., AND JEFFERY N. WILKINS, M.D.

Akinesia, a common side effect of antipsychotic drugs, often goes unrecognized by physician and patient. Akinetic apathy and lack of spontaneity can be mistaken for the negative symptoms of schizophrenia and add to the well-known social and emotional disability of schizophrenic patients on maintenance therapy. The authors attempted to identify a measure that might distinguish between akinesia and the negative symptoms of schizophrenia but found no relationship between plasma and saliva chlorpromazine levels or prolactin levels and akinesia. The fact that all of the akinetic but only 31% of the nonakinetic patients rated themselves as drowsy 12 hours after their bedtime dose indicates that drowsiness is a fairly accurate correlate of akinesia.

Akinesia, even in a mild form, is socially disabling. Defined by Rifkin and associates as a "behavioral state of diminished spontaneity characterized by few gestures, unspontaneous speech, and, particularly, apathy and difficulty with initiating usual activities" (1, p. 672), this side effect of antipsychotic drugs is often not recognized. Akinesia is mistaken for the negative symptoms of schizophrenia, adding to the well-known social and emotional disability of schizophrenic patients on maintenance therapy. Most important, the onus of detection is on the physician, for the patient seldom complains. Indeed, even when asked if he feels "slowed up" or "sluggish," the patient often denies any difficulty whatever, seemingly locked in some peaceful apathetic remoteness. These are bold assertions, born more of clinical experience than of controlled investigation, but there is some controlled evidence that akinesia has far-reaching consequences.

Falloon and associates (2) compared the social outcome of schizophrenic patients maintained on either pimozide or fluphenazine decanoate. At 1 year those on pimozide were rated as having a significantly more favorable outcome on aspects of sociability, use of leisure time, warmth of personal relationships, household tasks, and child rearing. These improvements could not be related to superior antipsychotic effect but seemed to be related to the fact that the patients on pimozide experienced less extrapyramidal rigidity. The authors speculated that akinetic "flatness of expression" and "psychomotor retardation" were responsible for the poorer social adjustment of the patients taking depot fluphenazine. Argyle (3) has suggested that nonverbal signals have a far greater impact than verbal ones on assertiveness and friendship, thus implicating a relationship between lack of spontaneity and social adjustment.

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Finally, in our own work, when we compared akinetic patients with a similar group without akinesia, we rated the former significantly worse on such measures as somatic concern, anxiety, depressive mood, and blunted affect, leading to a speculation that some postpsychotic depression is really akinetic depression (4).

It is difficult to differentiate schizophrenic apathy and blunting from extrapyramidal side effects. Nevertheless, we fear that some—perhaps many—schizophrenic patients are victims of akinesia. In a recent survey of community board and care facilities, the residents with schizophrenia were maintained on a mean (± SD) of 760±540 mg/day of chlorpromazine equivalent, and 59% appeared to be at least mildly akinetic (5). (It is possible that akinetic apathy and lack of initiative may even be welcome because these side effects promote organizational efficiency.)

We thought it would be helpful to have a measure that could distinguish between akinesia and the negative symptoms of schizophrenia or the psychomotor retardation of depression. Constriction of handwriting (micrographia) has turned out not to be a reliable correlate of akinesia (6). We have therefore attempted to determine whether patients with akinesia have higher plasma levels of antipsychotic drug (chlorpromazine) or of prolactin.

METHOD

The subjects were 43 schizophrenic patients newly admitted (or readmitted) to Brentwood Veterans Administration Medical Center who had given informed consent; most were in young middle age (mean age=32.3±9.9 years) and had a chronic disorder (mean duration=6.4±6.93 years) and a fair prognosis. They were rated on four different sets of diagnostic criteria for schizophrenia (7–11), and all 43 were considered to be definitely schizophrenic.

When this was clinically permissible, a 2- to 7-day washout period was provided; most patients, however, had not taken medication recently and could be presumed to be drug free on admission. We treated all 43 patients with a bedtime dose of chlorpromazine (6.6 mg/kg per day) for 28 days. Prophylactic antiparkinson drugs were not administered.

Chlorpromazine was measured in plasma and saliva by gas chromatography-mass spectography with *N*,*N*-bisperdeuteromethyl chlorpromazine ($^2H_6$-chlorpromazine) as an internal standard. Details of the collection and assay methods have been given elsewhere (12). Plasma prolactin levels, determined at the same time as and from the same blood samples used to determine chlorpromazine levels, were measured with a homologous radioimmunoassay technique (13).

One of us (T.V.P.), blind to the patient's plasma chlorpromazine and prolactin levels, continuously observed each patient for 28 days and rated the presence of akinesia on an extrapyramidal symptom scale, which has been described elsewhere (4).

Drowsiness was rated weekly at 9:00 a.m. by 1) the patient's self-rating, scored on a 7-point scale from 0 (not at all) through 3 (mild) to 6 (severe, actually asleep), and 2) laboratory staff on a 10-point scale from 0 (no drowsiness or sedative effect) through 5 (very drowsy, both eyes open, no nodding) to 9 (cannot be roused).

RESULTS

Of the 43 patients, 10 developed akinesia by day 26 of treatment (SD=13) and 29 did not; 4 developed a dystonic reaction early in the study and were maintained on an antiparkinson drug and excluded from further analyses.

Graphic representations of plasma and saliva chlorpromazine levels revealed nearly complete overlap between the akinetic and nonakinetic groups. The groups did not differ substantially in age or chlorpromazine dosage. In fact, the patients who developed akinesia received a lower dose of chlorpromazine (mean=495 mg/day) than the nonakinetic patients (mean=553 mg/day). Plasma prolactin levels also did not differ.

The patients who became akinetic were drowsier on both objective and subjective ratings. All of the akinetic patients rated themselves as drowsy (mean rating=2.9±1.6) in the morning 12 hours after their bedtime dose, compared with only 31% of the nonakinetic patients (mean rating=1.7±1.0). Objective ratings were similar to the subjective ratings: the akinetic group had a mean rating of 3.1±1.6, and the nonakinetic group had a mean rating of 1.4±0.7.

DISCUSSION

Our results lead us to conclude that plasma chlorpromazine or prolactin or saliva chlorpromazine levels are of no help in identifying akinesia and in distinguishing schizophrenic blunted affect, apathy, and motor retardation from akinetic lack of spontaneity. When blood was drawn 12 hours after the bedtime dose, patients who developed a definite mild akinesia had similar chlorpromazine and prolactin levels to those who did not. We doubt that the akinetic patients have higher levels of active chlorpromazine metabolites, although we cannot prove it.

We found that a simple and fairly accurate correlate of akinesia is drowsiness. If a patient rated himself as mildly drowsy 12 hours after his bedtime chlorpromazine dose, we calculated that the chances are 63% that he is akinetic and only 4% that he is not. If a patient in our sample denied any feelings of drowsiness, there was no chance of his being akinetic.

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CLINICAL AND RESEARCH REPORTS

This section contains 1) new research findings, including preliminary data from pilot studies, either clinical or laboratory; 2) worthwhile replication studies; 3) case reports that describe a truly new syndrome or cast new light on established ones; and 4) case reports that indicate a new therapeutic procedure of potential value or call attention to adverse effects of drugs or previously unreported complications of therapeutic interventions. Program descriptions and literature reviews cannot be printed in this section. Criteria for format are listed in "Information for Contributors" in each issue; papers that do not adhere to these criteria will be returned to the author.

Cerebral Blood Flow in Depression

BY ROY J. MATHEW, M.D., D.P.M., JOHN STIRLING MEYER, M.D., DAVID J. FRANCIS, KAREN M. SEMCHUK, B.S.N., KARL MORTEL, M.A., AND JAMES L. CLAGHORN, M.D.

Until 1975 quantitative measurement of the regional blood supply to the brain was difficult to determine because of the traumatic nature of the procedures available (1). Today, regional cerebral blood flow can be measured in a safe and reliable manner using the ^{133}Xe inhalation technique (2, 3).

In this technique ^{133}Xe, a chemically inert and freely diffusible tracer gas, is mixed with ambient air and inhaled through a sterilized, close-fitting face mask. After a 1-minute interval of inhalation, the desaturation activity of the isotope is monitored for 10 minutes by a system of 16 collimated probes mounted in a helmet and applied to the scalp and by the measurement of end-tidal ^{133}Xe activity. Values for regional cerebral blood flow are calculated by two compartmental analyses of the recorded curves by a minicomputer during the 10-minute desaturation interval. The analysis is based on a modification of the Fick principle and suitable computer programming. End-tidal ^{133}Xe curves, recorded from the face mask, are used for correction of xenon recirculation to the brain. ^{133}Xe emits gamma activity derived from the brain so that the desaturation curves of gamma activity are used for regional cerebral blood flow measurement. Previous reports (1–4) have demonstrated that the noninvasive ^{133}Xe inhalation method is highly reproducible and in good agreement with results obtained by the ^{133}Xe intracarotid injection method of Ingvar and Lasson.

The ^{133}Xe inhalation method has several advantages over the intracarotid method. First, it is noninvasive. The inhalation technique measures blood flow through both the cerebral hemispheres simultaneously and the brain stem, whereas the injection method measures blood flow through only one hemisphere at a time and it does not measure brain stem blood flow. Although positron emission tomography quantifies brain metabolism in three dimensions, the technique described above provides a two-dimensional measurement of brain blood flow. A comprehensive account of the procedural details and instrumentation of this system has been described elsewhere (1–4).

Little is known about human cerebral blood flow during depressed states. A recent study reported abnormal rheoencephalographic measurements in circular depressions with a return to more normal measurements following ECT (5). Rheoencephalographic measurements are based on impedance between scalp electrodes and are a complex index influenced by changes in pulse, scalp flow, cerebrospinal fluid, and cortical flow changes (1). This study examined regional cerebral blood flow by using the ^{133}Xe inhalation technique in a group of untreated depressed patients.

Method

Thirteen patients (4 men and 9 women; mean age (±SD)=30.07±7.33 years) with a diagnosis of major

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depressive disorder according to Research Diagnostic Criteria (6) were selected from the inpatient and outpatient clinics of the Texas Research Institute of Mental Sciences. We obtained informed consent, and we then excluded patients with physical illnesses by conducting a physical examination and routine laboratory tests. All subjects underwent a medication washout period of 2 weeks, at the end of which their regional cerebral blood flow was measured using the ^{133}Xe inhalation technique.

Immediately before the blood flow measurement, the patients underwent a detailed psychiatric examination by a Board-certified psychiatrist; their degree of depression was quantified using the Hamilton Rating Scale for Depression (7). Regional cerebral blood flow measurements were made under standard laboratory steady-state conditions (3). A control group consisted of 13 healthy volunteers matched for age, sex, and handedness who had undergone regional cerebral blood flow measurements under the same conditions.

Results

The depressed group of patients obtained a mean score (±SD) of 25±11.24 on the Hamilton Rating Scale for Depression. The gray matter flow values (Fg) for the two groups were compared using Student's t test. The depressed group was found to have significantly lower Fg values for the left hemisphere compared to the age-matched nondepressed normal controls (depressed group mean (±SD) for left hemisphere Fg=75.52±11.71 ml/100 g brain wt per minute; control group mean for left hemisphere Fg=88.66±16.94 ml/100 g brain wt per minute; $t=2.27$, $p<.03$). A similar trend was observed for the right hemisphere; however, the difference failed to reach statistical significance (depressed group mean for right hemisphere Fg=75.26±15.09 ml/100 g brain wt per minute; control group mean for right hemisphere Fg=86.18±14.48; $t=1.84$, $p<.07$).

The regional cerebral blood flow Fg values of the depressed group were also correlated against the Hamilton depression scores. The left and right hemispheric Fg values of these patients showed highly significant inverse correlations with the depth of depression (left hemisphere, $r=-.63$, $p<.009$; right hemisphere, $r=-.62$, $p<.01$).

Discussion

The present report indicates that in severe depression unassociated with organic brain disease there is a bilateral hemispheric reduction of cerebral blood flow similar to the reduction seen in conditions associated with the loss of brain tissue, such as in Alzheimer's disease, multi-infarct dementia, and aging (8). Symptoms of cognitive dysfunction, such as attention, concentration, and memory defects suggestive of mild diffuse organic dysfunction of the brain, are frequent accompaniments of depression. Serious impairments of these functions due to depression in the elderly may mimic organic dementia and are termed the pseudodementias (9). Regional cerebral blood flow has been shown to reflect levels of neural activity, and areas of increased neural metabolism have been associated with regional cerebral vasodilation (10). The present finding of decreased regional cerebral blood flow in depression seems to suggest neural hypoactivity in view of the fact that depression is a reversible illness unassociated with brain tissue loss. It must be pointed out that the association between cognitive dysfunction in depression and reduced brain blood flow reported here is speculative and that it needs replication with a larger sample of subjects and psychometric testing. It is difficult to compare the findings reported here with the data based on rheoencephalographic measurements because these measurements are influenced by several factors other than brain blood flow.

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A Case Report of Cimetidine-Induced Depressive Syndrome

BY MILES K. CROWDER, M.D., AND J. KIRBY PATE, M.D.

Cimetidine, a histamine H_2 receptor antagonist, is being prescribed increasingly to decrease gastric acid secretion in a variety of gastrointestinal disorders, particularly duodenal ulcer and Zollinger-Ellison syndrome (1). It is estimated that more than 11 million patients have been treated with cimetidine since its introduction in 1976 (2). Neuropsychiatric toxicity associated with cimetidine, with such manifestations as mental confusion, coma, visual hallucinations, lethargy, delirium, and agitation, has been noted in numerous case reports (3–9). The present report describes a patient in whom depression was probably induced by cimetidine.

Case Report

Mr. A, a 48-year-old man, was admitted to our hospital with a chief complaint of depression. He had noted mild subjective symptoms of depression 1 year previously and had experienced a definite increase in these symptoms 7 months before this admission. He reported sadness, feelings of worthlessness, diurnal variation in his mood, hypersomnia, loss of interest in usual activities, and a general sense of disappointment about his life. During the few weeks before his admission he had been unable to work and had contemplated suicide.

Mr. A's medical history was remarkable for recurrent peptic ulcer disease, dating from early adulthood, which had been treated successfully with 300 mg of cimetidine q.i.d. for the previous 7 months.

Because of the temporal relationship between the onset of Mr. A's severe depressive features and the initiation of cimetidine therapy 7 months before his admission, the cimetidine was discontinued on the 2nd hospital day. By the 10th hospital day (the 9th day Mr. A received no medication) his depressive symptoms had improved so dramatically that he no longer qualified for an antidepressant drug study. On the 13th hospital day 300 mg of cimetidine q.i.d. was reinstituted. A reevaluation of Mr. A on his 16th hospital day revealed that his depressive symptoms had returned, and a Hamilton Rating Scale for Depression score of 24 was obtained. Cimetidine was again discontinued, and again his depressive symptoms quickly remitted. His Hamilton Rating Scale for Depression score on the 23rd hospital day had decreased to 12.

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Discussion

Neuropsychiatric side effects related to cimetidine are rare, and when they do occur, they usually manifest themselves as an organic brain syndrome. To our knowledge there is only one previously reported case of depression related to cimetidine use; this occurred in an 81-year-old man whose depressive symptoms remitted rapidly after discontinuation of the drug (10). In that report Jefferson noted that neuropsychiatric complications are more likely to occur in the presence of advanced age, impaired renal function, associated serious medical problems, and a higher dose of cimetidine than that recommended.

To our knowledge this is the second documented case of clinical depression related to the use of cimetidine. It is especially remarkable for two reasons. It occurred in a middle-aged man who was medically well (with the exception of his peptic ulcer disease) and who lacked any of the predisposing characteristics cited by Jefferson. Also, there was a striking temporal relationship between his ingestion of the drug and the severity of his depressive symptoms, as well as his fluctuating Hamilton Depressive Scale scores.

Given the initial enthusiasm concerning the therapeutic efficacy of cimetidine, the drug is likely to be increasingly prescribed, with a resulting increase in the frequency of neuropsychiatric complications. It appears that both the organic brain syndrome and the less frequent depressive syndrome related to cimetidine are readily reversed by discontinuing the drug. Therefore, it is vitally important that psychiatrists, internists, and family practitioners be aware of the possible neuropsychiatric complications of this promising medication.

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Prolonged Confusional State and EEG Seizure Activity Following Concurrent ECT and Lithium Use

BY RICHARD D. WEINER, M.D., PH.D., ALAN D. WHANGER, M.D., C. WILLIAM ERWIN, M.D., AND WILLIAM P. WILSON, M.D.

In the recent APA survey on electroconvulsive therapy (ECT), only 20% of the psychiatrist respondents felt that lithium carbonate and ECT were incompatible (1). At the same time, however, there is growing evidence (2–6) that this combination may be associated with the development of a severe acute organic brain syndrome, even in patients who tolerate either treatment well when it is given separately. Although only Ray (3) and Small and associates (6) have reported the occurrence of seizure activity as a part of such a syndrome (3, 6), the common presentation of waxing and waning disorientation and confusion is similar to certain types of seizure manifestations. Therefore, we present here a case in which the patient's EEG showed evidence of status epilepticus while at the same time the patient was in a fluctuating confusional state.

Case Report

Ms. A was a 33-year-old woman with a 14-year history of intermittent psychotic depressions, rapidly fluctuating moods, occasional auditory hallucinations, and several suicide attempts. In the past her symptoms had been difficult to manage, and she had had only fair response to a wide variety of antidepressants and antipsychotic medications, hospitalizations, and ECT. About 2 years previously and at another facility she had received her last course of ECT, and then she was placed on lithium carbonate, tranylcypromine, and perphenazine. Lithium and ECT were never given concurrently. While taking 900 mg/day of lithium, Ms. A had a steady-state lithium level around 0.8 mEq/liter. During her current episode, which lasted several months, she had received no psychotropic medication. Ms. A had no personal or family history of epilepsy or other neurologic disease, and at the time of this admission and before her ECT treatment her waking and light sleep EEG was normal.

Because of Ms. A's persistent and severe level of psychotic and suicidal ideation, as well as her treatment history, it was decided to treat her with ECT and a course of bifrontotemporal treatments given three times a week was begun. The ECT treatments were modified by atropine, succinylcholine, and thiopental sodium for injection; the electrical stimulus was provided by a MECTA ECT device (using a bidirectional pulse stimulus). The sessions were monitored electroencephalographically with 80- to 90-second seizures produced at each treatment.

Ms. A responded well clinically to ECT; her depressive symptoms were relieved and by the fifth ECT treatment only mild confusion and memory impairment were noted. In order to facilitate Ms. A's early discharge from the hospital, lithium carbonate was begun the day after her fifth ECT treatment; the initial dose was 300 mg administered twice daily. The next day, approximately 6 hours after the second dose of lithium, it was noted that Ms. A's confusion was increasing; she became disoriented and, at times, unresponsive to questions. At no time were motor manifestations of convulsive activity present. The following morning, before her fourth lithium dose, Ms. A's lithium level was 0.37 mEq/liter. Later the same day the lithium was discontinued. Ms. A's behavioral changes continued to persist, however, and 2 days later an EEG recording revealed diffuse regular and irregular slowing that was punctuated by prolonged runs of bilaterally synchronous, high amplitude, triangular shaped waves at about three per second. During this recording her background EEG activity was markedly diminished, and she was unresponsive to questioning. The following day while Ms. A was under EEG monitoring, it was determined that both her extreme confusional state and the paroxysmal EEG activity were responsive to two brief 5-mg injections of diazepam i.v. given 10 minutes apart. Following these injections, Ms. A was maintained on oral diazepam. This was replaced by valproic acid when a repeat EEG administered 1 week later continued to show abortive paroxysmal activity manifested by brief runs of diffuse, bilaterally synchronous notched delta activity at 2 to 3 Hz. Ms. A was given an EEG every 1–2 weeks, which revealed a slow return to pretreatment EEG activity by 9 weeks, following the fifth and final ECT treatment. Lithium carbonate was reinstituted 6 weeks after completion of the sessions, and she had no apparent further episodes of confusion or deterioration.

Discussion

This case report documents the occurrence of a prolonged confusional state and electrographic seizure activity in a patient taking lithium carbonate during the latter portion of a course of ECT. In 1975, Ray (3) reported the case of a patient being maintained on lithium who developed absence seizures 4 days after the third ECT treatment; the patient's serum blood level was only 0.46 mEq/liter. More recently Small and associates (6) reported the occurrence of a right temporal lobe epileptic focus in a patient receiving a combination of lithium and right unilateral nondominant ECT. The clinical presentation of patients with generalized

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seizures restricted to absence manifestations, e.g., alterations of orientation, cognition, and consciousness, is quite similar to that given in some of the reports of acute organic brain syndrome associated with lithium neurotoxicity. Such side effects can occur with or without concurrent use of ECT or psychopharmacologic agents, and they do not appear to be related to serum blood levels. Because the EEG may often be a definitive procedure in uncovering the presence of underlying epileptiform activity, its use is crucial in the evaluation of such patients.

The mechanism of epileptogenicity of the ECT/lithium combination is unclear. The occurrence of spontaneous seizures following ECT itself has become exceedingly rare since the widespread use of anesthesia modification. Indeed, electrically induced generalized seizures may exert an anticonvulsive effect upon epilepsy in humans (7) and interrupt the "kindling" phenomenon in animals. Because CNS norepinephrine activity appears to correlate inversely with seizure susceptibility, it is tempting to relate such anticonvulsive action of ECT to its known potentiating effects upon CNS norepinephrine release, concentration, and turnover. The presence of such a relationship, however, is confounded by a reported inverse effect of repeated electrically induced seizures upon the density of CNS β-adrenergic receptors and the activity of norepinephrine-stimulated cyclic AMP (8). Yet, because lithium acts both to decrease CNS norepinephrine release and to inhibit the actions of anticonvulsant drugs (9), a possible role for norepinephrine mechanisms in the epileptogenicity of the lithium/ECT combination may be worthy of further consideration. Presumably such a shift in CNS norepinephrine balance might unmask the potentially epileptogenic effects of decreased GABAminergic transmission, which is also known to be associated with ECT (10).

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Thiazide-Induced Hypercalcemia in a Manic-Depressive Patient

BY G. DAVIS GAMMON, M.D., M.S., AND JOHN P. DOCHERTY, M.D.

A variety of neurological and psychiatric disturbances may be seen secondary to the overt classical disorders of calcium metabolism; these disturbances usually resolve when the primary disorder is corrected (1, 2). Carman and associates (3, 4) have also suggested a pathogenic role for even subtle derangements of calcium homeostasis in the periodic psychoses. Similarly, Alexander and associates (5) have reported a relationship between relatively minor shifts in serum calcium and neuroleptic-induced extrapyramidal symptoms. We describe a case that supports the hypothesis that mild derangements in calcium metabolism may play a crucial role in the pathogenesis of bipolar affective illness. This case illustrates the practical importance of ascertaining and monitoring serum calcium levels in lithium-refractory manic-depressive patients and suggests another potentially deleterious consequence of the combined use of lithium and thiazide diuretics.

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Case Report

Mr. A, a 60-year-old man with a 7-year history of a psychiatric disorder of late onset meeting Research Diagnostic Criteria (6) for manic-depressive illness, was admitted for the second time with lithium intoxication. He was the youngest of eight children. His father, an older brother, and a paternal cousin were reported also to have suffered from manic-depressive illness. Mr. A had been married 34 years. He had fathered and successfully reared two children who were currently happily married. Mr. A had always been moody, with somewhat prominent highs and lows, but he had functioned successfully socially and vocationally until the onset of the illness. The disorder was characterized by profound episodes of depression with apathy, withdrawal, psychomotor retardation, dysphoria, and marked neurovegetative symptoms, which alternated with elated states in which he demonstrated irritability, social gracelessness, poor judgment in financial matters, and antisocial behavior. Initially these episodes were relatively mild, lasting 4–8 weeks, and were separated with interpolar periods of 3 to 12 months.

The onset of Mr. A's illness occurred several years after he started receiving thiazide therapy (500 mg/day of chlorothiazide) for essential hypertension. At first he had an excellent response to lithium carbonate. He experienced nearly complete remission of his symptoms until approximately 3 years before this admission, when he became progressively resistant to lithium therapy. His symptoms recurred despite continued lithium therapy at ever higher doses. The symptomatic intervals were briefer—several weeks to a month—and more severe; before his admission the interpolar periods decreased to intervals as short as several weeks. Mr. A's increasingly severe and frequent symptomatic episodes necessitated his retirement from a job he had held many years, which plunged his domestic life into chaos. His medications just prior to admission were 900 mg/day of lithium carbonate, 50 mg/day of hydrochlorothiazide, and 300 mg/day of hydralazine.

A laboratory examination at admission disclosed that Mr. A had a serum lithium level of 2.3 mEq/liter, and repeated determinations showed his calcium levels were mildly elevated. We undertook a review of his medical history to ascertain the onset of frank hypercalcemia in relation to the emergence of lithium refractoriness, and we investigated his calcium metabolism. Before the onset of his manic-depressive illness and thiazide therapy the only serum calcium level that had been obtained in more than 20 years was 9.1 mg/dl. Two calcium levels obtained subsequently when the patient was becoming lithium refractory were mildly abnormal (10.6 mg/dl taken 2½ years before admission and 10.8 mg/dl taken 1½ years before admission). During this admission Mr. A's serum calcium levels varied between 10.6 mg/dl and 11.1 mg/dl, and his serum inorganic phosphorous levels were between 2.1 mg/dl and 2.8 mg/dl. The serum creatinine was 1.4 mg/dl and the creatinine clearance was 35 ml/minute. A 24-hour urine collection showed that his excretion of calcium was 900 mg and his excretion of phosphorous was 273 mg. His thyroid function studies were normal; serum protein electrophoresis revealed no abnormal calcium-binding protein. Hand X rays showed no changes pathognomonic of hyperparathyroidism. Nephrogenous cAMP was 3.1 nmol/100 ml glomerular filtration rate, and immunoreactive parathyroid hormone-like substance was 0.92 μlEq/ml, with a simultaneous serum calcium of 10.7 mg/dl. These data supported the diagnosis of low-grade hyperparathyroidism that had probably been subclinical until exacerbated by thiazide therapy. The provocative effect of thiazides on disorders of calcium metabolism is well known (7).

Because of the potential role of thiazide therapy in aggravating the disorder, the thiazide was discontinued and propranolol was instituted for the control of Mr. A's hypertension. His serum calcium and phosphorous gradually became normal at 10.1 mg/dl and 3.5 mg/dl, respectively. Nephrogenous cAMP was 0.7 nmol/100 ml glomerular filtration rate.

Coincident with restoration of normal calcium homeostasis, Mr. A recovered from a severe depressive episode and appeared to enter an interpolar period in which dysphoria and a mildly depressed mood were the only psychiatric stigmata. He was discharged over 2 years ago and was prescribed 10 mg of oral propranol q.i.d. for control of his hypertension. Since then, he has been free of the symptoms of manic-depressive illness.

Discussion

This case represents the late onset of bipolar affective illness in a seemingly genetically predisposed individual. The onset of manic-depressive illness late in life usually occurs secondary to some significant metabolic derangement or neurological injury. Low-grade hypercalcemia secondary to mild hyperparathyroidism appears a likely etiological agent in this case. The aberration in calcium metabolism appears to have been aggravated by the thiazide therapy, and normal homeostasis was restored after its discontinuation. Although the patient's apparent recovery may have only reflected a natural vicissitude of the illness, the remission of the illness may have resulted from resolution of the hypercalcemia. This seems likely because the resolution of the symptoms was coincident with rectification of calcium homeostasis. Furthermore, the remission has persisted well beyond any previous interpolar period.

Carman and associates have shown that small but significant increases in total serum calcium immediately precede or coincide with the switch into mania or an excited psychotic state in patients who suffered from periodic psychoses (3). Moreover, the experimental perturbation of serum calcium may significantly alter the frequency and severity of psychotic episodes in patients with such illnesses (4). This case lends support to their observations of the importance of subtle derangements in calcium homeostasis in manic-depressive illness. It also supports the notion that intervention through a variable of possible importance in the switch process may be useful in the treatment of such disorders. Interventions against minor increases in serum calcium might be accomplished in a number of ways, such as the removal of provocative agents like thiazides (7), the use of vitamin D antagonists, the use of beta-blockers (8) to suppress parathyroid hormone output, or the use of synthetic salmon calcitonin

(4) to increase bone sequestration of calcium. Treatments aimed at the switch mechanism are intriguing but largely untried.

Pragmatically, this case emphasizes the possible harmful effects of concomitant thiazide therapy on patients undergoing lithium treatment. At the least, as this case suggests, serum calcium must be carefully monitored in such patients, and the treating psychiatrist should be aware that vulnerability to lithium intoxication and the development of treatment refractoriness may be due to small shifts in the patient's level of serum calcium.

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Preliminary Data on Ethosuximide and the Episodic Dyscontrol Syndrome

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Over the past 2 years investigators at the Institute of Living have been conducting a formal study of the efficacy of ethosuximide in the treatment of patients with episodic dyscontrol syndrome. Detailed descriptions (1) of such patients include acts of violence, drug or alcohol abuse, traffic violations, arrests, job or school failures, suicide attempts, and resistance to conventional psychiatric interventions. As children, these patients often suffered from head trauma, hyperactivity, and learning disabilities. They have a family history of alcoholism, sociopathy, and violence. Their EEGs have nonspecific temporal and frontal lobe abnormalities, 14/second and 6/second positive spiking in the temporal lobes (2), and sharp or slow wave activity in the posterior quadrant. Soft neurological sign examinations are usually positive.

Other investigators (3-5) have postulated a role for the temporal lobe-limbic system in the etiology of violence. *DSM-III* has a new classification for such disorders of impulse control, namely, intermittent explosive disorders. Patients who fit this diagnosis impulsively and precipitously exhibit violence under little or no stress toward themselves, other persons, or property. Increasing tension often precedes the dyscontrol act. The explosive episodes are repetitive, short in duration (minutes to hours), and end abruptly. Afterward these patients experience partial amnesia, guilt, and relief of tension.

The focal point of this study is the possible relationship between this syndrome and an underlying disorder of the CNS. The hypothesis is that these patients display acts of violence that are triggered by complex partial ictal events resulting from recurrent excessive disorderly discharges from the temporal lobes to the limbic system. Ethosuximide, an anticonvulsant for petit mal epilepsy, may inhibit such discharges. This medication also is associated with positive changes in school and social behavior among children with learning disabilities and atypical EEGs (6). Ethosuximide is a particularly attractive medication because of its safety, known therapeutic blood level, and few side effects.

Method

In the ongoing controlled, double-blind, crossover study each patient is assigned to active medication for

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one or two 8-week periods and to a placebo for one or two 8-week periods, for a total of 24 weeks. An investigator who is not blind to each patient's medication schedule monitors the patient's ethosuximide blood level. The instruments used by investigators who are blind to the study to evaluate the patient's clinical change are the Minnesota Multiphasic Personality Inventory (MMPI), the Minnesota-Hartford Personality Assay (MHPA), computerized daily nursing notes (7), nursing special behavior reports, standard clinical and computer analyzed EEGs (8, 9), and soft neurological sign examinations.

To date 12 patients have been involved in the current research study; 7 have completed the full study protocol. Because of the serious psychopathology of the study population the number of research candidates has been limited, and the study is progressing more slowly than originally anticipated. However, the preliminary data from this study, together with the retrospective examination of clinical observations made on 70 patients exhibiting episodic dyscontrol treated with ethosuximide before this study, tend to support our hypothesis as stated above. Although sufficient data to warrant statistical analysis have not yet been accumulated, observations to date are encouraging and merit this preliminary report and, we think, the continuation of this study. Below we present an illustrative case history.

Case Report

Susan was 14 years old with a diagnosis of episodic dyscontrol syndrome characterized by self-mutilation, assaultiveness, property damage, and multiple suicide attempts. A standard EEG taken at the time of her admission showed slow wave activity in the posterior quadrant and 6–10 Hz positive spiking over the right temporal lobe during sleep. Her admission computer-analyzed EEG had marked slowing over all areas, especially in the temporal leads. As she became drowsy, very high-voltage slow activity in the 4–8 Hz range appeared.

The medication schedule for Susan was ethosuximide, placebo, and then ethosuximide. During the first period she had four dyscontrol episodes, including two of self-mutilation, before a therapeutic ethosuximide blood level was reached. When the medication was at a therapeutic blood level (1250 mg/day, 73.6 mg/liter), she had two episodes of verbal threats. Susan did not require thioridazine for dyscontrol during this period. In the second or placebo period, Susan had 11 dyscontrol episodes requiring 34 doses of thioridazine as necessary. During the third period Susan refused nine doses of ethosuximide, and she had eight dyscontrol episodes when the ethosuximide fell below the therapeutic blood level. She required seven doses of thioridazine during this period. However, when the ethosuximide was restored to the therapeutic blood level, Susan had no dyscontrol episodes and further thioridazine was not needed. Investigators who were blind to the drug treatment program made all the observations of Susan.

The MHPA results from both of Susan's active medication periods showed that she manifested much less hostility, self-criticism, defective superego integration, and paranoid thinking than during the placebo period. The computerized daily nursing notes reflected that Susan had less antisocial behavior and more acceptable behavior during her active periods. The MMPI showed substantially lower T scores during the active medication periods in all scales except the Masculine-Feminine and Ego Strength scales. Her neurological soft signs showed no significant change. A second computer analyzed EEG, recorded while she was taking active medication at a therapeutic blood level, showed a striking increase in alpha activity and a disappearance of the slow wave activity that was present when she was admitted to the study. In addition, there was a notable increase in the amount of synchronization occurring in all leads, especially the occipitals.

Discussion

The preliminary data tend to support the hypothesis that ethosuximide, when used in the treatment of patients exhibiting episodic dyscontrol, has the potential for producing an excellent clinical response with few side effects. Many of these patients suffering from episodic dyscontrol form a subcategory of the borderline syndrome. They are differentiated from other borderline states by the presence of dyscontrol episodes, abnormal EEGs, and a history suggestive of organicity. Our present study is being pursued to 1) verify the initial findings, 2) establish operational criteria for the *DSM-III* classification of intermittent explosive disorder, 3) validate the diagnostic role of the computer analyzed EEG in this syndrome, and 4) develop a medication prediction profile. We are also collecting data to identify the relationships between variables differentiating subcategories of the borderline syndrome, including episodic dyscontrol.

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Hypomania and Tricyclic Antidepressant Choice

BY THOMAS E. STEELE, M.D., AND C. INGA TAYLOR, M.D.

Evidence indicates that unipolar depression is clinically, biochemically, and therapeutically heterogeneous (1–4). Because some patients are more likely to respond to imipramine and others to amitriptyline, with little or no overlap between the two groups in most studies (1–3), the practitioner needs methods that require neither trial-and-error nor sophisticated biochemical assays to select the appropriate tricyclic antidepressant.

Recent work (5), as well as Kraepelin's earlier observations, suggests that full-blown manic-depressive illness is the extreme on a continuum that includes varying degrees of cyclothymia. Others (4) note that some unipolar depressives who to date have not experienced mood elevations resemble depressives with frank bipolar histories. Thus, the heterogeneous unipolar group probably contains two more homogeneous groups: the "pseudounipolars" (6), who represent milder degrees of cyclothymia and who are at greater risk to become frankly manic or hypomanic in the future, and the "true" unipolars, who have not and will not experience mood elevations.

Although contrary results have been reported, the preponderance of evidence suggests that depressed patients with low urinary 3-methoxy-4-hydroxyphenyl glycol (MHPG) are imipramine responders and that patients with normal or elevated MHPG are amitriptyline responders (1–3). Furthermore, depressed manic-depressives tend to have low MHPG (2, 7), although again this is not conclusive (8). Given this evidence and the breadth of the cyclothymic spectrum—from mild mood swings to frank bipolar illness—we reasoned that imipramine would be the tricyclic antidepressant of choice for depressed patients whose histories suggest periods of hypomania (whether in obvious or attenuated form). Conversely, amitriptyline would be the drug of choice for patients with histories devoid of subtle or obvious hypomanic behaviors. We report here an open pilot investigation designed to determine whether a history of hypomania can aid in selecting the appropriate tricyclic antidepressant.

Method

Our subjects included 55 patients who were admitted consecutively to a Veterans Administration psychiatric outpatient clinic and who met the following criteria: primary affective disorder by the criteria of Feighner and associates (9), dysphoria and neurovegetative disturbances severe enough to warrant antidepressant medication, and no history of mania. Thus, the sample (54 men and 1 woman; age range, 27–69 years) represented patients with unipolar depressions of moderate and marked severity. Because these patients were currently depressed, the assessment of each patient's history for hypomanic symptoms had to be retrospective. We chose the following three types of systems as indicative of a cyclothymic diathesis: 1) a period of increased energy and decreased need for sleep, with or without an increased sense of well-being; 2) behaviors such as impulsive traveling and spending of money, or making excessive long-distance telephone calls; and 3) episodes of irritability that were not concomitants of a depressive episode. Patients with a record of one or more of these three types of hypomanic symptoms (N=33) were treated with imipramine and patients without any of these symptoms (N=22) were treated with amitriptyline; final doses of both drugs ranged from 100–350 mg/day and the modal dose range was 150–200 mg/day. Upper dose limits were determined by therapeutic response or unacceptable side effects. Although a planned drug crossover was not performed during the study period, 19 of the 55 patients had previously received adequate but unsuccessful treatment with the other tricyclic antidepressant.

We assessed each patient's improvement on a four-point global change-of-state scale emphasizing affective and neurovegetative symptoms; only patients who showed moderate or marked improvement were considered positive responders to the drug. We used two-tailed t tests to analyze the data.

Results

Of the 33 patients who had a history of one or more hypomanic symptoms, 29 responded positively to imipramine; of the 22 patients who were negative for all three hypomanic symptoms, 20 responded positively to amitriptyline. Thus, the combined success rate was 89%.

Among the 49 patients who responded positively to imipramine or amitriptyline, the mean number (±SE)

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of positive responses to the three hypomanic history items was 2.2±0.1 for the imipramine group (N=29) and zero for the positive responders to amitriptyline (N=20) ($p<.001$).

The imipramine-responsive group was significantly younger than the amitriptyline-responsive group ($p<.001$); however, the age ranges for the two groups showed broad overlap. Current age (mean±SE) for the former was 42.3±1.9 years (range=27-61 years) and for the latter it was 57.4±1.4 years (range=45-69 years). The age of onset of the patients' first depressive episode for the imipramine group was 33.2±1.9 years (range=20-54 years); for the amitriptyline group it was 49.6±2.2 years (range=28-65 years) ($p<.001$).

There were no significant differences in agitation or retardation between the two groups. Of the 29 patients who responded to imipramine, 5 were agitated and 14 were retarded; of those patients who responded to amitriptyline, 4 were agitated and 12 were retarded.

Do patients who respond positively to one of these drugs represent spontaneous remissions, or are they patients who might have responded equally well to either drug? These two possibilities seem unlikely because 13 of the 29 patients who responded positively to imipramine had previously had adequate but unsuccessful treatment with amitriptyline and 6 of the 20 patients who responded positively to amitriptyline had previously had adequate but unsuccessful treatment with imipramine. Of the 6 patients who did not respond (4 of whom had one or more hypomanic symptoms), 1 patient did not respond to imipramine, 2 patients did not respond to amitriptyline, and 3 patients did not respond to either drug.

Discussion

This preliminary study suggests that the presence or absence of a history of hypomanic symptoms may aid us in choosing the appropriate tricyclic antidepressant. Many apparently unipolar depressed patients probably represent a forme fruste of bipolar disorder, given the broad-spectrum nature of that illness and the ease of overlooking the more subtle evidence of mood fluctuations (5). A failure to appreciate this heterogeneity may explain the lower response rates generally observed when a single tricyclic antidepressant is given to unselected unipolar patients. Because patients with unipolar depression represent a chemically heterogeneous group (1-3, 7), a differential drug response is to be expected. If one accepts the evidence that bipolar illness is associated with low MHPG and imipramine responsiveness (2, 7), the younger age of our patients who responded to imipramine is to be expected. Bipolar illness usually begins at a younger age (10), and one study (11) found increasing age to be related to amitriptyline responsiveness. Age alone, however, is not a sufficient guideline: although the mean ages were significantly different for the two groups, the age ranges showed considerable overlap. This was true not only for current age, which is an artifact of the moment, but also for age at onset of first depression, which may reflect more enduring biological characteristics. A history of subtle or obvious hypomania, if carefully obtained, also represents an enduring characteristic that is not influenced by transient changes in state. Given the controversial reports regarding MHPG and a patient's responsiveness to imipramine it would be most useful if controlled investigations would validate our suggestion that the results of a carefully obtained clinical interview can point the therapist to the appropriate tricyclic antidepressant.

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Effects of Thioridazine on the Cognitive Functioning of a Hypotonic Schizophrenic Boy

BY MOHAMED INAYATULLA, M.A., AND SHEILA CANTOR, M.D.

The uneven performance of schizophrenic children on tests of cognitive functioning has been described previously (1-2). Less is known regarding the effects of psychotropic medications upon cognitive functioning in schizophrenic children. Phenothiazines bind to cholinergic and dopaminergic receptors. Because there is evidence (3, 4) that both these neurotransmitter systems are involved in cognitive functioning, it would be anticipated that cognitive functioning would be affected by the chronic ingestion of these agents. Nonetheless, medication is prescribed in the hope that the benefits obtained from decreasing psychotic symptomatology will outweigh the deficits inherent in the adverse side effects.

Case Report

An 11-year-old hypotonic schizophrenic boy was referred to one of us (S.C.) for assessing the appropriateness of including him in a day hospital program. He is the subject of our study. The boy had been diagnosed as being "on the autistic continuum" at age 3 years, 2 months, and he had been observed by physicians for 8½ years. At no time did he have evidence of neurological deterioration or focal neurological signs, despite an EEG at 10 years, 2 months that revealed "excessive slow wave activity in the left posterior temporo-parietal areas." The child, in fact, resembled in every way the hypotonic schizophrenic children described elsewhere in detail (5 and unpublished data being prepared by Pearce and associates), and he met *DSM-III* criteria for schizophrenia (manifesting illogicality and incoherence in the presence of flat affect).

One of us (M.I.) administered the WISC-R to the boy on three separate occasions, and table 1 describes the results of this testing.

The first test was administered when the child was 10 years, 2 months old. He had received low doses of chlorpromazine beginning at age 3½, methylphenidate for a few months between the ages of 3½ and 4½ years (administered erratically by his parents), and constant low doses of thioridazine beginning at age 9. The psychotropic medications had been prescribed for hyperactivity, excessive anxiety, easy distractibility, and poor frustration tolerance and temper tantrums. His performance was superior on the block design portion. From the time he was 7 years old, it was surmised that he might have a superior ability in block design, as well as a markedly impaired verbal capacity.

The second test was administered when the boy was 11 years, 6 months old. At that time he had been taking 200 mg/day of thioridazine for 16 months. We began a gradual decrease in the psychotropic medication, and weekly psychotherapy sessions were initiated (with S.C.). While taking thioridazine, he had mildly decreased muscle tone and muscle power. His most striking symptoms were poverty of speech, poverty of content of speech, very flat affect with expressionless facies, and anxiety that escalated easily and was expressed by withdrawal and tearfulness. He had no evidence of delusional thinking or hallucinatory activity. His superior performance on the block design portion of the test was maintained (but did not improve), and he demonstrated superior ability on the object assembly subtest as well.

The third test was administered when the child was 11 years, 10 months old. He had not taken any medication for 3 months; his medication had been withdrawn with little difficulty over an 8-week period. Specifically, he had no sleep disturbance or dramatic exacerbation of psychotic symptoms. Clinically, however, he exhibited many changes. His affect remained severely restricted, but his facies were no longer expressionless, and he had a minimal affective range; episodes of giggling were common and at times obviously involuntary; he had a marked increase in grimacing; his speech had increased dramatically but with much evidence of disorganized thinking (illogicality, derailment, incoherence, and occasional clang associations); hyperacusis was noted; and bodily delusions appeared and visual hallucinations were suspected. Significant changes were also noted in his motor ability. Specifically, his muscle tone and power were now moderately decreased; his reflexes were difficult to obtain, particularly in the upper limbs; and he was unable to sit unsupported for prolonged periods of time. He now expressed increased anxiety by an increase in grimacing and mannerisms rather than by withdrawal and tearfulness. He related much more warmly to his therapist, family, and teachers. His cognitive rigidity decreased, and he was much more willing to attempt new tasks and risk failure. He reported that he felt "good." He performed significantly better on the block design and similarities subtests than in the first testing, which confirmed our previous impressions that he had an exceptional capacity in some areas of functioning.

Discussion

A child's developing years are critical in achieving normal cognitive development (6), and psychosis in childhood undoubtedly interferes with normal cognitive development. Therefore, it is essential that those who care for psychotic children develop means to improve the cognitive development of such children and avoid therapeutic interventions that may further inhibit such development.

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TABLE 1
WISC-R Scores for a Hypotonic Schizophrenic Boy

Subtest	Boy's Age at Time of Testing		
	10 years, 2 months[a]	11 years, 6 months[b]	11 years, 10 months[c]
Verbal			
Information	3	3	3
Similarities		1	5
Arithmetic		6	6
Vocabulary	4	1	1
Comprehension		1	1
Digit span		6	5
Performance			
Picture completion	10	11	13
Picture arrangement	6	6	6
Block design	14	14	19
Object assembly	8	13	14
Coding	1	3	2
Verbal		52	58
Performance	85	95	105
Full scale		72	79

[a]Test administered during initial psychiatric assessment; boy had received various psychotropic drugs beginning at age 3½.
[b]Test administered after boy had received 200 mg/day of thioridazine for 16 months.
[c]Test administered 3 months after all medication had been withdrawn.

There is evidence that many psychotic children are visually unimpaired (unpublished data being prepared by Pearce and associates and Dueck and associates) and, in fact, depend upon their visual apparatus to compensate for deficits in other systems (unpublished data being prepared by Ireland and Cantor). It seems likely that by helping such children develop their strengths (as reflected by their performance on the block design and object assembly subtests of the WISC-R) therapists will enhance the likelihood that these children will be able to function independently during their adult years. Therapeutic agents that interfere with the ability of these children to use these strengths should be avoided.

Despite the fact that the boy had an increase in thought disorder after his psychotropic medication was discontinued, he is functioning better without medication than he did with medication. His family, teachers, and therapist agree that when he is not taking medication he relates better, feels better, and has improved educability. Although his improvement in relating and subjective well-being could also reflect the benefits of psychotherapy, it is doubtful that his changes in cognitive functioning (represented by high scores on WISC-R) could be attributed to the minimal psychotherapeutic intervention he received—once a week for 3 months.

Controlled studies of the effects of psychotropic medication on cognitive functioning are needed, particularly in psychotic and functionally retarded children. There is evidence that with careful and intensive therapeutic intervention childhood psychosis need not have a bleak prognosis (7). However, the iatrogenic inhibition of cognitive functioning should be avoided.

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Seizures During Tricyclic Therapy

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It was noted in several reports (1-5) of early clinical trials of imipramine that seizures occurred as an infrequent side effect in up to 4% of the patients studied (4). Betts and associates (6) and Dallos and Heathfield (7) subsequently analyzed the histories of 16 patients who had seizures during antidepressant therapy (13 patients received amitriptyline, 2 patients received imipramine, and 1 patient received protriptyline). Thirteen of these patients were noted to be predisposed to the development of seizures by one or more of the following factors: a personal history of seizures, a family history of convulsive disorder (associated with epileptiform EEGs in 3 of these 4 patients), cerebrovascular disease, alcoholism, barbiturate withdrawal, prior ECTs, intramuscular administration of amitriptyline, and a combination of amitriptyline with chlorpromazine. Betts and associates (6) recommended that all patients taking such drugs be cautioned against driving for a few weeks after starting medication or after increasing the dosage and that patients with a presumed low convulsive threshold be closely supervised (preferably in a hospital) while taking tricyclics. As an alternative, Dallos and Heathfield (7) suggested that when risk factors such as the above are present, anticonvulsant medication should be administered simultaneously with antidepressants.

More recently Toone and Fenton (8) studied 41 patients who had seizures that were thought to be due to treatment with psychotropic drugs or withdrawal from sedatives; 10 patients had received amitriptyline and 7 had received imipramine. Patients with epilepsy or suspected organic brain disease were excluded from the study. Toone and Fenton were unable to demonstrate the importance of either a family history of convulsive disorder or a history of ECTs, but they found that the occurrence of seizures was associated with a history of postnatal brain damage and with an increased ingestion of psychotropic drugs the week before a seizure.

Although a number of factors are thus presumed to predispose patients to seizures during tricyclic treatment, to our knowledge no systematic study of these factors and their association with seizures has been conducted among a large number of patients undergoing antidepressant therapy. We made such a study using 400 patient records from the Iowa Psychiatric Hospital; our findings are reported below.

Method

The consecutive records of patients 15 years or older who were treated at Iowa Psychiatric Hospital since 1959 and who had an inpatient discharge or an outpatient summary diagnosis of depression were reviewed. We included patients who had received imipramine or amitriptyline for at least 1 week (or less, if they had had seizures in the first week of treatment) and divided them into two groups, those who received imipramine and those who received amitriptyline. Those factors presumed to predispose patients to seizures during tricyclic therapy were extracted from the patients' records (see table 1). Data were collected regarding each patient's most recent course of tricyclic medication. A total of 400 records was analyzed.

Results

The imipramine and amitriptyline groups were very similar with respect to age, sex, patient status, maximum dose of tricyclic prescribed, and length of follow-up available in the records (from first course of antidepressant medication at this hospital to last clinic visit). There were 353 (88%) inpatients and 47 (12%) outpatients, ranging in age from 15 to 84 years (mean± SD=50.6±14.6); 145 (36%) patients were men, and 255 (64%) patients were women. The patients were prescribed maximum doses of tricyclic medication ranging from 40 to 300 mg/day, with an average maximum dose of 150.9±50.3 mg/day. Length of follow-up time varied from 1 to 221 months, with a mean of 28.5±42.7 months.

Those factors presumed to predispose patients to seizures during tricyclic therapy are summarized in table 1. Five patients had seizures during childhood only, three in association with febrile episodes. Another patient had seizures immediately after a concussion, 2 patients had seizures during periods of withdrawal from sedative drugs, 1 patient had a seizure after an insulin overdose, 1 patient had seizures during eclampsia of pregnancy, and 2 had seizures at the time of strokes. Only 2 patients had histories of idiopathic grand mal convulsive disorder in adulthood.

Four patients out of the 400 had seizures during tricyclic treatment. All 4 of the patients were taking amitriptyline at the time. Two of these patients were the epileptic patients noted above and 2 were women with-

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TABLE 1
Number and Histories of Patients Who Had Seizures During Tricyclic Antidepressant Treatment

Factor Predisposing Patient to Seizure	Patients Treated with Tricyclics (N=400)		
	Amitriptyline (N=200)	Imipramine (N=200)	Patients Who Had Seizures (N=4)[a]
Convulsive disorder			
In patient	6	8	2
In family	8	2	4
Abnormal EEG	26	14	3
Postnatal brain damage	1	2	0
Head trauma	12	14	1
Cerebrovascular disease	4	5	1
Prior ECT	89	85	1
ECT during index drug treatment[b]	23	46	0
Phenothiazine during index drug treatment	40	29	1

[a]All patients who had seizures were in the amitriptyline group.
[b]Difference between drug groups is significant, $p<.01$ (chi-square with Yates' correction).

out a personal history of convulsions or epilepsy but with strong family histories of epilepsy.

One of the epileptic patients, a 58-year-old man, had a history of intermittent convulsions and abnormal (nonfocal, nonspecific) EEGs despite maintenance therapy with phenytoin and phenobarbital. He had a seizure after 2 weeks of taking amitriptyline; the seizure occurred the day after amitriptyline was increased from 25 mg/day to 50 mg/day. The amitriptyline treatment was then reduced to 25 mg/day, but he had another seizure 1 week later. He was shortly thereafter discharged, and no follow-up information was available to us. The other epileptic patient, a 53-year-old man, had not had a seizure in 9 months before his hospitalization and had had a normal EEG before starting amitriptyline. He had a seizure after 6 weeks of amitriptyline treatment—3 weeks after a reduction in dosage to 50 mg/day. The amitriptyline treatment was stopped and phenytoin treatment started, and his seizures were satisfactorily controlled for the next 8 years.

One of the women, age 40, suffered a generalized seizure on the third day of treatment while taking 150 mg/day of amitriptyline in combination with 6 mg/day of trifluoperazine. The latter drug was stopped, and she had no further seizures during 4 more weeks in the hospital, although a subsequent EEG revealed nonfocal sharp and spike activity. The second woman, age 50, had a history of chronic minor head trauma and paroxysmal bursts of generalized spike and slow activity on an EEG at the time her amitriptyline treatment was begun. She had 2 seizures after 2 weeks of drug therapy—1 week after reaching 200 mg/day and 1 day after the discontinuation of Fiorinal (2 tablets every 4 hours p.r.n. for headache). Phenytoin was added to her regimen, and she had no further seizures during 2 weeks of additional hospitalization.

Discussion

Two of the four patients who had seizures during tricyclic treatment were known epileptic patients. Their seizures may have been unrelated to drug treatment. The other two patients who had seizures also had strong family histories of epilepsy and abnormal, epileptiform EEGs. All of these factors have been associated with tricyclic-induced seizures in previous studies (1, 3, 6, 7). These factors probably explain the difference in the number of seizures found between the amitriptyline and the imipramine drug groups.

This study suggests that the overall incidence of seizures during tricyclic treatment is approximately 1% and among nonepileptic patients is perhaps ½%, which is an estimate consistent with the impression of Kiloh and associates (9).

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Reversal of Dexamethasone Nonsuppression with Sleep Deprivation in Primary Depression

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Under normal conditions, the administration of 1 mg of dexamethasone results in the suppression of plasma cortisol for the next 24 hours (1). This reflects the responsiveness of the hypothalamic-pituitary-adrenal axis to exogenously administered steroid. About 40% of depressed patients show abnormal resistance to dexamethasone suppression, although individuals with other psychiatric disorders have almost uniformly suppressed normally with dexamethasone (2, 3). Several investigations have considered abnormal dexamethasone suppression as evidence for disruption in the circadian rhythm of the hypothalamic-pituitary-adrenal axis (4, 5).

Sleep deprivation has been shown by numerous studies (6) to exert a transient antidepressant effect in up to two-thirds of depressed patients, particularly in patients with severe "endogenous" depression. Sleep deprivation is mainly a research tool in the United States, but it is widely used in Europe as a therapeutic modality (7) and as a predictor of response to tricyclic antidepressants (7). The mechanism for the antidepressant action of sleep deprivation remains unknown, but it has been postulated that it is related to correction of disturbed diurnal rhythm (8), which may be part of the pathophysiology of primary depression.

Taken together, the above findings suggest that dexamethasone nonsuppression in depression and the antidepressant effects of sleep deprivation may be related to a common physiological mechanism. This possibility led us to study the effects of repeated sleep deprivation treatments on the hypothalamic-pituitary-adrenal axis in a patient with endogenous depression.

Case Report

Mr. A was a 47-year-old divorced farmer, who was brought to the hospital by his children because of his inability to function. Retardation was a prominent feature of his clinical condition. He would stand in one spot, bow his head, and hardly move or interact at all. He had initial and terminal insomnia, decreased energy, increased weight loss, and impaired concentration and memory. He felt he did not deserve to be a veteran, and that whatever he did would end up in failure. He no longer cared about anything, and although he was not suicidal, he felt that it was "all over," he was going to die, and he should be shot.

Mr. A's psychiatric history included depressive episodes separated by complete remission at ages 21, 27, 29, 31, and 46; he had no history of mania or hypomania. His symptoms usually included psychomotor retardation, hypersomnia, and thoughts that all his livestock and children were sick and that he was going to die. At one time, he also reported the voices of his parents telling him he was "done for." His medical history included slight hypertension, gout, obesity, and adult onset diabetes 1 year before this admission. Mr. A had no family history of psychiatric illness, except for a sister who was hospitalized for an unknown nervous condition.

Mr. A consented to participate in a study comparing a course of overnight sleep deprivation (given three times a week for 2 weeks) to a course of ECT. Dexamethasone suppression tests were done at baseline, then after every two sleep deprivation treatments, and after the second, fourth, and seventh ECT treatments. At 11:00 p.m. 1 mg of dexamethasone was given orally and at 8:00 a.m. the next morning blood was drawn to determine Mr. A's cortisol level (radioimmunoassay). The first three ECT treatments were unilateral and the last five were bilateral. The Hamilton Depression Rating Scale was done at baseline and every week thereafter. Mr. A also completed a self-rating (visual analogue) mood scale every day.[1]

The results are summarized in table 1. Mr. A's baseline 8:00 a.m. cortisol level was 16.2 μg/dl, and his baseline postdexamethasone 8:00 a.m. cortisol level was 7.1 μg/dl. (At our center nonsuppression is defined as a plasma cortisol level exceeding 5.0 μg/dl at 8:00 a.m. 9 hours after 1 mg of dexamethasone is ingested.)

After the first two sleep deprivation treatments, Mr. A showed relatively mild clinical improvement with increased response to questions, better eye contact, and increased mobility and participation in ward activities. His daily self-ratings and the nurses' notes also reflected this improvement. Although he continued to look depressed and to express self-deprecatory ideas, his dexamethasone nonsuppression reverted to normal suppression (1.4 μg/dl) the day after the first two nights of sleep deprivation.

Mr. A showed less clinical improvement after the third sleep deprivation treatment and borderline dexamethasone suppression (4.8 μg/dl) 3 days later. He seemed to worsen after the fourth session of sleep deprivation and reverted to nonsuppression with dexamethasone (11.3 μg/dl) 2 days afterward. Although he continued to worsen after the fifth and sixth sessions of sleep deprivation his postdexamethasone cortisol level 1 day after the sixth session was barely nonsuppressed (5.2 μg/dl).

After the first two unilateral ECT treatments, Mr. A showed the highest degree of dexamethasone nonsuppres-

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[1]The patient's daily self-ratings on the visual analogue are available on request from Dr. Nasrallah.

TABLE 1
Postdexamethasone Cortisol Levels in a Depressed Patient Before, During, and After Sleep Deprivation and ECT

Hospital Day	Treatment	Postdexamethasone Cortisol Level (μg/dl)[a]	Hamilton Score[b]
2		16.2[c]	
3		7.3	
4			36
5	Sleep deprivation		
8	Sleep deprivation		
9		1.4	
10	Sleep deprivation		28
15	Sleep deprivation	4.8	
17			31
18		11.3	
19	Sleep deprivation		
21	Sleep deprivation		
23	Unilateral ECT	5.2	
25	Unilateral ECT		21
27		20.7	
28	Unilateral ECT		
32	Bilateral ECT		
34		2.1	
35	Bilateral ECT		
37	Bilateral ECT		8
39	Bilateral ECT		
41		1.0	
42	Bilateral ECT		
44			5

[a]All samples taken at 8:00 a.m.
[b]Hamilton Depression Rating Scale scores reflect resolution of the depression.
[c]Patient did not receive dexamethasone the night before.

sion (20.7 μg/dl); he was clinically unimproved and still very depressed. However, he reverted to normal suppression (2.1 μg/dl) after two more ECTs (one unilateral and one bilateral), which also coincided with the onset of significant improvement in his depression as reflected by the Hamilton Depression Rating Scale.

Mr. A continued to improve considerably and was fully recovered after the ECT course. His postdexamethasone cortisol level was normally suppressed (1.0 μg/dl) at that time.

Discussion

Our findings suggest that sleep deprivation treatment produced mild, transient antidepressant effects and a concomitant correction of dexamethasone nonsuppression in a patient with primary or endogenous depression. To our knowledge this is the first demonstration of the restoration of normal dexamethasone suppression with sleep deprivation.

It is noteworthy that in Mr. A, dexamethasone suppression was achieved without the onset of significant clinical improvement in his depressive state. It has been shown that dexamethasone suppression is restored only after the clinical depression responds to treatment (2). Thus, it may be that although clinical improvement of endogenous depression is usually associated with the correction of dexamethasone nonsuppression, the reverse may not necessarily apply.

Sleep deprivation is postulated to exert an antidepressant effect by correcting a disturbance of diurnal rhythm (8) in depression but other factors may also be involved. Because cortisol secretion in depression is also related to a diurnal rhythm disturbance (4), it is possible to suspect that the correction of dexamethasone nonsuppression with sleep deprivation in our patient may be associated with the normalization of a disturbed diurnal rhythm (without a simultaneous significant improvement in the depressive symptoms as measured by the Hamilton Depression Rating Scale). The corrective effect of sleep deprivation on dexamethasone nonsuppression was, however, not sustained beyond the fourth sleep deprivation treatment.

With ECT, unlike sleep deprivation, the onset of significant improvement in Mr. A's depression was temporally associated with the correction of dexamethasone nonsuppression, as was reported by others (9).

Our finding that sleep deprivation can affect the hypothalamic-pituitary-adrenal axis and reverse dexamethasone nonsuppression of cortisol without a concomitant and significant improvement in depression (as with tricyclic antidepressant and ECT) may offer an important and useful link in understanding the neuroendocrine correlates of the antidepressant response.

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Paranoid Schizophrenia and Platelet MAO Activity

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Monoamine oxidase (MAO), a crucial enzyme in the degradation of biogenic amines, is implicated in the etiology of several psychiatric disorders. Although most reports show reduced platelet MAO activity in chronic schizophrenia, the extent of reduction varies greatly across studies. Diagnostic heterogeneity may explain the variability in research results (1). Illness subtyping may also prove useful in dissecting biological forms of schizophrenia (2). Specifically, some investigators (1, 2) have claimed that paranoid schizophrenia is biologically different from other schizophrenias; their claims are based on the fact that abnormally low platelet MAO levels are found in chronic schizophrenic patients who are diagnosed as paranoid or as having secondary paranoid features. However, other investigators (3–5) have failed to replicate these findings.

The relationship of MAO activity to schizophrenic subcategories has not been studied extensively and the data reported have not been based on uniform diagnostic and assay procedures. As a result, we decided to reexamine this issue by correlating several diagnostic scales of the paranoid dimension with platelet MAO activity in a new sample of schizophrenic patients.

Method

We chose 24 men with chronic schizophrenia and 22 age- and sex-matched control subjects. Diagnostic assessments were based on the Schedule for Affective Disorders and Schizophrenia (SADS) (6) and the Research Diagnostic Criteria (RDC) (6). The patients represented consecutive admissions to a clinical research ward at New York State Psychiatric Institute. They had never been treated with tricyclics, MAO inhibitors, or lithium. Baseline blood samples were obtained from all patients after a 2-week washout period. Drug-free control subjects were recruited from local college students after an interview to exclude those with psychopathology. We also excluded subjects who had a history of alcoholism, drug abuse, and neurological or medical disorders. The schizophrenic patients were classified as paranoid or nonparanoid using the subtyping criteria of RDC (N=12 and N=12, respectively) and the criteria of Tsuang and Winokur (7) (N=10 and N=14, respectively). The paranoid dimension was further quantified by the MMPI paranoia scale and a 7-point severity scale of persecutory delusions and hallucinations derived from the SADS.

The method of Murphy and associates (8) was used for measuring MAO activity. We obtained 18-ml blood samples by venipuncture and collected them in Becton-Dickenson #4798 tubes containing 2 ml of acid-citrate-dextrose solution. Platelet-rich plasma was prepared by a 10-minute centrifugation at 1000 rpm. Aliquots of platelet-rich plasma were removed for platelet counting on a Coulter Counter and for MAO assay using ^{14}C-benzylamine as substrate. MAO was assayed using duplicate samples. Samples were collected concurrently from all subjects and determined at random. Product formation was measured by liquid scintillation of spectrometry. All determinations were made by investigators who were blind to the patient's diagnosis. We used two-tailed t tests to analyze the data.

Results

Platelet MAO activity was significantly lower in the chronic schizophrenic patients than in the control subjects (mean±SE=7.16±0.72 and 9.17±0.57 nmol/10^8 platelets per hour, respectively; $p<.05$). Using the subtyping criteria of the RDC and Tsuang and Winokur, we found no significant difference between the paranoid and nonparanoid subtypes. We also found an inverse correlation between MAO activity and the MMPI paranoia scale ($r=-.38$), as well as the SADS-based measure of persecutory delusions ($r=-.31$) and hallucinations ($r=-.30$), although neither result reached statistical significance.

Discussion

Because patients with schizophrenia are likely to be genetically and biologically heterogeneous, reliable means of differentiating among illness types are strongly needed. Chronic paranoid schizophrenic patients have recently been found to differ from nonparanoid schizophrenic patients in MAO activity (1, 2). It is also noteworthy that a significant inverse correlation between the paranoia scale and platelet MAO activity has been noted in normal subjects with elevated MMPI profiles (2).

The present results confirm the observation that decreased MAO activity occurs in a subset of patients

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with chronic schizophrenia. However, the paranoid-nonparanoid dichotomy, as determined by RDC and the criteria of Tsuang and Winokur, fails to correlate with MAO activity. These findings are consistent with the data of Berger and associates (3), Van Valkenburg and Crowe (4), and Baldessarini (5), but they are at variance with several other reports (1, 2). The apparent discrepancy among the various reports poses problems for the use of subtype diagnosis as a predictor of MAO activity in schizophrenia. Although conflicting results have previously been attributed to nonuniformity of descriptive criteria, these variables have been carefully considered in the present study. It is worth noting, however, that the paranoid dimension, as measured by two separate scales of psychopathology, is inversely correlated with MAO activity even though the results fall short of statistical significance. This may indicate that platelet MAO activity may have some predictive power with respect to the paranoid profile, but that extrapolation from this observation to explicit subtype diagnosis is premature.

More studies are needed to clarify the relationship between clinical description and platelet MAO activity. As proposed elsewhere (9), however, descriptive phenomenology will probably be of limited use in validating biological subdivisions of schizophrenia. Other predictors, such as the distinction between genetic and nongenetic types of the illness, may prove more useful in defining homogeneous subsets of schizophrenic patients who have differing biochemical characteristics (10).

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Manic Psychosis Following Rapid Withdrawal from Baclofen

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Baclofen, which is structurally related to GABA and presumably acts as a GABA agonist, has been shown to inhibit spinal reflexes and has been used successfully in the treatment of various spastic disorders. More recently, studies (1) presenting evidence that baclofen has an inhibitory effect on mesolimbic dopamine neurons have suggested that baclofen might be of benefit in the treatment of schizophrenia; these studies have led to clinical trials with schizophrenic patients. Adverse reactions to abrupt cessation of baclofen have included auditory and visual hallucinations, depersonalization, and paranoia (2). Although schizophreniform psychoses lasting up to several days have been reported, as well as brief periods of euphoria, to our knowledge there has been no instance of a clinical course consistent with a manic psychosis described in the literature. We describe a patient with no known history of affective illness who developed a manic psychosis after abrupt cessation of baclofen. We present this case as evidence for the primary role of dopaminergic mechanisms in mania and the switch process, and we discuss the implied role of substance P in dopamine release and dopamine receptor sensitivity.

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Case Report

Mr. A, a 71-year-old man, was brought to the Vanderbilt University emergency room by his family. He had a 3-day history of sleeplessness, progressive agitation, and "talking out of his head."

Mr. A had been evaluated by two of us 6 months previously when he complained of progressive weakness and paresthesias in his legs. In our physical examination we found a mild degree of weakness and spasticity in his right leg and mild hyperreflexia in all extremities. Significantly, his mental status was normal, and he had no history of psychiatric illness. A CT scan of the head was normal. A myelogram revealed significant boney degenerative changes in the cervical spine but no surgically correctable defect. We made a diagnosis of cervical myelopathy and prescribed 15 mg/day of baclofen in three divided doses.

Six months before this admission, Mr. A's dose of baclofen was gradually adjusted on an outpatient basis to 30 mg/day; he improved but did not have a complete resolution of his spasticity. Two weeks before Mr. A came to the emergency room his dose had been increased to 40 mg/day, but due to confusion concerning the instructions, he began taking 60 mg/day. His only other medications were indomethacin and allopurinol for gout. After 10 days on the increased dose of baclofen and 6 months after beginning baclofen therapy, Mr. A abruptly discontinued the drug.

Within a day after Mr. A stopped taking baclofen, his family noted that he was sleepless, increasingly active, and expressing grandiose ideas. Two days later he continued to be agitated and began talking rapidly as if "confused," and he became belligerent. He did not sleep during the 3 days preceding his emergency room visit, and he refused meals.

When the patient arrived in the emergency room, psychiatric and neurological consultations were obtained. Mr. A was found to be alert and able to give the details of his prior illness and present circumstances. He spontaneously remarked to the examiners that he had recently stopped taking his baclofen and had "felt great" since then. He was questioned and specifically denied hallucinations and ideas of reference. He was easily distracted; talked loudly, rapidly, and volubly; gestured expansively; and ordered members of his family and hospital personnel about the room. His speech was unimpaired, but its content revealed a preoccupation with sexual topics and grandiose delusional thought. For example, he repeatedly referred to his dealings with well-known entertainment figures, whom his family denied he had ever met. The findings from the general and neurological examinations were unchanged from those previously recorded.

Mr. A was treated with a total of 8 mg of haloperidol i.m. in two doses, and his baclofen was reinstituted at a dose of 15 mg/day. Within 24 hours he had returned to his previous mental status and behavior. At follow-up interviews over the next 8 months, it was noted that Mr. A remained free from recurrence of any disorder of affect or thought.

Discussion

The interval between Mr. A's discontinuation of baclofen and his onset of symptoms is consistent with previous reports (3–5). The psychological and behavioral features—increased vocalization and motor activity, aggressiveness, sleeplessness, and decreased appetite—correspond with *DSM-III* criteria for mania, although Mr. A's episode terminated four days after haloperidol was administered and baclofen was resumed. He also displayed characteristic symptoms of elevated mood and increased interest in sex.

Although this patient's manic psychosis occurred when baclofen was suddenly withdrawn, the underlying neurobiologic mechanism is unclear. Bunney (6) has reviewed the evidence for the primary role of dopamine, rather than norepinephrine, in mania and the switch process; Bunney and associates (7) hypothesized that supersensitive dopamine receptors develop before the onset of mania. Early theories concerning baclofen's mode of action focused upon baclofen's ability to inhibit dopamine release (1), and it was presumed that baclofen acted as a GABA agonist. More recently, Saito and associates (8) have demonstrated that baclofen is more properly characterized as an inhibitor of substance P. Substance P has been reported to stimulate monoaminergic neurons in the brain stem and to excite nerve terminals impinging on dopamine neurons (9). One might postulate that a "rebound phenomenon" occurs following the rapid release of inhibition of substance P and resulting neurobiologic events that lead to mania. Such a manic episode after sudden withdrawal of baclofen would lend support to the dopamine thesis proposed by Bunney and associates and would raise the question of the role of substance P in dopamine release and dopamine receptor sensitivity.

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LETTERS TO THE EDITOR

This section contains reactions to Journal *articles, statements of opinion, brief research findings or ideas, short case descriptions, requests for information, etc. Letters should be submitted in duplicate to the Editor, who makes all decisions regarding publication. Letters must be typed double spaced throughout and should not contain more than 500 words and 5 pertinent references. Criticisms of published articles will automatically be sent to the author(s) for response. Letters will be edited for clarity and conformance with* Journal *style. We regret that we cannot inform writers of the disposition of letters or return those which are not printed.*

Psychologists' Reaction to *DSM-III*

SIR: From comments made by Robert L. Spitzer, M.D., and associates in "*DSM-III*: The Major Achievements and an Overview"(February 1980 issue), one forms the impression that psychologists have had their pressing questions about the new order answered and their initial doubts dispelled. Our fear of medical hegemony being thus assuaged, psychologists should now be able, according to Dr. Spitzer and associates, to evaluate the incipient nosology as a scientific document. Further, that evaluation is said to be quite favorable.

This is neither a complete nor an accurate portrayal of events and reactions. A number of prominent psychologists have emerged as outspoken critics of *DSM-III*. Schacht and Nathan (1), the latter a member of the *DSM-III* Liaison Committee of the American Psychological Association, have evaluated the new system and described it as being "very bad for psychologists." Joseph Zubin, whose Biometrics Research Unit of the New York State Psychiatric Institute greatly influenced the development of *DSM-III*, has questioned the basic assumptions on which it rests and evaluated it as being detrimental to the science of psychopathology (2). Taking the next logical if radical step, Zubin has proposed to psychologists that an interdisciplinary commission on the classification of human behavior be established. Garmezy (3), during his tenure as president of the Division of Clinical Psychology of the American Psychological Association, publicly decried the probably adverse effects *DSM-III* will have on the well-being of America's children. I presented the argument to psychologists that *DSM-III* was not born of medical or social science and, therefore, is not a useful tool for either discipline (4). On a broader, organizational level, the Board of Directors of the American Psychological Association recently voted to withhold comment on *DSM-III* while at the same time reactivating its own Task Force on Descriptive Behavioral Classification to explore the development of an empirically based alternative classification system. These events do not bespeak satisfaction with the new order.

Psychologists' misgivings center around two basic issues. The first is conceptual. A physical disease or mental illness conception of many of the new categories of disorder is not heuristic. For instance, to consider the many childhood disturbances as mental disorders after the manner of diseased states does violence to both our thinking about the nature and alleviation of these problems and to the children so labeled. Independent of medical content, categorical representations of behavioral process have not proven fruitful in psychology.

DSM-III also raises fundamental issues of science. Much of the new order lacks empirical grounding. The basis of the scores of new disorders is not one of scientific discovery and justification. The inclusiveness of *DSM-III* and great breadth of the new definition of mental disorders reflect the politics of mental health in an increasingly competitive marketplace rather than the progress of scientific inquiry and enlightened practice. (This is not to disregard the relatively isolated advances that have been made in the classification of affective disorders.)

Many psychologists want no part of the new order. Unfortunately, the development of an empirically based alternative is not a matter of simply finding the best committee or writing a new manifesto. In the meantime, the politics and business of mental health grind on. Psychology's future is unclear at the moment. It is apparent, however, that psychologists *and* psychiatrists should contemplate their futures vis-à-vis *DSM-III*. Neither social nor medical science can sustain statements of order in nature that present the world as one group wants it known instead of how logic and inquiry reveal it to be.

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Dr. Spitzer and Ms. Williams Reply

SIR: Dr. McReynolds apparently believes that *DSM-III* is primarily a political, rather than a scientific, document

meant to ensure medical hegemony in order to corner the mental health dollar. Nonsense! During the development of *DSM-III* great efforts were made to avoid including anything that would have only a political, rather than a clinical or scientific, justification. For example, a statement was originally planned for the introduction to *DSM-III* asserting that mental disorders were a subset of medical disorders. (The motivation for this was not to justify medical hegemony, but rather to refute the Szaszian charge that "mental illness" is only a myth and has nothing to do with medicine.) The words "physician" and "psychiatrist" do not appear in *DSM-III* because, like its predecessors, *DSM-III* will be used by a variety of mental health professionals.

On a conceptual level, Dr. McReynolds takes issue with the concepts of "physical disease," "mental illness," and "diseased states," yet these terms do not appear in *DSM-III*. Although Dr. McReynolds claims that the *DSM-III* concept of mental disorders (made explicit in the introduction to *DSM-III*) "reflects the politics of mental health . . . rather than the progress of scientific inquiry," he offers neither an alternative concept nor a specific critique of the *DSM-III* concept.

Dr. McReynolds states that "much of the new order lacks empirical grounding." If he means that many of the *DSM-III* categories have not been fully validated, we would heartily agree but would also argue that the specificity of the *DSM-III* classification and diagnostic criteria will facilitate studies that will provide data toward a more valid system in the future. Is there a better way? (Strictly speaking, the dictionary definition (*Webster's*, 1974) of "empirical" is "relying on experience and observation . . ." and all of the *DSM-III* categories do reflect the experience and observations of clinicians.)

Dr. McReynolds claims that "many psychologists want no part of the new order." It is true that Drs. Zubin, Garmezy, Schacht, and Nathan have written articles highly critical of *DSM-III*. However, they would acknowledge that these articles were based on earlier drafts of *DSM-III* and that their assessment of the final document is far more positive. There is no reason to believe that psychologists (or any one else, for that matter) should be entirely satisfied with *DSM-III*. However, what has impressed us is the degree to which *DSM-III* is viewed as a significant advance by psychologists. For example, the 1980 edition of *Abnormal Psychology, Current Perspectives* (coordinating author Richard R. Bootzin), one of the most widely used introductory abnormal psychology texts, incorporates *DSM-III* concepts and terms throughout the text. In addition, the soon-to-be published *Behavioral Assessment of Adult Disorders*, edited by David H. Barlow, acknowledges the advances reflected in *DSM-III* and relies heavily on the classification of *DSM-III* as the first step in a behavioral assessment. Finally, as we noted in our article, the American Psychological Association's own liaison committee concluded that *DSM-III* represents "substantive advances in the 'state of the art' of psychopathologic diagnosis." Dr. McReynolds forgot to mention that when the American Psychological Association voted to "withhold comment" on *DSM-III*, it was their own committee's positive assessment of *DSM-III* that they voted to suppress. Who's being political?

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Regional Suicide Rates and the Hazards of Minority Status

Wechsler and Pugh (1) reported that individuals whose social characteristics are relatively uncommon in a community should have a higher rate of psychiatric disturbance than those living in communities where their social characteristics are more common. In "Ethnic Density and Psychiatric Hospitalization: Hazards of Minority Status" (December 1979 issue), Judith Godwin Rabkin, Ph.D., found that ethnic groups in New York City had higher rates of psychiatric hospitalization if they lived in geographic areas where their ethnic group was in a minority.

These two studies generate the hypothesis that a similar result might be found if the regional variation of the suicide rate of nonwhites in the United States were examined. In the continental U.S. those states with a relatively lower proportion of nonwhites would be expected to have higher suicide rates among nonwhites than would states with a relatively higher proportion of nonwhites.

To test this hypothesis, I obtained the suicide rates of whites and nonwhites for 1959–1961 from Massey (2) and the proportion of nonwhites in each state in 1960 from the *Statistical Abstract of the United States*. The product-moment correlation between the suicide rate of nonwhites and the proportion of nonwhites in each state was −.41 ($N=48$, $p<.01$). Thus states with a higher proportion of nonwhites had a lower suicide rate for nonwhites. (Incidentally, no similar association was found for the rate of death from homicide for nonwhites.)

The relationship between the suicide rates of whites and the proportion of nonwhites was −.12 and was not statistically significant. This lack of association makes sense because in no state were whites in the minority. In fact, the state with the highest proportion of nonwhites was Mississippi, where 42% of the population was nonwhite.

In 1973 I reported that suicide rates vary with longitude and latitude in the continental United States (3). For my current study the latitude and longitude of each state were operationally defined as the latitude and longitude of the capital of the state.

The suicide rate of whites showed no north-south variation ($r=.04$) but was higher in the west ($r=.42$, $p<.01$). In contrast the suicide rate of nonwhites was higher in the north ($r=.35$, $p<.02$) but showed no east-west variation ($r=.20$, n.s.). To control for the effects of the proportion of nonwhites in each state on the variation by latitude and longitude of the suicide rate of nonwhites, partial correlation coefficients were calculated. The partial correlation between the suicide rate of nonwhites and latitude, holding the proportion of nonwhites constant, was .07 (n.s.). The partial correlation between the suicide rates of nonwhites and longitude, holding the proportion of nonwhites constant, was .13 (n.s.). Thus there appears to be no regional variation in the suicide rates of nonwhites by latitude and longitude if we hold the proportion of nonwhites in each state constant. The regional variation in the suicide rates of nonwhites is predicted more effectively by the proportion of nonwhites in the population of each state.

These data support the hypothesis proposed by Wechsler and Pugh and recently confirmed by Rabkin. In particular, for minority ethnic groups, the lower the proportion of the minority ethnic group in a region, the higher the suicide rate and thus, perhaps by inference, the higher the rate of psychiatric disturbance.

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Anticholinergics and Tardive Dyskinesia

SIR: In "Neuroleptic-Induced Supersensitivity Psychosis: Clinical and Pharmacologic Characteristics" (January 1980 issue) Guy Chouinard, M.D., M.Sc., and Barry D. Jones, M.D., raised several issues relevant to the pathophysiology and treatment of tardive dyskinesia. They stated that patients who develop tardive dyskinesia while receiving neuroleptics exhibit few parkinsonian symptoms. Therefore, the authors concluded that drug-induced hypokinesia masks tardive dyskinesia. This view is not accurate pharmacologically and may lead to confusion in understanding the pathophysiology and management of tardive dyskinesia. The authors' corollary suggestion that all patients receiving neuroleptics be given concomitant anticholinergic agents to "unmask" tardive dyskinesia is inappropriate in most cases, and could be potentially harmful.

Although the symptoms of chorea and parkinsonism may appear to be clinically opposite, the two are neuropharmacologically distinct and cannot be viewed as poles of a spectrum. Both abnormalities relate to alterations of striatal dopaminergic systems, but at least two separate populations of striatal dopamine receptors seem to be involved in the pathophysiology of these conditions (1). The existence of these different striatal dopamine receptors helps to explain the frequent occurrence of both tardive dyskinesia and parkinsonism in the same patient. In previous clinical studies this has been substantiated by the observation that, when neuroleptic doses were lowered, patients developed tardive dyskinesia well before their drug-induced parkinsonism abated (2). These data suggest that when neuroleptics diminish dyskinesia, they do so by selectively altering specific hypersensitive dopamine receptors, rather than by aggravating drug-induced parkinsonism as Drs. Chouinard and Jones suggested.

Since cholinergic systems influence these different dopamine receptor site populations (3), the use of anticholinergics to unmask tardive dyskinesia must be viewed with caution. Human studies demonstrate that anticholinergic drugs exacerbate tardive dyskinesia, and it has been suggested that anticholinergic agents may in fact potentiate the development of this iatrogenic disorder (1). In one large series the concurrent administration of neuroleptics and anticholinergics appeared to correlate with the development of tardive dyskinesia (4). This report has led several authors to advise against combined anticholinergic and neuroleptic administration unless patients have drug-induced parkinsonism (1, 2, 4). Although anticholinergic drugs may unmask tardive dyskinesia, they may do so by participating directly in the pathogenesis of the condition. Thus, if they act additively to potentiate tardive dyskinesia, they are harmful rather than beneficial and should not be used routinely.

Drs. Chouinard and Jones suggest the routine use of anticholinergics to detect early tardive dyskinesia. The same information can be obtained from periodic neuroleptic withdrawal or dose reduction. This practice has the advantage of allowing the simultaneous assessment of both psychiatric need for continued neuroleptic administration and the early detection of tardive dyskinesia. Since tardive dyskinesia appears to relate directly to neuroleptic administration, the basic mode of prevention and diagnosis should be withdrawal of the pathogenic agent, not the addition of a second, potentially harmful drug.

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Drs. Chouinard and Jones Reply

SIR: Dr. Tanner and associates proposed withdrawal or dose reduction of neuroleptics as the basic mode of prevention and diagnosis for tardive dyskinesia. However, such a reduction or discontinuation is not feasible for most patients—a dilemma clinicians have been facing since becoming aware of tardive dyskinesia. Thus we must examine other methods to detect tardive dyskinesia. We suggested that short-term concomitant use of anticholinergic drugs with neuroleptics will permit earlier detection of tardive dyskinesia without exacerbating psychosis and that it may prevent severe forms of the disorder. Dr. Tanner and associates criticized this suggestion by arguing that 1) it is not pharmacologically accurate because the effect of anticholinergics on tardive dyskinesia is most likely a direct one rather than an indirect one through parkinsonism; and 2) the central effect of anticholinergics may be deleterious and contribute to the development of tardive dyskinesia.

We agree that some of the effect of anticholinergics on tardive dyskinesia may be the result of a direct central action, probably due either to inhibition of dopamine reuptake or exaggeration of a dopaminergic-cholinergic imbalance. However, there is strong evidence for an indirect effect through improvement of parkinsonism. In a previous study of 261 schizophrenic outpatients chronically treated with neuroleptics, we found that patients manifesting tardive dyskinesia tended to have fewer parkinsonian symptoms than those without the disorder, especially when tremors and akathisia were excluded from consideration (1). Although this view, according to Dr. Tanner and associates, may not be

accurate "pharmacologically," it is clinically accurate and is supported by the study of De Fraites and associates (2). We have also observed in our clinical work that improvement of hypokinetic symptoms, whether by removal of neuroleptics, use of anticholinergics, or by the addition of other drugs such as L-dopa, is usually associated with exacerbation of tardive dyskinesia. In contrast, we have seen no such relationship between hyperkinetic symptoms such as tremor and akathisia and tardive dyskinesia. This would suggest that a peripheral masking effect by hypokinetic symptoms of parkinsonism is also involved in the masking of tardive dyskinesia. Dr. Tanner and associates incorrectly cited Crane's paper as evidence for a dissociation between parkinsonism and tardive dyskinesia. They attributed to Crane the observation that "patients developed tardive dyskinesia well before drug-induced parkinsonism abated." This result did not apply to Crane's study but to a study of Degkwitz and associates which Crane quoted. In fact, Crane found the opposite and reported that in patients whose medication was changed, tardive dyskinesia varied inversely with symptoms of parkinsonism. He also described a transitional stage between tremor and dyskinetic movements. Thus, the article by Crane in fact contradicts Dr. Tanner and associates' point.

Dr. Tanner's second point is that the concomitant use of anticholinergics is "inappropriate in most cases, and may be potentially harmful." This does not correlate with our own findings. We are currently preparing a paper in which a neurologist, participating in a double-blind study of depot fluphenazine, found that 92% of the patients had some parkinsonian symptoms when withdrawn from anticholinergics. The psychiatrist who prescribed the anticholinergics also found that 85% needed this medication. These subjects were outpatients chronically treated with depot neuroleptics. Since a large number of schizophrenic patients are now being treated with depot fluphenazines, the comment that the use of anticholinergics "is most often inappropriate" does not apply to the most recent findings on the incidence of drug-induced parkinsonism (3).

Dr. Tanner and associates also mentioned that the use of anticholinergic drugs "may be potentially harmful." The subject needs further investigation and at this time no long-term controlled study has to our knowledge shown that the use of anticholinergic drugs may be harmful. To support their view, Dr. Tanner and associates cited a long-term study by Crane and Navanjo showing that concurrent administration of neuroleptics and anticholinergics correlates with the development of tardive dyskinesia. We were unable to locate such a finding or statement within the Crane and Navanjo study. In any event we offer the following evidence to justify our conclusions: 1) we found in a study of 260 schizophrenic patients chronically treated with neuroleptic drugs that the use of anticholinergic drugs was not a factor related to the development of tardive dyskinesia; 2) in a study (4) including 20 patients with tardive dyskinesia, we found that exacerbation of tardive dyskinesia by temporary administration of an anticholinergic drug could be reversed to previous baseline severity by discontinuing the drug; 3) Christensen and Nielsen (5) have demonstrated that the administration of anticholinergic drugs in mice during the phase of dopaminergic supersensitivity development induced by neuroleptics had no influence on the development of that supersensitivity.

Thus we think that there is currently substantial evidence to suggest that anticholinergic drugs uncover tardive dyskinesia by both a central and peripheral mechanism, that the central mechanism is reversible on short-term administration and does not appear to contribute to the development of tardive dyskinesia. We also think that anticholinergics are useful drugs in allowing the clinician to identify more rapidly the presence of tardive dyskinesia and, by reducing disabling parkinsonian symptoms, to facilitate social rehabilitation.

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Another Case of Nocturnal Enuresis

SIR: I am writing to you in regard to "Nocturnal Enuresis Caused by Psychotropic Drugs" by Ramakrishnan S. Shenoy, M.D. (June 1980 issue).

It may well be that nocturnal enuresis is, as Dr. Shenoy suggested, not so uncommon. In the late 1960s I submitted a communication to the *American Journal of Psychiatry* reporting the same side effects. Rejection by the *Journal* was predicated on the fact that my patient had had a transurethral resection of the prostate, although there was no evidence of residual abnormality or defective urinary tract function on testing. The patient showed nighttime, and only nighttime, enuresis. My patient, like one of those described by Dr. Shenoy, had been treated with thioridazine. At the time my article was submitted, inquiries were made of the manufacturer of this product, who denied any previous reports of a comparable side effect.

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Glutamate Neurotoxicity and Tardive Dyskinesia

SIR: We read with interest the letter to the editor "MSG: A Possibility in Treating Tardive Dyskinesia" by Peter J. Holland, M.D. (December 1979 issue). Dr. Holland's suggestion that glutamate may be useful in treating tardive dyskinesia should be regarded with extreme caution.

Microinjection of glutamate or an analogue, kainic acid, into the rat striatum destroys intrinsic neurons, but spares axons of passage and the termination of extrinsic neurons (1). The biochemical, anatomical, and behavioral sequelae appear remarkably similar to those of Huntington's chorea

(1, 2). Based on these results and the fact that the striatum receives a major glutamatergic pathway from the neocortex, it has been proposed that excessive glutamate neurotransmission may offer an explanation for the degeneration that occurs in patients with Huntington's chorea (1, 2).

This excitotoxic hypothesis of the pathogenesis of Huntington's chorea also leads to an explanation of tardive dyskinesia, which occurs after long-term therapy with dopaminergic blockers. Treatment with neuroleptics removes the presumed inhibition of the dopaminergic nigro-striatal pathway onto the striatal cholinergic interneurons. In addition, it is possible that neuroleptics may block presynaptic dopaminergic inhibition located on the terminals of the glutamatergic cortico-striatal pathway (3). Thus, the lack of this inhibition could cause an otherwise normal excitatory glutamatergic input from the cortex to become relatively excessive and to gradually destroy striatal interneurons. This would result in a gradual shift in the dopamine-acetylcholine balance in the striatum, a biochemical condition thought to underlie involuntary movements (4). Dr. Holland suggested that treatment with glutamate may normalize this shift by producing an increase in acetylcholine. This appears unlikely because glutamate has been demonstrated to stimulate the release of dopamine from the terminals of the nigro-striatal pathway (5), which would be contraindicated in the treatment of tardive dyskinesia.

As Alan J. Gelenberg, M.D., and associates pointed out in their reply to Dr. Holland (December 1979 issue), there is debate over how much glutamate can penetrate the blood-brain barrier. However, we feel that the increasing number of supportive studies on the neurotoxic properties of glutamate and its analogues further suggests that glutamate may be potentially dangerous in treating tardive dyskinesia and related disorders.

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Dr. Holland Replies

SIR: Drs. Sanberg and Mark raised two important considerations regarding MSG. 1) How much, if any, glutamate crosses the blood-brain barrier? and 2) What potential neurotoxic effects might glutamate have in the CNS?

In a review of recent literature, Kizer and associates (1) remind us that as late as 1951 large doses of MSG (20–30 g/day) were being administered to treat retardation and petit mal, without apparent lasting effect.

The Chinese have used seaweed extract containing glutamate for at least 1,000 years. Today, tons of MSG are added to food merely to enhance flavor. Knowledge of MSG's effects in man is inadequate. The potential uses and hazards of this ubiquitous compound need to be defined.

I thank Drs. Sanberg and Mark for their interest in my hypothesis and hope that this dialogue stimulates the much needed research in this field.

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"To Love and to Work": Historical-Theoretical Considerations

SIR: "To Love and to Work," the theme of the 1980 annual meeting, is generally attributed to Freud, but cannot be found in his writings. Rather, it was introduced by Erik Erikson (1) in 1950 as Freud's curt reply to the question, "What should a normal person be able to do well?"

Erikson used the anecdote to counteract the frequent understanding that for Freud the highest human value was "to have good orgasm, with a fitting 'object,' " and that regularly (1). Freud had left himself open to such an understanding through his sexual reductionism, as in the following: "Primitive man . . . made his work agreeable . . . by treating it as the equivalent of and substitute for sexual activities" (2). Freud influenced psychoanalytic authors such as Feldman to contend that "manual labor . . . contains elements of anal sadism and masturbation," while intellectual labor contains more "elements of oral masochism and female identification" (3). If Freud said that ultimately "to love and to work" are the essentials of life, he left this casual remark theoretically quite suspended.

In contrast, Adler regarded love and work, together with communal life, as the three major problems of human existence, or life tasks. Mental health depends on the successful solution of these life tasks through social interest expressed in cooperation. "Their solution decides the destiny and the welfare of humanity" (4). From the 1920s on, Adler stressed this conception of the three life tasks, in slightly varying nomenclature, as being central to his theory.

"To love and to work" has in recent years been cited innumerable times as the quintessence of Freud's wisdom, even becoming the theme of an APA annual meeting. This phenomenon indicates the general trend in psychoanalysis and psychiatry toward humanistic value theory. This welcome development is in fact a movement toward the position Adler had taken as early as 1911. Other Adlerian concepts such as goal striving, the quest for self-esteem, the dialectics of intense ambition and inferiority feelings, and resistance in therapy as an attempt to humiliate the therapist, have increasingly found their way into psychoanalytic writing,

while the validity of much of Freud's metapsychology has been questioned. However, this movement toward Adler's position has so far remained largely unrecognized. The proper recognition of Adler would have the practical value of making the training of all mental health professionals more efficient.

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The Danger of Hyperalimentation

SIR: In "Treatment of Severe Weight Loss in Anorexia Nervosa with Hyperalimentation and Psychotherapy" (March 1980 issue) Michael J. Maloney, M.D., and Michael K. Farrell, M.D., properly stated that hyperalimentation is not a panacea and also noted that the anorectic patient's symptoms may be caused by physical starvation.

In 25 years of experience with many anorectic patients, we have been fortunate in not having one die of inanition (1, 2); we admit this may be due to chance. However, we believe that responsibility for eating belongs to the patient, and that even the most disturbed patients are able to assume that responsibility. When the physician assumes the responsibility, we feel it may be dynamically counterproductive. Nevertheless, the threat of a patient's death is anxiety-provoking and a strain on the omnipotent feelings of physicians.

A physician has a great need to do something active other than psychotherapy when treating an anorectic patient, who might look like an inmate of Dachau. Over the years we have lived with the anxieties of our colleagues and have managed most of these patients without hospitalization, let alone drugs or tube or intravenous feedings. Our anxiety has often been great, but we have taken comfort in the fact that physicians are not all-powerful, and in our belief that patients, supported by their physicians and families, do want to live.

Ambivalence about the effectiveness of psychotherapy is a problem not only for nonpsychiatrists but for psychiatrists as well. Total parenteral hyperalimentation is not a benign procedure, even in the best hands, and collaboration is not always easy.

This comment is intended to offer a word of caution and to state that management of anorectic patients is very complex. The use of total parenteral nutrition, which may seem rational, can result in even more problems and should not be undertaken lightly.

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JOHN B. REINHART, M.D.
PATRICIA L. GOETZ, M.D.
Pittsburgh, Pa.

Drs. Maloney and Farrell Reply

SIR: We are aware of Drs. Reinhart and Goetz's work with anorectic outpatients. The majority of our anorectic patients are also treated outside the hospital with no drugs, tube feedings, or intravenous feedings.

Our article was written to describe the comprehensive treatment of four patients with extreme weight loss. These patients had lost up to 44% of their body weight by self-starvation. They were admitted to the hospital when their weights dropped from 30.4 kg down to 16.6 kg (67 pounds down to 36 pounds). In the past these patients might have died. Drs. Reinhart and Goetz have not published the lowest weights of their patients. The majority of anorexia nervosa patients never reach this extreme.

In our article we were careful to explain that "the weight stabilization by means of hyperalimentation was at times life-saving for these severely anorectic patients. However, this was only the first stage in the rehabilitation process. The real work was then continued in psychotherapy to help the patients explore and master their conflicts over food, autonomy, and self-esteem." Drs. Reinhart and Goetz falsely inferred that we are ambivalent about the effectiveness of psychotherapy. Perhaps they are ambivalent about the medical treatment of severely malnourished patients. The comprehensive treatment of severe weight loss in anorexia nervosa requires attention to both psychiatric and medical problems.

We agree (and mentioned in our article) that hyperalimentation or total parenteral nutrition is a complex, difficult procedure. However, it can be life-saving for severely malnourished anorectic patients. Total parenteral nutrition is safe and effective in experienced hands (1). In addition, we found that improved affect, verbalization, and sociability from weight gain have assisted our patients in cooperating in psychotherapy.

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MICHAEL J. MALONEY, M.D.
MICHAEL K. FARRELL, M.D.
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Psychoanalytic Concepts and Inpatient Settings

SIR: In "Psychoanalytic Concepts in Psychiatric Training" (May 1980 issue), Herbert Pardes, M.D., suggested that the emphasis can be divided into exclusively psychoanalytic programs, those which exclude psychoanalytic concepts, and cafeteria-type programs. This last type seems increasingly prevalent today, especially in university centers where, in the competition for residents, program directors eagerly display their wide menus to demonstrate their commitment to eclecticism.

I support Dr. Pardes' recommendations for inclusion of psychoanalytic concepts, especially the teaching of their applicability in a variety of settings. A first-year resident who clings to the strict medical model may often find dynamic issues to be irrelevant to his day-to-day work. Brief hospitalization, the utilization review committee, and effective pharmacotherapy, can create the illusion that psychoanalytic concepts are only for outpatient work, some time in the future. Inpatient supervisors frequently fall prey to those same pressures, and thus both faculty and students miss this opportunity.

Outpatient supervisors are generally more psychoanalytically oriented, often to the exclusion of such factors as diagnosis (according to *DSM-II* or *DSM-III*), specific treatment goals, and pharmacotherapy. This tends to perpetuate the illusion that inpatients and outpatients are light years apart, requiring that residents adopt one "set," then another, depending on whether they are on the ward or in the clinic.

The usefulness of psychoanalytic concepts is not limited to outpatient psychotherapy. If supervisors can demonstrate and teach such utility in the inpatient or emergency setting, it will help demystify the role of psychoanalytic concepts.

RICHARD MOLDAWSKY, M.D.
Orange, Calif.

Reconsidering Some Recent Trends in Psychotherapy

SIR: Judd Marmor, M.D., has once again provided us with a landmark contribution to the literature in his article, "Recent Trends in Psychotherapy" (April 1980 issue). I wholeheartedly endorse his warnings about charismatic leaders who promote simplistic solutions to a hungry but all too vulnerable public. Nevertheless, I fear that Dr. Marmor may have wounded a number of ethically intentioned, compassionate, and balanced movements in his criticisms of the religious and inspirational therapies. Admittedly, the totalitarian attitudes promoted by some zealots warrant our fear and skepticism. On the other hand, some religious movements have endured by providing wisdom, meaning, and a sense of belonging, which are crucial not only to human survival but to human growth. My own experience indicates that helping patients become aware of the positive aspects of these traditions serves as an effective complement rather than competitor to psychotherapy. The temporal validity of these movements warrants our respect—what has endured through various ages may have as much merit as treatment techniques that survive today's empirical tests. If psychoanalysis can lay claim to this temporal validity, so may it be said of other belief systems that have endured for a far longer period.

THOMAS E. BITTKER, M.D.
Phoenix, Ariz.

SIR: I am reluctant to criticize Dr. Judd Marmor's "Recent Trends in Psychotherapy" because it was so interesting and informative, and also because Dr. Marmor has been one of the leaders in constructively criticizing the doctrinaire psychoanalytic approach as a psychotherapeutic method. However, there are a few points that I believe warrant criticism.

Dr. Marmor stated that, regardless of their differences, dynamic psychotherapy and behavior therapy are both firmly rooted in the scientific tradition. Agreement with this statement depends on the latitude we allow the concept of scientific tradition. Karl Popper put psychoanalysis and other dynamic psychologies among the nonsciences, because, unlike behavior therapy, their hypotheses are not open to refutation (1, 2). For Popper, the insights of psychoanalytic investigation may be correct, but the nature of its hypotheses and theoretical structure disqualifies it from being classified as a science.

Is it fair for Dr. Marmor to belittle the new cognitive therapies by representing them merely as a revolt against the emphasis on the unconscious in psychoanalytic theory, and suggesting that they are like psychoanalysis and psychoanalytically oriented psychotherapy because they "share a cognitive orientation"? The cognitive therapies of Ellis, Beck (whom Dr. Marmor did not mention), and others seem intended to make the individual aware of his conscious unrealistic fearful thoughts and judgments. Psychoanalytic therapy, on the other hand, in "making the unconscious conscious," may not be simply making the individual aware of what he was previously unaware but may in fact be indoctrinating him in a certain way of conceptualizing his problems. As far as results of therapy are concerned, this may not matter. But the cognitions which are the focus of attention in cognitive therapy cannot be equated with the cumbersome metapsychology of psychoanalytic theory. As Dr. Marmor and others have pointed out (3, 4), variables related to the therapist and the therapeutic interaction are likely to be more important than the manner in which the therapist or the patient conceptualizes the patient's problems. Surely Dr. Marmor will agree that one cannot rely on a psychoanalytic therapist, strongly committed to psychoanalytic theory by his own analysis and training, to dispassionately assess and classify the phenomena of mental disorder. Of course no one in psychiatry is completely uninfluenced by theoretical conceptualizations, but at least some psychiatrists (including Dr. Marmor himself) have changed their minds about formulations of problems through their years of experience. It would be refreshing if more psychoanalysts found exceptions to the all-encompassing explanations of psychoanalytic theory.

Finally, by quoting Dejerine, it seems Dr. Marmor intended to stress that psychotherapy depends exclusively on the beneficial influence of one person on another. I do not see how this point of view can be taken as a criticism of cognitive therapy, during which much interpersonal influence occurs through the medium of rational discussion. Cognitive therapists, to my knowledge, have never said that rational discussion is the only factor in their psychotherapy, and I am sure they would agree that nonverbal and subtle interpersonal influences are very important in making the patient receptive to the cognitive therapy approach.

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PAUL C.S. HOAKEN, M.D.
Kingston, Ont., Canada

SIR: I read "Recent Trends in Psychotherapy" by Judd Marmor, M.D., with interest and disappointment. My disappointment stemmed from the disturbing superficiality of Dr. Marmor's coverage of the subject, his generally discounting tone, his grounds for criticism of some of the modalities, and his omission of a major recent development, namely, Transactional Analysis.

In writing about Gestalt therapy and bioenergetics, Dr. Marmor cited inadequate training and/or ethical foundations as arguments against the techniques themselves. This does not address the question of the efficacy of the techniques, and is a criticism that could be applied, regrettably, to even "scientifically oriented" psychotherapists. I know many highly trained (many of them "scientifically trained"), ethical practitioners, who use Gestalt and bioenergetic techniques with excellent results. Dr. Marmor's article created the impression that these techniques are used only by marginal, unprofessional, guru-type practitioners, and therefore are worthy of little study. I agree that professional ethics are a legitimate concern for all of us, but that was not the subject of the article.

I cannot understand Dr. Marmor's omission of Transactional Analysis, which is practiced widely and effectively by a significant number of psychiatrists. Transactional Analysis addresses itself to the cognitive, affective, and behavioral aspects of personality, is rooted in psychoanalysis, and admirably fits Dr. Marmor's own criteria for effective psychotherapy, listed near the end of his article. In addition, the International Transactional Analysis Association has developed a searching and thorough training and accreditation process that examines all certified Transactional Analysis therapists for clinical competency. This model may be the most efficient measure currently in existence for evaluating what therapists actually do in their practices. After expressing concern over competence and training, it was strange that Dr. Marmor omitted Transactional Analysis, particularly because he did include Arica and est training, which lay no claims to being psychotherapy.

As a "scientifically trained," Board-certified psychiatrist, with extensive training in Transactional Analysis, Gestalt therapy, and bioenergetics, I found the article to convey a condescending attitude toward new approaches. If such an attitude is widely shared among psychiatrists, it may explain why so many troubled people are turning away from conventional psychiatry and seeking effective treatment elsewhere.

ROBERT A. MITCHELL, M.B., CH.B.
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SIR: Dr. Judd Marmor should be commended for attempting to inform the general medical psychiatric constituency of new approaches in therapy. Unfortunately, the scope of the work is much broader than could reasonably be discussed in a single paper. As a result, few of the new therapies or their theoretical underpinnings were given due consideration. For example, the direct association of Carl Rogers with encounter groups, although literally accurate, is for the most part an artifactual one. Though Rogers has had much influence on group experience and even educational theory, few would argue that either of these would take precedence over his contributions to the practice of individual "client-centered" psychotherapy. In fact, Rogers' empathic existential approach is more closely linked to an intensive dynamic orientation than it is to emotive abreactive therapy, as classified by Dr. Marmor. For interested readers, Havens (1) has discussed the potential of Rogerian influence on existential therapeutic methods from an analytic perspective.

Isadore From, a student and coworker of Fritz Perls currently affiliated with the New York Gestalt Institute, presents the practice of Gestalt therapy as requiring an understanding of fundamental dynamic principles. In his lectures Dr. From emphasizes the need to recognize that Gestalt theory is not well represented by the staged display gimmicks of the "hot-seat" or "topdog-underdog." Instead, apart from the faddism to which it has sadly become subject, Gestalt therapy is often done one-to-one over an extended period of time, without denial by the therapist of historical influences on the patient's current experience. Sidney Rosen (2) has written a highly intelligible paper discussing his personal experiences using Gestalt techniques in corroboration with dynamically oriented methods.

One other approach unfortunately not done justice by Dr. Marmor's review (and for which he curiously cites no references) is Arthur Janov's primal therapy. David Rosen (3) presented a telling account of his direct observations of primal work and also in very sensible fashion the experience of his own personal primal therapy. In concluding his paper he wrote, "Primal Therapy is solidly based in psychoanalytic theory. It uses techniques and information based on Gestalt and Reichian therapy, as well as knowledge from studies of the effects of social isolation. It is too powerful to be dismissed as another fad and too effective to be ignored."

It is with such "reconnaissance" psychiatry that an unbiased, in-depth evaluation of new techniques and their applicability can be presented. Discussions of therapeutic approaches made from afar without actual personal exposure as participant or observer cannot reasonably be considered objective scientific study. Dr. Marmor failed to present the new glass as half full with promise rather than half empty. Unless this is done, the merit of the new techniques may be too easily overlooked and, what is worse, it may be lost.

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GEORGE M. DOMINIAK, M.D.
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Dr. Marmor Replies

SIR: Dr. Bittker may have misread me when he referred to my "criticism of the religious and inspirational therapies." The section in my article referring to that group of therapies

was quite brief, with no evaluative judgments except for the comment that charlatans are involved in the leadership of some of the Eastern religious movements (an observation that I believe is warranted). I agree with Dr. Bittker that religious movements can and do provide meaning and an important sense of belonging to millions of people.

Dr. Hoaken's statement that the "hypotheses and theoretical structure [of psychoanalysis] disqualify it from being classified as a science" is only partially correct. Although it is true that much psychoanalytic metapsychology (Freud himself called it his "mythology") is indeed more fanciful than scientific, the solid core of psychoanalysis is based on the accumulation of clinical data and the drawing of inferences from those data, inferences that do lend themselves to validation no less than the various hypotheses underlying the behavioral therapies do. Therefore, it is not inaccurate to say that psychoanalytic therapy is rooted in scientific tradition.

It was not my intention to "belittle" the new cognitive therapies but only to point out that it is simplistic to assume that therapeutic changes achieved by them are due primarily to increasing cognitive awareness. I have made similar criticisms of the assumption that the therapeutic results of psychoanalysis were the consequence of simply "making the unconscious conscious." As I indicated in my article, there are other significant processes taking place in these therapies, not the least of which is the quality of the therapist-patient relationship. This is not to imply that increased cognitive awareness is of no value in the therapeutic process. I do believe that it is.

Dr. Mitchell overlooked the fact that I was not attempting (nor could I, within the limits of my essay) to "evaluate" the different therapies. Without denying that many of them were therapeutically helpful, I called attention to the inadequacy of therapist training and patient screening that characterized many of these approaches. My omission of Transactional Analysis was not pejorative. I consider it to be an offshoot of psychoanalytic theory and would include it in the scientifically oriented group of therapies. I am glad to hear that there is now an organized postgraduate training program for that approach. However, I hope that everyone admitted into that program is required to have the kind of admirable training that Dr. Mitchell himself has had.

Dr. Dominiak correctly pointed out that the scope of my topic was indeed broader than could be encompassed in a single paper. My article was not intended to be comprehensive but merely to give a sampling of some of the new therapies.

Similarly, my reference to Carl Rogers was deliberately limited to his considerable influence on the encounter group movement. I am fully aware of his significant contribution to the development of individual client-centered psychotherapy and have, in fact, briefly discussed his technique in another context (1).

I also realize that some competent dynamic psychotherapists utilize various principles and techniques of Gestalt therapy. However, I have personally witnessed Fritz Perls in action, seen his audiovisual tapes, and read his books. I think it is not unfair to say that in his later years his technique was primarily focused on promoting and achieving emotional abreaction in his patients.

Finally, I am appalled by Dr. Dominiak's uncritical view of Janov's primal therapy. Has he taken the trouble, as I have, to read Janov's books? Janov, himself, depreciates psychoanalytic theory and therapy, rejects the significance of interpretation, the therapeutic usefulness of transference, and unabashedly asserts, among a host of other extraordinary claims that strain credibility, that primal therapy is the only cure for "neurosis"—an all-encompassing term under which he subsumes the psychoneuroses, the personality disorders, schizophrenia, paranoia, the sexual deviations, and drug addictions. I leave it to *Journal* readers to draw their own conclusions about the scientific value of his approach.

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JUDD MARMOR, M.D.
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The Use of the Term "Public Health"

SIR: George Gardos, M.D., and Jonathan O. Cole, M.D., may wish to reconsider the term "public health" as used in their overview, "Public Health Issues in Tardive Dyskinesia" (July 1980 issue).

During my training years "public health" was reserved for those illnesses that were communicable or were caused by poor sanitation or environmental conditions. They were a separate class of illnesses because it took public measures to bring about a major aspect of their alleviation; no private doctor could easily trace down all the contacts in a case of syphilis, no private doctor could reengineer the sanitation conduits, no private doctor could drain the swamps where mosquitoes that carried malaria breeded.

In recent years the use of the term public health has shifted to diseases that are widespread throughout the population. Hypertension has been referred to as a public health problem. It remains for those who have extended this usage to justify it. Drs. Gardos and Cole reported no such widespread incidence of tardive dyskinesia in their overview to warrant this extended usage.

In the future Drs. Gardos and Cole may wish to reestablish the fine distinction between diseases that can be handled by a personal physician and those that require the intervention and power of the state. Today, when the state is making inordinate intrusions into the practice of medicine, it seems particularly imperative to maintain such distinctions.

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BOOK FORUM

NANCY C. ANDREASEN, M.D., EDITOR

The Second Coming, *by Walker Percy. New York, N.Y., Farrar-Straus-Giroux, 1980, 360 pp., $12.95.*

The fragile wisdom that inheres in the perceptions and thoughts of madmen is an irresistibly delicious subject for writers with a taste for the ironic. Like most classic subjects, it seems fresh and new in the hands of a good writer. Walker Percy's most recent novel, *The Second Coming,* portrays a world of madness. It is a world in which only the mad are sane.

The male protagonist, Will Barrett, is suffering from depression and falling spells at the beginning of the novel. His wife, a cheerful, obese, wealthy Christian doer of good works, has recently died and left him sole heir to her fortune of 60 million dollars. Will, who married her in order to be able to enjoy giving and pleasing more than in order to enjoy her great wealth, has recently retired from a successful Wall Street law practice. To all envious eyes around him in Linwood, N.C., he has managed to "do it all," to earn wealth himself in addition to marrying it and inheriting it. But Will is sad and confused: by memories of his father's bungled attempt to kill him and then to commit suicide one day when they were hunting together during his twelfth year, by his father's statement that they are "two of a kind," by his father's later completed suicide, by the shallowness and hypocrisy and inhumanity of the world around him, by an existential need to find meaning in his own life and in life in general. He decides to conduct a Pascalian experiment. He retreats to a cave with a bottle of barbiturates to await the last days, attempting to perform both the perfect test of God's existence and the perfect suicide if God does not exist. The perfect experiment is marred, however, by human imperfection in the form of a toothache that drives him out from the cave, and we are left wondering about the meaning of its outcome. Will concludes that the answer is muddy, that he did not have a chance to put his question to a fair test. But the possibility exists that the toothache was providential, the very sign he was awaiting, a sign he fails to notice because it is too small and insignificant a revelation for a man looking for the Apocalypse.

Will emerges from the cave to find himself in the care of Allison Vaught, a recent escapee from Valleyhead Psychiatric Hospital. She had been there two or three years. Her escape occurs after her parents make one of their exceedingly rare visits and present hopeful plans for her future, motivated by parental love and concern that have recently been stimulated by the discovery that Allison has inherited a multimillion dollar fortune from her Aunt Sally Kemp. She escapes in order to avoid further ECT, referred to as "buzzing" and portrayed as an unpleasant and painful punishment ordered by a rather insensitive and stupid psychiatrist, and to seek her own identity in the real world. Discovering that the farmhouse she has inherited has burned down, she turns the surviving greenhouse into a home. In the process she displays intelligence, imagination, and creativity. She also speaks (but does not think in) a wonderfully funny language evocative of schizophrenic language but more poetic. Unlike the chronic schizophrenic patient, Allison views the world with the joyous curiosity of a happy child.

The attraction between Will and Allison is irresistible. He has falling spells, and she is "a hoister." He doubts and denies, while she affirms. Their mutual need and interdependence satisfy each. But their union is nearly thwarted. During Will's disappearance in the cave, his greedy family and friends close in, trying to get control of his wife's inheritance—Will's daughter and a minister named Jack Curl to set up a religious community, his friend Dr. Vance Battle to build a new hospital. In fact, they attempt to cure his existential longings by controlling an aberrant blood pH, which temporarily reduces him to an unthinking inhabitant of a rest home for the elderly. Like Allison, Will eventually chooses to escape and to live in the saner world of apparent madness. Their madness will permit them both to survive and to find meaning in a topsy-turvy apocalyptic world, in which "the best lack all conviction, while the worst are full of passionate intensity."

As the plot suggests, this is a tightly constructed book, rich in ideas and symbols, which gives the reader much to ponder. It also sometimes has the excitement and drama of an old-fashioned thriller, and it is full of mocking and ironic humor. Born-again and traditional Christians, doctors and dentists, the Old South and the New, runners and golfers, psychiatrists and patients, are all seen through Percy's ironic eyes. Some psychiatrists may find their portrayal unfair and troubling (and perhaps it is), but this portrayal is necessary in order for Percy to present his theme, the sanity inherent in madness and the insanity of the apparently sane. Thoughtful readers will find this book fun and funny as well as intellectually exciting.

Walker Percy is one of America's major living novelists. Trained as a physician at the University of North Carolina and Columbia-Presbyterian, he turned to writing when he developed tuberculosis during a pathology residency. His works show a series of recurrent fascinations and concerns characteristic of Christian existentialism: the emptiness and materialism of our society, our preoccupation with popular psychology and clichés, the loneliness and isolation experienced by modern man, his search for a meaning inherent in his existence, pressures from society to give in and give up, an awareness of sin, and a hope that redemption is possible. His subjects often deal obliquely or directly with medicine and psychiatry. Readers who have not yet discovered him will enjoy doing so. *The Second Coming* is a good beginning.

N.C.A.

Loss: Sadness and Depression. Attachment and Loss, Vol. III, *by John Bowlby. New York, N.Y., Basic Books, 1980, 462 pp., $22.50.*

The full impact of John Bowlby's work has not yet been realized in the United States. The appearance of this final volume in his trilogy on attachment and loss should place Bowlby alongside such other important psychiatric and social theorists as Erikson, Fromm, Meyer, and Sullivan.

The principal theme in Bowlby's concept is that intimate attachments to other human beings are the core around which a person's life revolves and from which one obtains strength and pleasure. Many of the most intense emotions arise during the formation, maintenance, disruption, and renewal of attachment relationships. Threat of loss arouses anxiety, actual loss gives rise to sorrow, and both arouse anger. The unchallenged maintenance of a bond is experienced as security, and the renewal of a bond is a source of joy. Attachment behavior is rooted in primate life and is characteristic of many species during the course of evolution because it keeps the species close to caretakers, thus reducing the risk of harm.

Drawing somewhat from psychoanalysis, but more often from ethology and cognitive psychology, Bowlby's paradigm dispenses with many abstract concepts, such as psychic energy and drive, and relies considerably on empirical data. His concepts are of interest to psychoanalysts and compatible with those of neurophysiology and developmental psychology; they are capable of meeting the ordinary requirements of a scientific discipline.

Unlike psychoanalytic theorists, Bowlby sees attachment behavior as active throughout life and not indicative of pathology or regression. Like psychoanalytic theory, his view is that faulty attachment behavior in adults may be the result of faulty (i.e., anxious, insecure, incomplete, unresponsive) attachment experiences occurring during the early formative years.

Drawing from clinical and epidemiologic data, Bowlby applies this conceptual framework to the understanding of loss and grief reactions. In separate chapters he describes the emotional experiences of the loss of a spouse and a child, mourning in different cultures, disordered variants of mourning, and mourning of young children in response to the loss of a parent. In one chapter he attempts to understand why some people follow a pathologic course of mourning and others do not. What is in the person, the social situation, personality, or early experiences that makes one vulnerable?

The absence of more discussion about depression following losses other than death was a disappointment. Chapter 14 shows promise of approaching this topic, but one chapter dealing primarily with the work of Brown and Harris on life events (1) is not sufficient.

Bowlby's theories are so eminently applicable to understanding clinical depression and the role of psychotherapy that it is disappointing these ideas are not developed in the remaining half of the book.

This book is a classic for understanding the grieving process in the light of human attachments. I hope it is followed by a fourth volume that will deal with clinical depression and the place of disrupted and disturbed attachments in its onset and vulnerability.

Western urban life, with its high divorce rate, geographic mobility, and differential mortality rate between men and women, provides rich opportunities for disrupted attachments, which may explain the high rate of depression, especially among women. Bowlby's theories, derived from ethology and ultimately rooted in biology, could link environmental triggers with biological disposition as a framework for understanding clinical depression. Bowlby's writings, as they stand, should be part of all behavioral science and medical school curricula.

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MYRNA M. WEISSMAN, PH.D.
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Progress in the Functional Psychoses, *edited by Robert Cancro, M.D., Lester E. Shapiro, M.D., and Martin Kesselman, M.D. New York, N.Y., SP Medical & Scientific Books (Spectrum Publications), 1979, 187 pp., $20.00.*

It is useful, from time to time, to have in one volume articles that summarize developments in a clinical field for the benefit of practitioners who are busy or who simply want a picture of the state of the art. This book is such a publication. The subjects covered are the two functional psychoses, schizophrenia and affective disorder.

The book has three editors; one writes a general introduction to the whole book, the second an introduction for the 8 chapters devoted to schizophrenia, and the third an introduction to the 5 chapters on affective disorder. Seventeen authors contribute 13 chapters. Each chapter has its own bibliography; the references mainly cover the past 10 years. A subject index for the whole volume is also included.

Past experience with books constructed like this one may give rise to an expectation of uneven writing, good and poor content, and a lack of transition from one chapter to another, but a pleasant surprise is in store for the reader. The book does not suffer from such negative expectations. Careful editing makes it quite the reverse. The chapters are uniformly good, they narrate recent progress, and they leave the reader with a feeling of having come abreast of the times.

Topics for discussion in the 8 chapters on schizophrenia are well chosen. These are the history of treatment, diagnosis, sociocultural features in etiology, patterns of research, psychopharmacological treatment, intrapsychic and family dynamics, brief hospitalization, and rehabilitation. The chapters on affective disorders concern the *DSM-III* classification of depression and affective disorders, the biological basis of affective disorders, clinical pharmacology and antidepressant drugs, lithium and manic-depressive disorders, and the current status of treatment of depressive disorders.

This book should be of value to the psychiatrist who wishes to get a modern review of the two major diagnostic categories presented. To those who want to organize for themselves a self-study course for continuing education credits, the text plus selected readings from the bibliographies at the end of the chapters should prove useful.

PAUL E. HUSTON, M.D.
Iowa City, Iowa

Handbook of Adolescent Psychology, *edited by Joseph Adelson. New York, N.Y., John Wiley & Sons, 1980, 597 pp., $39.95.*

Researchers in adolescent psychology, like students of other mental life and human behavior, are struggling to integrate recent important contributions from biological, social-historical, behavioral, and psychodynamic approaches. This collection of 18 chapters reflects that struggle. Instead of the simple exposition and evenhandedness one expects from a handbook format, the papers selected are relentlessly critical, and the reader is likely to come away with more questions than answers. If there ever was a commonly accepted body of knowledge about adolescence (there certainly is an overworked one), it is admittedly set aside here in favor of only *new* researches, and too often we are left with the authors to wallow in morasses of semidigested data and in endless methodological dilemmas. Often, however, when we yearn to make a conceptual leap or two to find some unity, if only in common sense or personal conviction, the admirable academic and scientific rigor displayed here restrains us again and again, saves us from the particular orthodoxy to which we are personally inclined.

The orthodoxy point for this text is a psychodynamic one. The first section, titled New Perspectives on Adolescence, is fundamentally a consideration of alternative motives for behavior, alternative to drive, conflict, and defensive motives. The initial chapter, "Adolescence in Historical Perspective," raises many sociological and historical questions in fundamentally defining adolescence and adolescent phenomenology. The second chapter, "Perspectives on Adolescence from Longitudinal Research," raises many questions about the behavioral forces and processes from childhood experience leading to and through adolescent developmental phenomena. Chapter three is Dr. Adelson's personal, direct, but affectionate attack on concepts of dynamic motivations of behavior. Well set out are the traditional ideas of Sigmund and Anna Freud and the less traditional but popular ideas of Blos and Kohut. Nonetheless, Dr. Adelson leaves us with this lament:

> It is hard to imagine a satisfying theory of adolescence without the strongest contribution from psychodynamic theory. No other approach can offer, potentially, a comparable depth and range of observation; no other approach is as well suited—again, potentially—to pull together evidence drawn from other sources. Yet it is even harder to imagine that happening today, given the state of the art. It is at the moment a fossilized doctrine, resisting innovation in method, unshakably parochial in outlook. The illusion of being up to date is occasionally provided by the attention (excessive, in our view) given to fashionable topics; the politically active young or new modes of pathology. That work is rarely illuminating, given an insular theory and tired, overemployed concepts—above all, the idea of regression, which is used as a universal solvent for all conceptual difficulties.

The title of chapter four, "The Biologic Approach to Adolescence: Biological Change and Psychological Adaptation," is misleading. The chapter actually deals only with pubertal biological changes and psychological adaptations to them. The author states that "no studies exist of the relation between endocrine and psychological changes in puberty." The "direct-effect" biologically based motivation model is given scant consideration. This will be very unsatisfying to the medically trained clinician and is a major gap in otherwise rigorous considerations, diminishing the conceptual presentations of this first part.

The second and largest section of the book, The Processes of Adolescence, presents thinly disguised discussions of some elements of the ego function and superego development. Noteworthy because they are very challenging and clinically useful are chapters on identity, ego development, thinking processes, and sexuality in adolescence (ego functions, always the forte of the psychologist). Less useful and more woolly are chapters on values, moral development, political thinking, friendship and peer groups, and strategic interactions in adolescence.

Part three, titled Variations in Adolescence, is a catchall of odds-and-ends issues stuck on at the end and only loosely related to the conceptualizations of the preceding parts. It begins with a chapter titled "Psychopathology in Adolescence." It covers schizophrenia, depression, suicidal behavior, problems of school attendance, and problems of school achievement in a very few pages and in simply too cursory a fashion. Brief chapters on the gifted adolescent, youth movements, and variations in adolescent psychohormonal development are all somewhat esoteric and interesting but also cursory. A single chapter on delinquency is an exception in this group. It is a comprehensive and useful discussion of this much overworked problem.

In sum, this is a reputable resource text for most of the major issues, and a few of the minor ones, confronting adolescent psychology today. It is primarily psychodynamic in spirit, with stress on research in the development of ego functions and aspects of superego specific to adolescence. Chapters vary in quality, utility, and readability, but all are at least rigorously scholarly.

PETER C. PLISHKA, M.D.
Bronx, N.Y.

Mental Retardation and Developmental Disabilities: An Annual Review, Vol. XI, *edited by Joseph Wortis, M.D. New York, N.Y., Brunner/Mazel, 1980, 240 pp., $17.50.*

The editor of this series has garnered many interesting and relevant contributions, ranging from topics of general interest (education, employment, psychometrics, genetics, psychiatry, nutrition, poverty, and retardation, to name a few) to reviews of obscure entities of interest to specialists in the area (congenital malformations).

The first section deals with items that might be relevant for anyone in the mental health field who deals with the developmentally disabled. The editor begins by briefly discussing "dyslexia, learning disability, brain damage, organicity and other non-entities." The chapter on clinical aspects is, unfortunately, not as clinical as the author's previous contributions in other volumes. It does attempt an overview of the current major issues in mental retardation, although the author is British and he discusses legislation, social services, and epidemiology as they stand in Great Britain. The epidemiology chapter is the best in the monograph. It is well written, well documented, and an excellent review of the better epidemiologic studies of retarded populations. Finally, the deinstitutionalization and community services chapter de-

scribes those services generally available for the retarded with some follow-up data detailing how various groups have fared in different placements.

The next section is much more technical. Previous editions have addressed the subjects of hydranencephaly, megalencephaly, and lissencephaly. There are two chapters in this volume on congenital malformations—one on hypertelorism, the other on arhinencephaly. Each describes these rare entities in exhaustive detail. On the other hand, the chapter on chromosomal syndromes is comprehensive and mercifully less detailed. The final section addresses developments in Southeast Asia.

An examination of previous editions of this series reveals that volume XI is neither as interesting nor as comprehensive as its predecessors. As such, it is likely that most psychiatrists will not find it useful except for reference purposes. The series, however, is a valuable one for those involved in this mental health subspecialty.

GABRIELLE A. CARLSON, M.D.
Los Angeles, Calif.

Artful Scribbles: The Significance of Children's Drawings, *by Howard Gardner. New York, N.Y., Basic Books, 1980, 274 pp., $15.00.*

This book begins on an aesthetic note and ends on a romantic one. The body of the book is another matter. The author declares himself to be an "inveterate development psychologist" and presents a series of chapters on scribbles, children's drawings as art, object copying, realism, and adolescent art. The author has read widely and selected some very interesting quotations from psychology and art criticism.

Dr. Gardner is a frequent contributor to *Psychology Today*. He has studied with the distinguished art critic Rudolph Arnheim, to whom the book is dedicated, and his citations from Arnheim are cogent statements of his teacher's Gestalt psychology. However, in Gardner's main sections there is a lot of material from his own children and an ingenuous style that seems aimed toward a lay audience. Even though he is listed as a staff member of a Veterans Administration hospital, the material is devoid of clinical awareness or much that would strike a psychiatrist as developmental. Childhood psychosis is described accurately but only to present the drawings of a child cut off from reality who shows artistic talent. Erikson is not mentioned. The work of Lauretta Bender on children's drawings is absent, and the rich field developed in the past 50 years on projective psychology is ignored. Harrower, Machover, Goudenough, Piotrowski, and standard psychological studies in drawings are unplumbed and absent from the index. Although Sir Ernst Gombrich is mentioned, his profound critical writings and his psychoanalytically sophisticated work with Ernst Kris are not mentioned. Gardner briefly discusses Jung's studies of the Mandala, including some intriguing material on the origins of symbols and basic graphic forms. He mentions how long Picasso worked to be able to draw like a child, but Picasso's drawings are not those of children.

Gardner opens with a quote of Malraux's view that a child may be artistic but cannot become an artist until he or she gains experience and can integrate his or her stored-up knowledge and mastery. He ends paraphrasing Rank on the need for an artist to have a rich feeling for life to produce significant art and notes that the seeds of artistic achievement are found in childhood. By choosing a naïve cognitive approach, the author has not advanced our knowledge. There are some fine insights from varied sources; there is no systematic or innovative integration of cognitive knowledge and a step backward in the deployment of knowledge about affective development. The sure and learned hand of psychologically sophisticated art critics—like Arnheim, Read, Wind, and Gombrich—is absent. The significance claimed in the book title is missed.

NORMAN R. BERNSTEIN, M.D.
Chicago, Ill.

BRIEF MENTION

The Psychiatric Clinics of North America, Vol. 2, Number 3, December 1979, *edited by Hagop Souren Akiskal, M.D. Philadelphia, Pa., W.B. Saunders Co., 1979, 212 pp., $17.00.*

The classification of affective disorders has been an important but unresolved issue for many years. Recent advances in both phenomenological and biological research have suggested the usefulness of several new categories and led to wide acceptance of the bipolar/unipolar subdivision. This volume provides a systematic review of these recent advances. Several chapters address the problem of schizoaffective disorder or atypical mania and depression, reviewing the evidence for treating these disorders as variants of schizophrenia or affective disorder or as an independent third illness. Other chapters examine the evidence that supports such new categories as the depression spectrum, hysteroid dysphoria, or the borderline concept. Although rapidly cycling bipolar illness and cyclothymic personality have been recognized for many years, they have not been studied through objective research until quite recently; this research is also carefully reviewed.

This volume should give readers an excellent summary of recent developments in the classification of affective disorders. Although the subject of classification provides the unifying theme of the volume, most of the chapters also provide a thorough discussion of work in neuropharmacology. Nearly all of the chapters have been written by prominent researchers who have been responsible for exploring and developing these new approaches to classification. As a consequence, the volume is well written, contains thorough documentation, and is fresh and up-to-date.

Anxiety and Emotions: Physiological Basis and Treatment, *by Desmond Kelly, M.D. Springfield, Ill., Charles C Thomas, 1980, 388 pp., $24.75.*

American psychiatrists often think of anxiety as rooted either in psychological conflict or in environmental stress. On the other hand, British psychiatrists have traditionally been interested in behavioral and biological aspects of anxiety. This volume, written by a British psychiatrist well known for his work on physical aspects of anxiety, provides a useful summary for clinicians who would like to update their knowledge on biological aspects of anxiety.

The volume begins with an introductory chapter on clinical aspects of anxiety that covers such issues as the classification of anxiety disorders and differential diagnosis. Subsequent chapters discuss the relationship between the cardiovascular system and anxiety, the role of the endocrine system, the lactate infusion test, and the impact of the limbic system in causing anxiety. A final chapter discusses treatment issues, and it ranges from psychotherapeutic approaches and transcendental meditation to beta-adrenergic blockers and monoamine oxidase inhibitors. In some chapters Kelly becomes too absorbed in basic research and wanders into rather arcane areas, and at times his documentation seems incomplete. On the whole, however, this book provides a useful and interesting summary of behavioral, neurological, genetic, and physiological aspects of anxiety.

N.C.A.

Short-Term Dynamic Psychotherapy: Evaluation and Technique, *by Peter E. Sifneos, M.D. New York, N.Y., Plenum Medical Book Co., 1979, 239 pp., $18.95.*

The short-term psychotherapist's lament is a familiar one. He or she is inevitably criticized for doing the very thing that makes brief therapy possible, namely, excluding many issues in order to focus on a few. The traditionalists ask how can this be treatment. Dr. Peter Sifneos, a pioneer in the field of focal treatment, adequately addresses this challenge in his second work on short-term anxiety-provoking psychotherapy. My own views are undoubtedly biased by the fact that Dr. Sifneos is both my teacher and colleague. However, I have tried as best as possible to judge whether his book conveys the effectiveness of his technique.

He explores in detail how to select patients and do the treatment. The patients who are chosen are highly motivated and have achieved mutuality of give and take in their relations with others. The therapeutic work must be organized around the specific applicability of oedipal dynamics to the patient's chief complaint. What are called "pregenital characterological issues," e.g., reactions to the therapist's absence, must not be explored and are quickly interpreted as regressive maneuvers to avoid the major oedipal theme. The therapist makes full use of the transference in order to make relevant links to the patient's past objects as well as to open up access roads to more material. The emphasis is always on giving patients a cognitive understanding of their dynamics that they may use when newer versions of their issues present themselves. Dr. Sifneos demands of himself that he see evidence of the effectiveness of this new learning on the patient's life before the treatment is regarded a success. From Dr. Sifneos' cogent and plentiful case examples and interview transcripts there can be little doubt that his is a very creative and helpful mode of intervention.

Dr. Sifneos' critics will question his emphasis on an oedipal focus and the complete avoidance of earlier issues. These restrictions in effect may unnecessarily limit the patients to whom short-term psychotherapy could be beneficial. Furthermore, some might be discomforted by Dr. Sifneos' apparent disparagement of the endeavors of long-term therapists, who must confront a greater panoply of issues, not all of them conflictual, over a lengthy period of time. The central point is not whether what is excluded is valuable. It is. However, Dr. Sifneos asks that the question be raised as to whether significant results can be achieved by applying a highlighted yet purposely limited concept of work. He demonstrates that they can.

The volume is very readable and clear. The richness of the many separate patient vignettes is supplemented by a superb presentation of an entire case, including all important interview segments, in the appendix. An outline of the research data offers objective testimony to the benefits of short-term anxiety-provoking psychotherapy. Dr. Sifneos' book is very valuable reading for everyone interested in dynamic psychotherapy of any kind.

GEORGE G. FISHMAN, M.D.
Boston, Mass.

Psychosomatic Obstetrics and Gynecology, *by David S. Youngs, M.D., and Anke A. Ehrhardt, Ph.D. New York, N.Y., Appleton-Century-Crofts, 1980, 287 pp., $18.50.*

This volume adds to the increasing number of books on emotional aspects of medicine that are addressed to the practitioners in various specialties. However, unlike books that stress clinical management, it consists of 18 chapters with the purpose of exposing the interested reader to behavioral science research as well as clinical experience of relevance to the practicing gynecologist and obstetrician. The chapters are grouped in five sections reflecting five main themes: 1) Critical, Biological, Social, and Cultural Influences During the Female Life Cycle, 2) Psychological Needs in the Doctor-Patient Relationship, 3) New Obstetric Techniques: Implications for the Patient, 4) The Adolescent Patient: Sexuality and Reproduction, and 5) Common Gynecological Perspectives. The chapters range in depth, sophistication, and focus.

Of the 18 authors, 4 are psychiatrists. The others are obstetricians and gynecologists, other medical specialists, psychologists, or researchers in other areas. Several chapters are primarily summaries of the inconclusive state of research in particular areas and consist of reports of studies. Examples of these are "Sex Hormone Administration During Pregnancy" and "Behavioral Sequelae in the Offspring," both by Meyer-Bahlburg and Ehrhardt, and "Emotional Parameters of Infertility" by Mozley. In contrast, other chapters are sensitive presentations of psychiatric-psychological issues, such as "Prenatal Genetic Counseling" by Jackson, "Reactions to Electronic Fetal Monitoring" by Starkman, and "Psychosocial Issues of Therapeutic Abortion" by Adler. "Psychosocial Aspects of Breast Cancer" by Kushner seems to be a superficial treatment of this important subject. "Elective Gynecologic Surgery" discusses these issues in a sensible and straightforward manner. In "Health Care for Women—A Prescription for Change" Elkin approaches important problems in the doctor-patient relationship from a historical as well as a critical perspective. The chapter on "Social and Emotional Aspects of Menstruation, Birth, and Menopause" by Parlee raises important methodological considerations affecting research in this entire field.

Combining these five main themes represented by the five sections into one book serves to bring to the attention of the reader approaching only one of these topics the work done in other areas and can thus stimulate interest in unexpected directions.

Although the psychosomatic focus promised by the title is not entirely adhered to and the chapters vary in clinical application and depth on the whole, the book does extend the reader's understanding of emotional aspects of reproductive functioning. It avoids sexism, offers many interesting observations, and covers issues of importance to the consultation-liaison psychiatrist as well as to the gynecologist for whom it is presented. There are few books of comparable quality in this field.

MALKAH T. NOTMAN, M.D.
Boston, Mass.

Psychiatric Consultation in Mental Retardation. Publication 104, *by the Group for the Advancement of Psychiatry Committee on Mental Retardation. New York, N.Y., GAP, 104 pp., $4.00 (paper).*

This small book presents in very brief form the various types of consultation regarding mental retardation that the psychiatrist might face in private or community practice. The appendix furnishes a convenient classification of types of consultation, including direct case evaluation and therapy and indirect consultation through agencies whose programs serve the mentally retarded and their families.

The discussions do not demand a high level of knowledge of the field; indeed, in-depth discussions of the complexities of pathologies that are encountered more often than not are notably lacking. In spite of this, the text has an aura of condescension about it; for example: "In order to be useful, consultants must know something more about the special area in which they are consulting than the person requesting help" (p. 601). If they do not, and they accept the consultation, somebody ought to call the ethics committee.

The book is very elementary in its approach. Perhaps it may help nonprofessional workers to use consultation better. One hopes psychiatrists are considerably beyond this level of education.

PAUL V. LEMKAU, M.D.
Baltimore, Md.

Countertransference, *edited by Lawrence Epstein, Ph.D., and Arthur H. Feiner, Ph.D., for the William Alanson White Psychoanalytic Society. New York, N.Y., Jason Aronson, 1979, 467 pp., $30.00.*

This book brings together 18 contributions by 17 authors. The chapters are grouped in three divisions—The Concept of Countertransference, The Therapist's Use of Countertransference, and Countertransference with Particular Types of Patients. A useful introduction by the editors succinctly describes the various ways countertransference can be defined and traces the historical development of these different points of view. Overall the book contains more ideas about countertransference in the context of treating serious disorders than it does ideas that developed in treating the neuroses. As such, countertransference as a potential instrument for understanding and treating patients receives strong emphasis, and countertransference as a potential impediment receives less attention.

The qualities of the chapters range from high levels of conceptual effectiveness (e.g., Langs) and excellent sharing of clinical experience and wisdom (e.g., McDougall and Searles) to worthy clinical essays (e.g., Feiner) to, in my opinion, some chapters in which ideas are sparse or loosely presented or idiosyncratic and unhelpful.

The book is enriched by the individualistic approaches of the various authors. However, this involves the sacrifice of cohesiveness, and further review of the contents of the book can be provided only in the form of a series of comments about certain chapters.

Langs offers his well-conceived interactional framework for delineating types of countertransference. His is a balanced, densely packed, rigorous presentation of countertransferences that interfere with treatment. It will contribute substantially to the theoretical and practical clinical understanding of many readers. Some will also be stimulated to read his broader body of work if they have not already.

Wittenberg writes from an orientation that places the highest value on the achievement of object relatedness as opposed to the value placed by other authors on narcissistic achievements. He stresses the fundamental importance of the allocentric attitude in the therapist. That is, the therapist must emancipate himself or herself from the gratification of his or her own needs in order to perceive the patient continuously altogether in the patient's own right. The therapist maintains full interest, openness, and receptivity in relation to the patient. Anything other than an allocentric attitude is considered by Wittenberg to be a countertransference that interferes with treatment and must be corrected by self-analysis.

Feiner's rather poetic essay concerns an important but seldom considered problem. Patients under the sway of transference ascribe qualities to the therapist that run counter to his or her actual unique qualities as a person and tend to influence the therapist to assume an identity that corresponds to the transference. The analyst naturally objects to this influence and must be alert for countertransference defensiveness against it. His or her professional identity as an analytic self provides the resources for avoiding and correcting this countertransference.

From a Kleinian perspective Grinberg describes the phenomenon of projective identification, then delineates a kind of countertransference response to it that he calls projective countertransference. This consists of an unconscious response in the therapist that is independent of his or her own emotional life; it is derived as a reaction to the projective identification the patient directs onto the therapist.

Epstein presents the ways in which the skillful use of countertransference hate can benefit therapy. Searles shares fine clinical accounts demonstrating phenomena of jealousy in primitive patients and the parallel countertransference jealousies experienced by therapists who undertake to treat them. Giovacchini, in an engaging clinical chapter, describes the "counter-delusional impact" in the therapist that occurs when a patient's delusions become all-pervasive in the analysis. The impact on the analyst derives from his or her responding in such a way as to lose the analytic stance, with resultant threat to his or her analytic identity. The analytic stance can be restored 1) by confronting the patient with the need to conform with those requirements which enable the analyst to continue functioning as analyst or 2) by the patient's confronting the analyst in such a way as to enable the analyst to resume his calm, effective analytic attitude.

McDougall's richly rewarding clinical chapter concerns

work with patients who have suffered major trauma before they acquired the capacity for verbal communication. Breakdown in communication with the mother in infancy would be one form of such trauma. These patients defend most vigorously against any conscious awareness of painful derivative expressions of the trauma. The therapist may become aware of the trauma through the way it is expressed in the patient's modes of being and speaking rather than through what the patient actually says. The therapist's countertransference reactions to the patient's activity may be the only way to gain awareness of the trauma. Such countertransference may be purely useful, but it can also be deleterious.

Boyer presents good clinical examples of using countertransference responses as means of understanding severely regressed patients. Epstein writes from a Kleinian orientation about countertransferences as clues about the projected aspects of the patient's intrapsychic relations between self and objects. Wolf presents a summary of Kohut's understanding of countertransference in the setting of self-psychology.

DAN H. BUIE, M.D.
Boston, Mass.

This Month's Reviewers

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Books Received

This list acknowledges the receipt of recent books. Books of particular interest to the readers of this journal will be reviewed as space permits, and copies of the reviews will be sent to the publishers. Books cannot be returned to the publishers.

The Community Imperative: Proceedings of a National Conference on Overcoming Public Opposition to Community Care for the Mentally Ill, *edited by Richard C. Baron, Irvin D. Rutman, Ph.D., and Barbara Klaczynska, Ph.D. Philadelphia, Pa., Horizon House Institute for Research and Development, 1980, 474 pp., $10.00 (paper).*

Neurochemistry and Clinical Neurology: Progress in Clinical and Biological Research, Vol. 39, *edited by Leontino Battistin, George Hashim, and Abel Lajtha. New York, N.Y., Alan R. Liss, 1980, 492 pp., $38.00.*

Workshops on Cognitive Processes, *by A. Bennett, S. Hausfeld, R.A. Reeve, and J. Smith. Boston, Mass., Routledge & Kegan Paul, 1980, 120 pp., $9.75 (paper).*

Aging, Isolation and Resocialization, *edited by Ruth Bennett, Ph.D. New York, N.Y., Van Nostrand Reinhold Co., 1980, 207 pp., $14.95.*

Advances in Human Psychopharmacology: A Research Annual, Vol. 1, 1980, *edited by Graham D. Burrows and John S. Werry. Greenwich, Conn., Jai Press, 1980, 329 pp., $37.50.*

The Thinking Skills Workbook: A Cognitive Skills Remediation Manual for Adults, *by Lynn Tondat Carter, Ph.D., John L. Caruso, Ph.D., Mary A. Languirand, and M.A. Berard, M.D. Springfield, Ill., Charles C Thomas, 1980, 210 pp., $13.95 (paper).*

Teaching Social Skills to Children: Innovative Approaches, *edited by Gwendolyn Cartledge and JoAnne Fellows Milburn. New York, N.Y., Pergamon Press, 1980, 306 pp., $25.00; $9.95 (paper).*

Harry Stack Sullivan's Concepts of Personality Development and Psychiatric Illness, *by A.H. Chapman, M.D., and Miriam C.M.S. Chapman, M.D. New York, N.Y., Brunner/Mazel, 1980, 193 pp., no price listed.*

The Skills of Community Service Coordination, Psychiatric Rehabilitation Practice Series: Book 6, *by Mikal R. Cohen, Ph.D., Raphael L. Vitalo, Ph.D., William A. Anthony, Ph.D., and Richard M. Pierce, Ph.D. Baltimore, Md., University Park Press, 1980, 113 pp., $7.50 (paper).*

Practice of Geriatric Psychiatry, *by Alex Comfort. New York, N.Y., Elsevier, 1980, 105 pp., $15.95.*

Approaches to Insanity: A Philosophical and Sociological Study, *by Jeff Coulter. Totowa, N.J., Martin Robertson, 1980, 163 pp., $22.50; $7.95 (paper).*

Psychiatric Nursing: A Basic Manual, 5th ed., *by Annie Laurie Crawford, M.Ed., R.N., and Virginia Curry Kilander, M.Ed., R.N. Philadelphia, Pa., F.A. Davis Co., 1980, 140 pp., $7.25 (paper).*

Birth and Fortune: The Impact of Numbers on Personal Welfare, *by Richard A. Easterlin. New York, N.Y., Basic Books, 1980, 195 pp., $11.95.*

Burn-Out: Stages of Disillusionment in the Helping Professions, *by Jerry Edelwich, M.S.W., with Archie Brodsky. New York, N.Y., Human Sciences Press, 1980, 252 pp., no price listed.*

The Suicide Syndrome, *edited by Richard Farmer and Steven Hirsch. London, England, Croom Helm, 1980, 264 pp., $37.50.*

The Year Book of Psychiatry and Applied Mental Health, 1980, *edited by Daniel X. Freedman, M.D., Arnold J. Friedhoff, M.D., Lawrence C. Kolb, M.D., Reginald S. Lourie, M.D., Sc.D., John C. Nemiah, M.D., and John Romano, M.D. Chicago, Ill., Year Book Medical Publishers, 1980, 358 pp., $37.50.*

A Method of Psychiatry, *edited by Stanley E. Greben, M.D., Robert Pos, M.D., Vivian M. Rakoff, M.B., B.S., Alexander Bonkalo, M.D., Frederick H. Lowy, M.D., and George Voineskos, M.D. Philadelphia, Pa., Lea & Febiger, 1980, 357 pp., $20.00.*

Intelligence and Adaptation, *by Stanley I. Greenspan. New York, N.Y., International Universities Press, 1980, 398 pp., $22.50.*

Sigmund Freud's Dreams, *by Alexander Grinstein; M.D. New York, N.Y., International Universities Press, 1980, 486 pp., $22.50.*

A Concordance to the Standard Edition of the Complete Psychological Works of Sigmund Freud, *edited by Samuel A. Guttman, Ph.D., M.D., Randell L. Jones, Ph.D., and Stephen M. Parrish, Ph.D. Boston, Mass., G.K. Hall, 1980, $525.00 (set of 6 books).*

Law in the Practice of Psychiatry: A Handbook for Clinicians, *by Seymour L. Halleck, M.D. New York, N.Y., Plenum Medical Book Co., 1980, 284 pp., $21.50.*

Human Judgment and Decision Making: Theories, Methods, and Procedures, *by Kenneth R. Hammond, Gary H. McClelland, and Jeryl Mumpower. Washington, D.C., Hemisphere Publishing Corp., 1980, 244 pp., $22.50.*

A Society Under Stress: Children and Young People in Northern Ireland, *by Jeremy and Joan Harbison. Somerset, England, Open Books (New York, N.Y., Brunner/Mazel, distributors), 1980, 189 pp., $20.00.*

The Anatomy of Loneliness, *edited by Joseph Hartog, M.D., J. Ralph Audy, Ph.D., M.D., and Yehudi A. Cohen, Ph.D. New York, N.Y., International Universities Press, 1980, 603 pp., $30.00.*

Group Psychotherapy: A Peer Focused Approach, *by Richard A. Hogan. New York, N.Y., Holt, Rinehart and Winston, 1980, 280 pp., $21.95.*

Pain and Society: Report of the Dahlem Workshop on Pain and Society, Berlin, Nov. 26–30, 1979, *edited by H.W. Kosterlitz and L.Y. Terenius. Deerfield Beach, Fla., Verlag Chemie, 1980, 511 pp., $39.40 (paper).*

Proceedings of the International Year of the Child Conference, Child Advocacy 1979, *edited by Susan Lustman Katz. New Haven, Conn., Yale Child Study Center, 1980, 383 pp., $8.95 (paper).*

Responsible Parenthood: The Child's Psyche Through the Six-Year Pregnancy, *by Gilbert W. Kliman, M.D., and Albert Rosenfeld. New York, N.Y., Holt, Rinehart and Winston, 1980, 334 pp., $14.95.*

The Age of Structuralism: Lévi-Strauss to Foucault, *by Edith Kurzweil. New York, N.Y., Columbia University Press, 1980, 246 pp., $20.00; $5.95 (paper).*

Theories on Drug Abuse: Selected Contemporary Perspectives. NIDA Research Monograph 30, *edited by Dan J. Lettieri, Ph.D., Mollie Sayers, and Helen Wallenstein Pearson. Rockville, Md., National Institute on Drug Abuse, 1980, 488 pp., $8.50 (paper).*

Higher Cortical Functions in Man, 2nd ed., *by Aleksandr Romanovich Luria. New York, N.Y., Basic Books, 1980, 625 pp., $27.50.*

From Borderline Adolescent to Functioning Adult: The Test of Time, *by James F. Masterson, M.D., with Jacinta Lu Costello, M.S.W. New York, N.Y., Brunner/Mazel, 1980, 291 pp., $19.50.*

People and Cultures of Hawaii: A Psychocultural Profile, *edited by John F. McDermott, Jr., Wen-Shing Tseng, and Thomas W. Maretzki. Honolulu, Hawaii, University Press of Hawaii, 1980, 234 pp., $7.50 (paper).*

Stress and Tension Control, *edited by F.J. McGuigan, Wesley E. Sime, and J. Macdonald Wallace. New York, N.Y., Plenum Press, 1980, 311 pp., $29.50.*

Who Is the Client? The Ethics of Psychological Intervention in the Criminal Justice System, *edited by John Monahan. Washington, D.C., American Psychological Association, 1980, 168 pp., $7.50 (paper).*

Individual and Environment: Monozygotic Twins Reared Apart, *by Niels Juel-Nielson. New York, N.Y., International Universities Press, 1980, 292 pp., $22.50.*

Choice and Change: The Psychology of Adjustment, Growth, and Creativity, revised ed., *by April O'Connell and Vincent F. O'Connell. Englewood Cliffs, N.J., Prentice-Hall, 1980, 531 pp., $12.95 (paper).*

The Rehabilitation of the Mentally Ill in the 1980s: A Report of the Fourth Mary E. Switzer Memorial Seminar, Feb. 1980, *edited by Leonard G. Perlman, Ed.D. Washington, D.C., National Rehabilitation Association, 1980, 126 pp., $4.00 (paper).*

The Skills of Career Counseling: Psychiatric Rehabilitation Practice Series, Book 4, *by Richard M. Pierce, Ph.D., Mikal R. Cohen, Ph.D., William A. Anthony, Ph.D., Barry F. Cohen, Ph.D., and Theodore W. Friel, Ph.D. Baltimore, Md., University Park Press, 1980, 96 pp., $6.95 (paper).*

The Skills of Career Placement: Psychiatric Rehabilitation Practice Series, Book 5, *by Richard M. Pierce, Ph.D., Mikal R. Cohen, Ph.D., William A. Anthony, Ph.D., Barry F. Cohen, Ph.D., and Theodore W. Friel, Ph.D. Baltimore, Md., University Park Press, 1980, 98 pp., $6.95 (paper).*

The Diagnosis of Stupor and Coma, 3rd ed., *by Fred Plum, M.D., and Jerome B. Posner, M.D. Philadelphia, Pa., F.A. Davis Co., 1980, 364 pp., $22.00.*

Effective Counseling of Adolescents, *by Helen G. Rabichow and Morris A. Slansky, M.D. Chicago, Ill., Follett Publishing Co., 1980, 199 pp., $10.95.*

The Quiet Therapies: Japanese Pathways to Personal Growth, *by David K. Reynolds. Honolulu, Hawaii, University Press of Hawaii, 1980, 135 pp., $8.95.*

Psychology and Law: Can Justice Survive the Social Sciences? *by Daniel N. Robinson. New York, N.Y., Oxford University Press, 1980, 216 pp., $14.95; $5.95 (paper).*

Sex Education for the Health Professional: A Curriculum Guide, *edited by Norman Rosenzweig, M.D., and F. Paul Pearsall, Ph.D. New York, N.Y., Grune & Stratton, 1980, 327 pp., no price listed.*

OFFICIAL ACTIONS

Position Statement on Medical Participation in Capital Punishment

This statement was approved by the Board of Trustees at its June 20, 1980, meeting, upon recommendation of the Assembly of District Branches, which approved the statement during its May 2-4, 1980, meeting.

SINCE 1977 four states (Oklahoma, Texas, Idaho, and New Mexico) have adopted legislation providing for capital punishment by drug injection. Other states have similar legislation under consideration. Oklahoma law specifically calls for intravenous injection of a barbiturate and a paralytic agent. The physician's role varies from ordering and preparing the substance, injecting or supervising the injection of the drug, and/or monitoring the procedure and the prisoner. It is said that the procedure is "more humane," will encourage the death penalty, will not be regarded as "cruel and unusual punishment," and will be cost effective.

Historically, physicians have participated in capital punishment, and in this century heinous crimes by physicians causing death and torture have been documented in Germany, Russia, and other countries. In this regard, some psychiatrists were notoriously cooperative with the official aims of Nazi Germany. The Declaration of Tokyo stated that doctors shall not condone or participate in any inhumane or degrading procedures, no matter what the offense of the person, and shall not provide the instruments, knowledge, or substances to facilitate "cruel, inhumane, or degrading treatment, or diminish the ability of the victim to resist such treatment." Thus, active medical participation in capital punishment would violate those principles. The physician's serving the state as an executioner, either directly or indirectly, is a perversion of medical ethics and of his or her role as healer and comforter.

APA therefore strongly opposes any participation by psychiatrists in capital punishment, that is, in activities leading directly or indirectly to the death of a condemned person as a legitimate medical procedure.

Position Statement on Teenage Parents and Their Children

This statement was approved by the Assembly of District Branches at its May 2-4, 1980, meeting and by the Board of Trustees at its June 20, 1980, meeting. It was prepared by the Task Force on Teenage Parents and Their Children and the Council on Children, Adolescents and Their Families.[1]

THE TASK FORCE has reviewed the existing models of service to teenage parents and their children, the rationale underlying such programs, and their funding. Teenage pregnancy and parenthood are increasing at a rapid rate, affecting a high-risk population of adolescents and their offspring physically, socially, and psychologically. Adolescents under 15 years of age are at greatest risk, for example, with increased rates of eclampsia and low infant birth-weight. Data collection and service programs tend to group all teenage parents together, failing to recognize the different developmental needs and characteristics of the younger, middle, and older teenager. Sound service programming must allow for the distinctiveness of each adolescent phase.

Many deficiencies in services can be traced to current funding patterns and policies. Fragmentary, short-term funding undermines the continuity and longitudinal support services required by teenage parents and their infants. The total amount of local, private, and federal support is grossly inadequate for suitable programming.

A model for effective programs would incorporate the following elements: 1) provision of service to adolescent parents, their offspring, and their own families, with outreach capability; 2) when possible, availability of a child or adolescent psychiatrist serving in a clinical role; 3) availability of a primary worker who would relate to each adolescent mother and child on a continuing basis, following them through the child's preschool stages of development; and 4) liaison with appropriate facilities to meet the educational, vocational, medical, and psychological needs of adolescent parents and their families.

In the interest of a pressing public health and mental health issue, the American Psychiatric Association:

1. Urges measures to make the public, legislators, funding resources, and service providers more aware of the need for adequate programs to reduce serious *psychological, social, and physical risks associated with teenage parents and their children.*
2. Affirms the need for comprehensive, multidisciplinary, and continuous services to the parent-child unit, from the prenatal period through the child's preschool development, *including prevention.*
3. Emphasizes that appropriate services be extended not only to the teenage parent-child unit but also to the families of each of the teenage parents.
4. Recommends that statistics on teenage pregnancies be broken down to identify younger, middle, and older teenagers and that appropriate service programs be designed for each group.
5. Stresses that child and adolescent psychiatrists should play a central role in assessing service needs and participating in programs for these teenagers and their children.
6. Supports adequate funding of federal programs relating to these issues.

[1]The Task Force on Teenage Parents and Their Children included Jo Ann B. Fineman, M.D., chairperson, Taghi Modarressi, M.D., Carol A. Leal, M.D., John B. Reinhart, M.D., Max Sugar, M.D., Carlos H. Salguero, M.D., and Ruth W. Lidz, M.D., consultant. The Council on Children, Adolescents and Their Families included Edward H. Futterman, M.D., chairperson, Leonard Lawrence, M.D., Viola Bernard, M.D., John F. McDermott, M.D., Silvio J. Onesti, M.D., Nellie L. Mitchell, M.D., observer-consultant, Herbert S. Sacks, M.D., Henry B. Brackin, Jr., M.D., Frank Rafferty, M.D., consultant, and Jeanne Spurlock, M.D., staff liaison.

Guidelines for Psychiatrists in Consultative, Supervisory, or Collaborative Relationships with Nonmedical Therapists

The following guidelines were approved by the Assembly of District Branches at its May 2-4, 1980, meeting and by the Board of Trustees at its June 20, 1980, meeting with the understanding that they do not represent official policy but rather a "living document" to be adapted to local custom and practice.

THE PRACTICE OF PSYCHIATRY and of other mental health disciplines increasingly occurs in the framework of organized health delivery systems. Psychiatrists are working with other professionals and nonprofessionals in hospital settings, community mental health centers, health maintenance organizations, as well as in group practices, and in consultative work with schools, family agencies, court clinics, etc. Interprofessional relationships are an essential aspect of good patient care and should be encouraged. They serve as a valuable educational experience and contribute to the continuing development of all who are concerned with patient care. The addition of other professionals and extenders to the health team enlarges the capacity to provide service. In turn, this requires a review of the role and responsibilities of psychiatrists in the entire range of consultative, supervisory, and collaborative relationships.

In December 1972 the Board of Trustees adopted and later published a Position Statement on Psychiatrists' Relationships with Nonmedical Mental Health Professionals (1). This statement reaffirmed the objectives of psychiatry in regard to relationships with nonmedical mental health professionals. (In 1977 these objectives were constitutionally amended by the Board of Trustees to include item f.)

OBJECTIVES OF THE PROFESSION OF PSYCHIATRY

The American Psychiatric Association, in its Constitution, states the following to be its objectives:

(a) to improve the treatment, rehabilitation, and care of the mentally ill, the mentally retarded, and the emotionally disturbed; (b) to promote research, professional education in psychiatry and allied fields, and the prevention of psychiatric disability; (c) to advance the standards of all psychiatric services and facilities; (d) to foster the cooperation of all who are concerned with the medical, psychological, social, and legal aspects of mental health and illness; (e) to make psychiatric knowledge available to other practitioners of medicine, to scientists in other fields of knowledge, and to the public; and (f) to promote the best interests of patients and those actually or potentially making use of mental health services.

The psychiatrist is a physician whose work constantly involves the differential diagnosis of disease and disorder and whose practice is concerned with elucidating and addressing the complex intertwined relationships of the physical and emotional aspects of diseases and disorders. In this work with patients the psychiatrist continually monitors, reassesses, and refines diagnoses and pursues therapeutic aims, using a wide range of treatment modalities, including psychotherapies, medications, and other somatic treatments and, at times, hospital care.

The 1973 position statement defined the psychiatrist's responsibilities to other professions:

Psychiatry's Responsibilities to Other Professions

Definition and delineation of interprofessional relationships. To recognize that every professional group has a right to establish and maintain its identity and independence by defining its own functions and areas of competence, setting up its own educational and training programs, and establishing its own standards of service. As a responsibility to the public, society may recognize the profession and set up such controls as it may deem necessary. No profession should attempt to define the functions and responsibilities of any other profession.

Education and Training. The responsibilities of psychiatry include the following:

- To inform members of other professions regarding training, experience, areas of special competence, and appropriate spheres of activity of the psychiatrist.
- To assist when invited in the education and training of other professionals in any areas in which psychiatry has a contribution to make, and on subjects within the scope of those other professions as they may be legally defined.
- To encourage and foster ongoing mutual education programs through joint meetings, seminars, and workshops with other professional organizations.

Standards of service. To work in close cooperation with other professionals when requested to do so in order to utilize the expertise of all available professions in the provision of high standards of service to the public.

Ethics. To recognize that the establishment and maintenance of codes of ethics are an internal responsibility of each profession and that complaints against other professionals should be directed to the responsible authorities of the profession concerned.

Manpower, research, and prevention. To collaborate with other professions in these important areas.

Liaison responsibilities. To recognize such responsibilities, the American Psychiatric Association offers the following guidelines in reference to the settings and situations in which problems in interprofessional relations often arise:

- In medical settings: The American Psychiatric As-

sociation, in concurrence with the American Hospital Association, endorses the principle of appointing members of other professions such as nurses, dentists, social workers, sociologists, ministers, and psychologists to the staff of hospital and other facilities to bring to the treatment of patients their specialized knowledge, skill, and experience. In such a setting the physician-psychiatrist retains the primary medical responsibility, established by law and custom, for the admission, diagnosis, treatment, rehabilitation, and discharge of patients.

- In nonmedical settings: Psychiatrists function increasingly in many settings such as mental retardation centers, regular and special education schools, correctional institutions, social service agencies, and others that do not have a primarily medical orientation. A psychiatrist working as a consultant, supervisor, therapist, or administrative staff member in such a setting has essentially the same relationship to the organization as other professionals have in a medical setting. However, he must retain ultimate responsibility for the psychiatric and medical care of the patients or clients whom he serves.
- In office practice: The psychiatrist recognizes the special skills and competencies of other professionals and sends or refers people to them for appropriate services, based on the other professionals' education, training, experience, competence, and reputation.
- Third-party payments: Psychiatry endorses payment under medical insurance plans such as Medicaid or Medicare to nonmedical health professionals when their services are rendered as part of a plan of treatment that is supervised and/or prescribed by a physician. However, professionals in various allied fields often elect to engage independently in health-related practices in a nonmedical setting. A person should feel free to select the practitioner of his choice from among qualified professionals. If there is a public demand for health-related services that are not normally regarded as medical or offered as part of a total medical treatment program, insurance companies may offer insurance for such health-related services as they currently do for medical treatment. If the federal government wishes to provide publicly supported programs for such services, appropriate legislation will have to be passed.
- Legal responsibility: When psychiatrists extend their services to other health professionals in meaningful collaboration (which includes supervision of cases or participation in interdisciplinary teamwork), they are obliged to know about and to be willing to assume the established legal responsibilities involved."

Since the publication of this document, psychiatrists have raised questions about the nature of their working relationships with members of the nonmedical mental health professions and extenders. In 1976 the Board of Trustees adopted this statement: "The Board of Trustees, in recognition of the need of psychiatric patients to have the total care that is provided only through a physician trained in psychiatry, strongly reaffirmed its conviction that the treatment of psychiatric patients within a hospital setting must be under the supervision of a physician."

The appropriate use of consultative, supervisory, and collaborative relationships is basic to good patient care. These guidelines are not in any way an attempt to define the roles and conditions of practice that exist in other disciplines and should not be interpreted or used for that purpose.

1. *Consultative relationship between a psychiatrist and a therapist who is not a physician.* In this type of relationship the psychiatrist does not assume responsibility for the patient's care. The psychiatrist evaluates the information provided by the therapist and offers a medical opinion which the therapist may or may not accept. In this instance the therapist must have met professional and institutional standards for independent functioning (e.g., certification). Consultation is not a one-way process and psychiatrists do and should seek appropriate consultation from members of other disciplines in order to provide more comprehensive services to patients (see sections 4 and 5).

2. *Supervisory relationships between a psychiatrist and a nonmedical therapist.* In a supervisory relationship the psychiatrist retains direct responsibility for patient care and gives professional direction and active guidance to the therapist. In this relationship the nonmedical therapist may be an employee of an organized health-care setting or of the psychiatrist. The psychiatrist is clinically responsible for the initial workup, diagnosis, and prescription of a treatment plan, as well as for assuring that adequate and timely attention is paid to the patient's physical status and that such information is integrated into the overall evaluation, diagnosis, and planning. The psychiatrist remains ethically and medically responsible for the patient's care as long as the treatment continues under his or her supervision. The patient should be fully informed of the existence and nature of, and any changes in, the supervisory relationship.

3. *Collaborative relationship between a psychiatrist and a therapist who is not a physician.* Implicit in this relationship is mutually shared responsibility for the patient's care in accordance with the qualifications and limitations of each therapist's discipline and abilities. The patient must be informed of the respective responsibilities of each therapist; neither discipline's responsibilities diminish those of the other. Both the psychiatrist and nonmedical therapist are responsible for periodic evaluation of the patient's status to ascertain that the collaboration continues to be appropriate. The therapists must inform the patient, either jointly or separately, if they decide to terminate their collaborative relationship.

4. *Appropriateness of care.* It is unethical for the psychiatrist to continue an administrative supervisory, consultative, or collaborative relationship if it is ascertained that his or her role is being misrepresented, or when he or she is convinced that the care being provided is inappropriate or inadequate. An exception may be made when the psychiatrist is specifically accepting a responsibility with the goal of raising the quality of care to an adequate level. Psychiatrists with ongoing administrative supervisory, consultative, or collaborative relationships should undertake such relationships with a therapist only if they are able to keep themselves appropriately informed of the nature of treatment and the progress of those patients for whom they are acting as supervisor, consultant, or collaborator, and can assure themselves that the treatment is being carried out competently and adequately.

In some institutional settings nonpsychiatric professionals or mental health workers may be supervised by a senior nonpsychiatric mental health worker of the same discipline. The psychiatrist must function in this system as if the relationship were a collaborative or supervisory one. In no circumstances can the relationship be defined as collaborative if the other treating professional has not met professional and in-

stitutional standards for independent functioning.

5. *Frequency of supervision, consultation, and collaboration.* Because of the wide range of competence and training that currently exists among nonphysician therapists, it is difficult to specify precisely what an optimum number and frequency of supervision, consultation, or collaboration contacts between a psychiatrist and a nonphysician therapist should be. However, it is incumbent upon the psychiatrists to satisfy themselves as to the competence, level of training, and, where required, licensure of the therapist. They must then provide an amount of supervision, consultation, or collaboration sufficient to assure that their ethical, medical, and legal responsibilities toward the patient are met and are consistent with any local or state guidelines outlining these responsibilities.

6. *Billing practices for psychiatrists.* A psychiatrist's bill should reflect the services actually rendered. Psychiatrists should not bill a patient, insurance company, or other third-party payer for a service that they do not personally provide directly to the patient, except in circumstances noted in sections (a) and (b) below. (This does not apply to direct consultations provided to agencies and institutions.)

a) When a psychiatrist employs a nonphysician mental health therapist on a salary or fee-for-service basis, the patient or third-party payer may be billed on a psychiatrist's billhead. However, the bill should note the name of the therapist actually providing the service, his or her training (Ph.D., M.S.W., R.N., Mental Health Counselor), number of visits, the rate per visit, and the total charges.

b) In some circumstances psychiatrists may bill for their own time spent in behalf of the treatment of the patient, but not actually with the patient. Such circumstances include extensive time spent in discussing past treatment with other physicians or mental health therapists and occasional necessary meetings with therapists of important individuals in a patient's life. Such charges should usually be made after first informing the patient. Psychiatrists should keep careful, detailed records of all billed contacts in accordance with the APA and AMA guidelines.

c) Except as noted in section (a) above, psychiatrists should not bill patients, third-party carriers, or others in their own names for services that they do not provide, nor should psychiatrists allow their names to be used to imply that they have provided therapy that they have not actually provided. Rather, nonphysician mental health therapists should bill the patient, insurance company, or other third-party payer in their own names for their own services at whatever rate they have agreed on with the patient, using their own billhead.

d) When psychiatrists personally interview patients, as part of their supervisory, consultative, or collaborative responsibilities, they may bill the patient, insurance company, or other third-party payer for such services on their own billhead, but only at the rate which corresponds to the time or service they actually provided to the patient.

e) Except as cited in sections (a) and (b) above, psychiatrists may bill only for the time they actually spend with the patient. Financial reimbursement of psychiatrists for consultative and/or supervisory time provided directly to the nonphysician mental health therapist, and which did not involve seeing the patient, is up to individual arrangement between the psychiatrist and the nonphysician mental health therapist. This reimbursement should be based on the actual amount of time spent on supervision or consultation, plus appropriate administrative costs such as rental office space and sharing of secretarial services. An arrangement in which the psychiatrist is reimbursed by the nonphysician therapist with a percentage of the latter's fees or gross income is not acceptable; this could constitute fee-splitting. Under no circumstance should a psychiatrist be compensated directly or indirectly for the referral of a patient to a nonphysician therapist. The mutually agreed upon set fee or salary should be open to renegotiation when a change in the time demand occurs. It is recommended that these arrangements be made in writing, specifying the obligations of both parties. Third parties may be notified of such a supervisory relationship when documentation is required for the nonphysician therapist to receive payment.

REFERENCE

1. Position statement on psychiatrists: relationships with nonmedical mental health professionals. Am J Psychiatry 130:386–390, 1973

The American Board of Psychiatry and Neurology

The following successfully completed the Board examination given in Milwaukee, WI, June 30-July 1, 1980.

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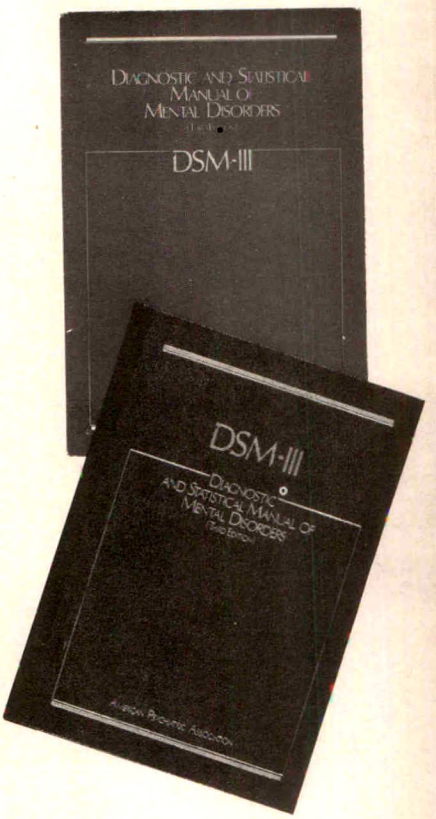
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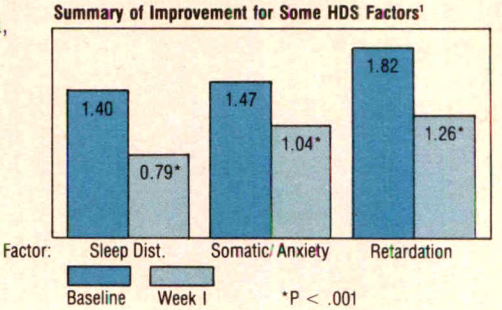


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Depressed patients often present with fatigue, anorexia, anxiety, mood swings, loss of interest, *and most frequently, early awakening and insomnia*. In a summation of 9 clinical studies involving 198 patients, significant clinical improvement was noted after the first week of doxepin treatment.[1] However, optimal antidepressant response may not be evident for two to three weeks.

Summary of Improvement for Some HDS Factors[1]



Normalized sleep patterns

In a study of clinically depressed patients with symptoms of insomnia, treatment with doxepin significantly reduced difficulty falling asleep, difficulty staying asleep and frequency of early morning awakening.[2]

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The incidence of tachycardia and hypotension, which have been observed during treatment with tricyclic antidepressants, has been reported infrequently with doxepin. In 452 patients given doxepin, the incidence of tachycardia was 2.88%; in 495 patients, the incidence of hypotension was 2.62%.[3]

Now available... 75 mg capsules for h.s. dosage

Brief Summary of Prescribing Information ADAPIN® (doxepin HCl) Capsules

Indications–Relief of symptoms of anxiety and depression.

Contraindications–Glaucoma, tendency toward urinary retention or hypersensitivity to doxepin.

Warnings–Adapin has not been evaluated for safety in pregnancy. No evidence of harm to the animal fetus has been shown in reproductive studies. There are no data concerning secretion in human milk, nor on effect in nursing infants.

Usage in children under 12 years of age is not recommended. MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug, as serious side-effects and death have been reported with the concomitant use of certain drugs and MAO inhibitors.

In patients who may use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage.

Precautions–Drowsiness may occur and patients should be cautioned against driving a motor vehicle or operating hazardous machinery. Since suicide is an inherent risk in depressed patients they should be closely supervised while receiving treatment. Although Adapin has shown effective tranquilizing activity, the possibility of activating or unmasking latent psychotic symptoms should be kept in mind. This product contains FD&C Yellow No. 5 (tartrazine) which may cause allergic-type reactions (including bronchial asthma) in certain susceptible individuals. Although the overall incidence of FD&C Yellow No. 5 (tartrazine) sensitivity in the general population is low, it is frequently seen in patients who also have aspirin hypersensitivity.

Adverse Reactions–Dry mouth, blurred vision and constipation have been reported. Drowsiness has also been observed.

Adverse effects occurring infrequently include extrapyramidal symptoms, gastrointestinal reactions, secretory effects such as sweating, tachycardia and hypotension. Weakness, dizziness, fatigue, weight gain, edema, paresthesias, flushing, chills, tinnitus, photophobia, decreased libido, rash and pruritus may also occur.

Dosage and Administration–In mild to moderate anxiety and/or depression: 25 mg t.i.d. Increase or decrease the dosage according to individual response. Daily dosage, up to 150 mg may be taken at bedtime without loss of effectiveness. Usual optimum daily dosage is 75 mg to 150 mg per day not to exceed 300 mg per day.

Antianxiety effect usually precedes the antidepressant effect by two or three weeks.

How Supplied–Each capsule contains doxepin, as the hydrochloride: 10 mg, 25 mg, 50 mg, 75 mg and 100 mg capsules in bottles of 100 and 1000.

For complete prescribing information please see package insert or PDR.

References:

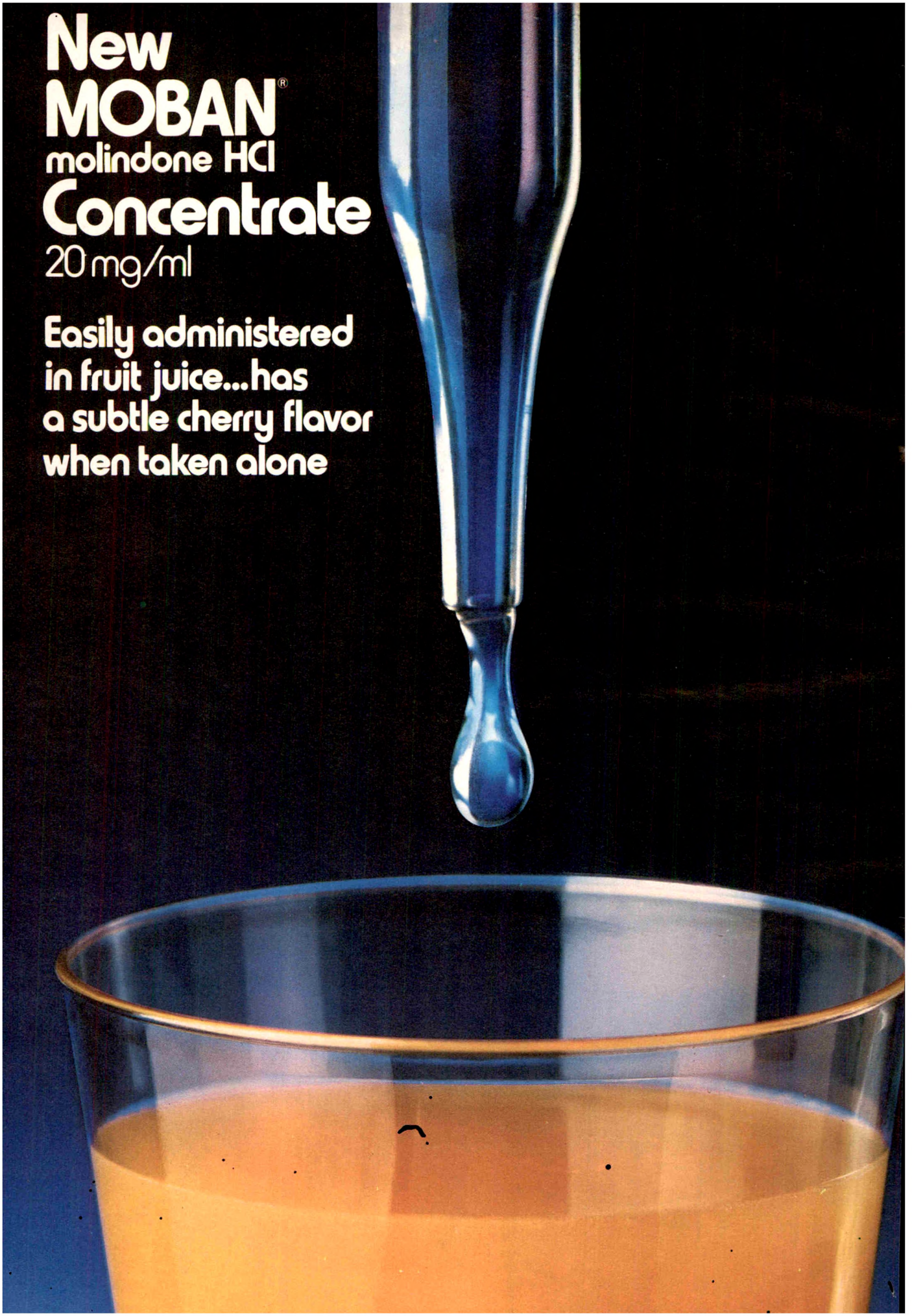
1. Barranco SF, Thrash ML, Hackett E, et al: Early onset of response to doxepin treatment. *J Clin Psychiatry* 40:265-269, 1979 (Sinequan®).
2. Karacan I, Blackburn AB, Thornby JI: The Effect of Doxepin HCl (Sinequan®) on Sleep Patterns and Clinical Symptomatology of Neurotic Depressed Patients with Sleep Disturbance, in Mendels J (ed): *Sinequan®: A monograph of recent clinical studies*, Princeton, NJ, Excerpta Medica, 1977, pp 4-22.
3. Pitts NE: The clinical evaluation of doxepin–A new psychotherapeutic agent. *Psychosomatics* 10:164-171, 1969.

Sinequan® brand of doxepin HCl was the drug used in studies referenced as 1, 2 and 3.

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Adapin® (doxepin HCl) ... effective single-entity drug with antidepressant and antianxiety effects

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in fruit juice...has
a subtle cherry flavor
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An effective antipsychotic agent with a favorable benefit/risk profile

NO incidence	excessive weight gain hepatotoxic changes ophthalmological changes persistent tardive dyskinesia skin pigmentation thyroid function changes tolerance development
LOW incidence	adverse drug interactions hematological changes hypotension nonpersistent symptoms of tardive dyskinesia sedation
MODERATE incidence	extrapyramidal reactions

This table compiled from clinical and field reports, according to the best available information.

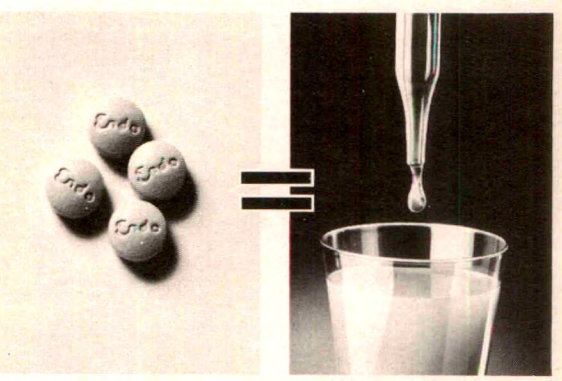
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New MOBAN® molindone HCl Concentrate 20 mg/ml

Concentrated for maximum convenience of higher dosages...easy titration... enhanced compliance

Initial dosage with MOBAN®

- usual starting dose is 50-75 mg/day
- may be increased to 100 mg/day in three to four days
- may be adjusted up or down according to response
- some patients with severe symptomatology may require an increase to 225 mg/day
- elderly and debilitated patients should be started on lower dosage



four 25 mg tablets = 5 milliliters (100 mg)

Please see next page for prescribing information.

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An effective antipsychotic agent

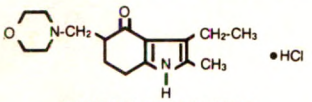
MOBAN® molindone HCl

New dosage form... Concentrate 20 mg/ml

continuing...Tablets 5 mg, 10 mg, 25 mg

DESCRIPTION MOBAN (molindone hydrochloride) is a dihydroindolone compound which is not structurally related to the phenothiazines, the butyrophenones or the thioxanthenes.

MOBAN is 3-ethyl-6, 7-dihydro-2-methyl-5-(morpholinomethyl) indol-4 (5H)-one hydrochloride. It is a white crystalline powder, freely soluble in water and alcohol and has a molecular weight of 312.67.



MOLINDONE HYDROCHLORIDE

ACTIONS MOBAN (molindone hydrochloride) has a pharmacological profile in laboratory animals which predominantly resembles that of major tranquilizers causing reduction of spontaneous locomotion and aggressiveness, suppression of a conditioned response and antagonism of the bizarre stereotyped behavior and hyperactivity induced by amphetamines. In addition, MOBAN antagonizes the depression caused by the tranquilizing agent tetrabenazine.

In human clinical studies tranquilization is achieved in the absence of muscle relaxing or incoordinating effects. Based on EEG studies, MOBAN exerts its effect on the ascending reticular activating system.

Human metabolic studies show MOBAN (molindone hydrochloride) to be rapidly absorbed and metabolized when given orally. Unmetabolized drug reached a peak blood level at 1.5 hours. Pharmacological effect from a single oral dose persists for 24-36 hours. There are 36 recognized metabolites with less than 2-3% unmetabolized MOBAN being excreted in urine and feces.

INDICATIONS MOBAN (molindone hydrochloride) is indicated in the management of the manifestations of schizophrenia.

CONTRAINDICATIONS MOBAN (molindone hydrochloride) is contraindicated in severe central nervous system depression (alcohol, barbiturates, narcotics, etc.) or comatose states, and in patients with known hypersensitivity to the drug.

WARNINGS Usage in Pregnancy: Studies in pregnant patients have not been carried out. Reproduction studies have been performed in the following animals:

Pregnant Rats oral dose— 20 mg/kg/day—10 days no adverse effect
40 mg/kg/day—10 days no adverse effect

Pregnant Mice oral dose— 20 mg/kg/day—10 days slight increase resorptions
40 mg/kg/day—10 days slight increase resorptions

Pregnant Rabbits oral dose— 5 mg/kg/day—12 days no adverse effect
10 mg/kg/day—12 days no adverse effect
20 mg/kg/day—12 days no adverse effect

Animal reproductive studies have not demonstrated a teratogenic potential. The anticipated benefits must be weighed against the unknown risks to the fetus if used in pregnant patients.

Nursing Mothers: Data are not available on the content of MOBAN (molindone hydrochloride) in the milk of nursing mothers.

Usage in Children: Use of MOBAN (molindone hydrochloride) in children below the age of twelve years is not recommended because safe and effective conditions for its usage have not been established.

MOBAN has not been shown effective in the management of behavioral complications in patients with mental retardation.

PRECAUTIONS Some patients receiving MOBAN (molindone hydrochloride) may note drowsiness initially and they should be advised against activities requiring mental alertness until their response to the drug has been established.

Increased activity has been noted in patients receiving MOBAN. Caution should be exercised where increased activity may be harmful.

MOBAN does not lower the seizure threshold in experimental animals to the degree noted with more sedating antipsychotic drugs. However, in humans convulsive seizures have been reported in a few instances.

The physician should be aware that this tablet preparation contains calcium sulfate as an excipient and that calcium ions may interfere with the absorption of preparations containing phenytoin sodium and tetracyclines.

MOBAN (molindone hydrochloride) has an antiemetic effect in animals. A similar effect may occur in humans and may obscure signs of intestinal obstruction or brain tumor.

ADVERSE REACTIONS—CNS EFFECTS The most frequently occurring effect is initial drowsiness that generally subsides with continued usage of the drug or lowering of the dose.

Noted less frequently were depression, hyperactivity and euphoria.

Neurological—Extrapyramidal Reactions Extrapyramidal reactions noted below may occur in susceptible individuals and are usually reversible with appropriate management.

Akathisia Motor restlessness may occur early.

Parkinson Syndrome Akinesia, characterized by rigidity, immobility and reduction of voluntary movements and tremor, have been observed. Occurrence is less frequent than akathisia.

Dystonic Syndrome Prolonged abnormal contractions of muscle groups occur infrequently. These symptoms may be managed by the addition of a synthetic antiparkinson agent (other than L-dopa), small doses of sedative drugs, and/or reduction in dosage.

Autonomic Nervous System Occasionally blurring of vision, tachycardia, nausea, dry mouth and salivation have been reported. Urinary retention and constipation may occur particularly if anticholinergic drugs are used to treat extrapyramidal symptoms.

Hematological There have been rare reports of leucopenia and leucocytosis. If such reactions occur, treatment with MOBAN may continue if clinical symptoms are absent. Alterations of blood glucose, liver function tests, B.U.N., and red blood cells have not been considered clinically significant.

Metabolic and Endocrine Effects Alteration of thyroid function has not been significant. Amenorrhea has been reported infrequently. Resumption of menses in previously amenorrheic women has been reported. Initially heavy menses may occur. Galactorrhea and gynecomastia have been reported infrequently. Increase in libido has been noted in some patients. Impotence has not been reported. Although both weight gain and weight loss have been in the direction of normal or ideal weight, excessive weight gain has not occurred with MOBAN.

Cardiovascular Rare, transient, non-specific T wave changes have been reported on E.K.G. Association with a clinical syndrome has not been established. Rarely has significant hypotension been reported.

Ophthalmological Lens opacities and pigmentary retinopathy have not been reported where patients have received MOBAN (molindone hydrochloride). In some patients, phenothiazine induced lenticular opacities have resolved following discontinuation of the phenothiazine while continuing therapy with MOBAN.

Skin Early, non-specific skin rash, probably of allergic origin, has occasionally been reported. Skin pigmentation has not been seen with MOBAN usage alone.

MOBAN (molindone hydrochloride) has certain pharmacological similarities to other antipsychotic agents. Because adverse reactions are often extensions of the pharmacological activity of a drug, all of the known pharmacological effects associated with other antipsychotic drugs should be kept in mind when MOBAN is used. Upon abrupt withdrawal after prolonged high dosage an abstinence syndrome has not been noted.

Tardive Dyskinesia Although rarely reported with MOBAN (molindone hydrochloride) symptoms were reversible upon discontinuation of therapy.•

Tardive dyskinesia associated with other agents has appeared in some patients on long-term therapy and has also appeared after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). There may be involuntary movements of extremities.

There is no known effective treatment of tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time the syndrome may not develop.

DOSAGE AND ADMINISTRATION Initial and maintenance doses of MOBAN (molindone hydrochloride) should be individualized.

Initial Dosage Schedule The usual starting dosage is 50-75 mg/day.
- —Increase to 100 mg/day in 3 or 4 days.
- —Based on severity of symptomatology, dosage may be titrated up or down depending on individual patient response.
- —An increase to 225 mg/day may be required in patients with severe symptomatology.

Elderly and debilitated patients should be started on lower dosage.

Maintenance Dosage Schedule
1. Mild—5 mg-15 mg three or four times a day.
2. Moderate—10 mg-25 mg three or four times a day.
3. Severe—225 mg/day may be required.

DRUG INTERACTIONS Potentiation of drugs administered concurrently with MOBAN (molindone hydrochloride) has not been reported. Additionally, animal studies have not shown increased toxicity when MOBAN is given concurrently with representative members of three classes of drugs (i.e., barbiturates, chloral hydrate and antiparkinson drugs).

MANAGEMENT OF OVERDOSAGE Symptomatic, supportive therapy should be the rule.

Gastric lavage is indicated for the reduction of absorption of MOBAN (molindone hydrochloride) which is freely soluble in water.

Since the adsorption of MOBAN (molindone hydrochloride) by activated charcoal has not been determined, the use of this antidote must be considered of theoretical value.

Emesis in a comatose patient is contraindicated. Additionally, while the emetic effect of apomorphine is blocked by MOBAN in animals, this blocking effect has not been determined in humans.

A significant increase in the rate of removal of unmetabolized MOBAN (molindone hydrochloride) from the body by forced diuresis, peritoneal or renal dialysis would not be expected. (Only 2% of a single ingested dose of MOBAN is excreted unmetabolized in the urine.)

However, poor response of the patient may justify use of these procedures. While the use of laxatives or enemas might be based on general principles, the amount of unmetabolized MOBAN in feces is less than 1%. Extrapyramidal symptoms have responded to the use of diphenhydramine (Benadryl*) and the synthetic anticholinergic antiparkinson agents, (i.e., Artane*, Cogentin*, Akineton*).

HOW SUPPLIED As tablets in bottles with potencies and colors as follows:

100's and 1000's:
5 mg orange
10 mg lavender
25 mg light green

As a concentrate containing 20 mg molindone hydrochloride per ml in 4 oz. (120ml) bottles.

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*Artane—Trademark, Lederle Laboratories
*Cogentin—Trademark, Merck Sharp & Dohme
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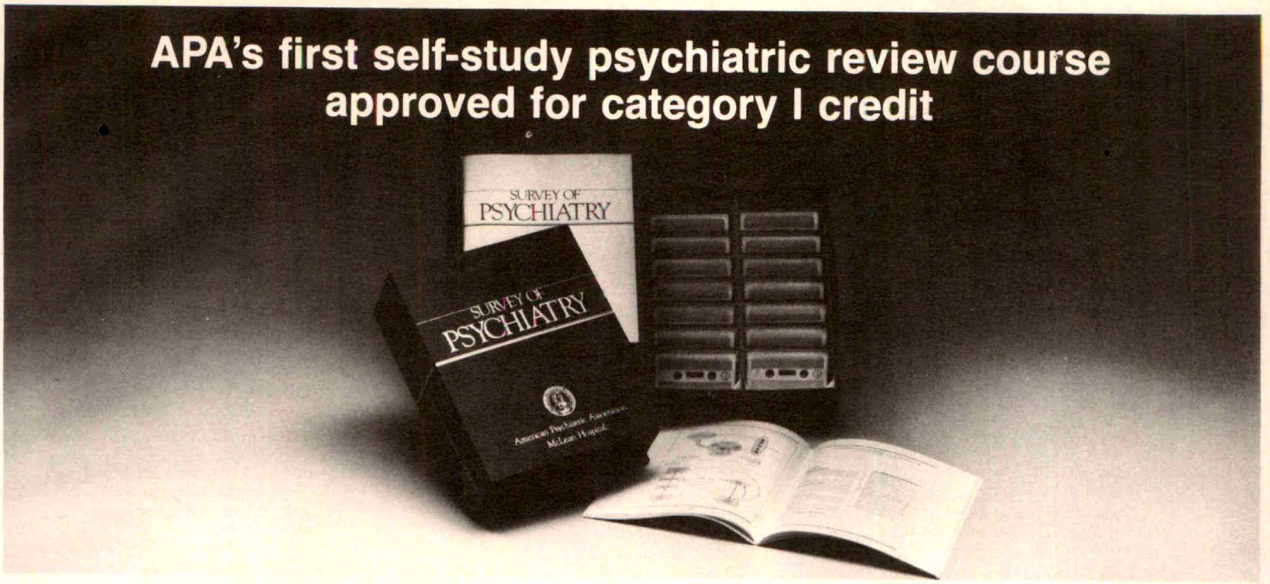
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(flurazepam HCl/Roche)

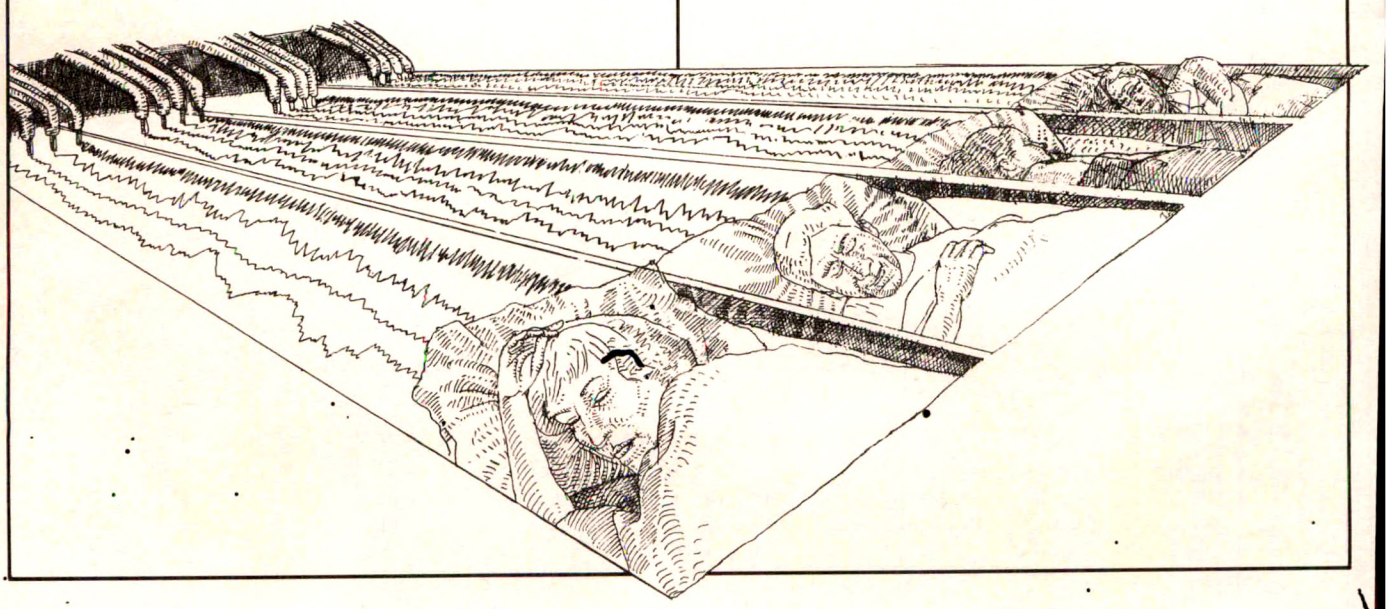
MORE POLYGRAPHIC EVIDENCE

Sleep research laboratory testing of insomnia patients for 995 subject nights demonstrates efficacy of Dalmane (flurazepam HCl/Roche)[1]

In 14 separate studies, subjects given Dalmane were monitored with polygraphic recordings for a total of 995 nights, objectively demonstrating the efficacy of Dalmane.

Efficacy from the first night through 28 nights, without need to increase dosage

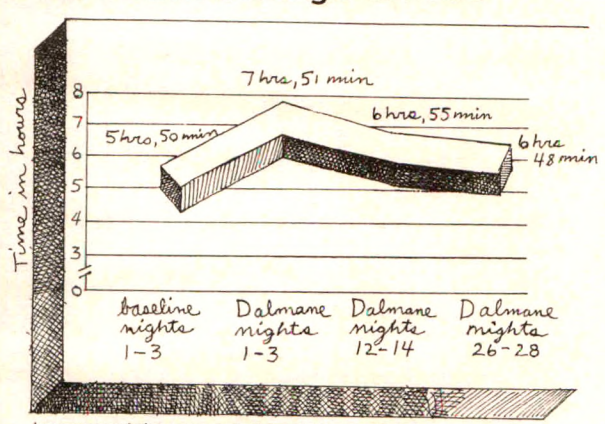
Dalmane (flurazepam HCl/Roche) is the only sleep medication objectively proven effective through 28 consecutive nights of administration in two separate studies in the sleep research laboratory.[2-4] The prolonged use of Dalmane is seldom necessary; should it be, appropriate patient evaluations such as periodic blood counts and liver and kidney function tests are recommended.



OFFERS

OF EFFICACY FOR INSOMNIA

Dalmane® (flurazepam HCl/Roche) improved sleep during short, intermediate and longer-term use



Improvement shown represents mean improvement, in hours, of total sleep time. n=5

Adapted from Dement WC, *et al: Behav Med* 5:25-31, Oct 1978

Since most insomnia is transitory in nature, initiate therapy at the lowest effective dose for the shortest time advisable to achieve a predetermined therapeutic goal.

References:
1. Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley NJ
2. Kales A, *et al: Clin Pharmacol Ther* 19: 576-583, May 1976
3. Dement WC, *et al: Behav Med* 5: 25-31, Oct 1978
4. Kales A, *et al: Clin Pharmacol Ther* 18: 356-363, Sep 1975

Clinical documentation of efficacy in 4950 patients with insomnia[1]

Dalmane (flurazepam HCl/Roche) efficacy was evaluated in 18 clinical studies. These included paired-night, double-blind crossover, double-blind comparison and patient preference studies. Results of these studies demonstrated and confirmed the effectiveness of Dalmane.

Periodic evaluation of patient's sleep habits may help prevent subsequent insomnia

Should insomnia recur, the patient may need guidance in setting up a regular sleep program. This may include establishment of a regular bedtime hour, taking a warm bath prior to retiring and avoidance of alcohol, stimulants or heavy meals late in the evening.

DALMANE® (IV)

flurazepam HCl/Roche

15-mg and 30-mg capsules

UNMATCHED PROOF, UNMATCHED EFFICACY

Please see summary of product information on following page.

ROCHE®

Protocol for insomnia management

☐ Treat physical and/or psychological factors that may underlie an inability to sleep.

☐ Thoroughly question and evaluate new patients who specifically request sleep medications.

☐ Consider other medications the patient may be taking (including alcoholic beverages) and be aware of possible drug interactions.

☐ Select a sleep medication with objectively *documented* proof of efficacy and a wide margin of safety.

☐ Initiate treatment at the lowest effective dose for the period of time deemed necessary *to achieve a reasonable therapeutic goal.*

☐ Monitor response to therapy and, based upon patient reassessment, determine need for continuing medication on an individual basis.

☐ Advise patients who ingest alcoholic beverages or other CNS depressants against doing so while on sleep medication. CNS stimulants such as caffeine should also be discontinued.

☐ Caution patients against engaging in hazardous occupations requiring complete mental alertness such as operating machinery or driving a motor vehicle shortly after ingesting the drug. In addition, as with all CNS depressants, patients may have reduced motor performance while on hypnotic therapy and should be advised accordingly.

☐ Avoid prescribing for suicidal or dependency-prone individuals whose histories indicate an inability to handle any psychoactive substances including alcohol.

Now available from Roche—important guidance for your patients with insomnia.

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DALMANE® (IV)
flurazepam HCl/Roche

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One 30-mg capsule *h.s.*—usual adult dosage
(15 mg may suffice in some patients).

Before prescribing, please consult complete product information, a summary of which follows:
Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Objective sleep laboratory data have shown effectiveness for at least 28 consecutive nights of administration. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended.
Contraindications: Known hypersensitivity to flurazepam HCl.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

Usage in Pregnancy: **Several studies of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) suggest increased risk of congenital malformations during the first trimester of pregnancy. Dalmane, a benzodiazepine, has not been studied adequately to determine whether it may be associated with such an increased risk. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.
Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies, or with impaired renal or hepatic function. Periodic blood counts and liver and kidney function tests are advised during repeated therapy.
Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase.
Dosage: Individualize for maximum beneficial effect.
Adults: 30 mg usual dosage; 15 mg may suffice in some patients.
Elderly or debilitated patients: 15 mg recommended initially until response is determined.
Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

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Psychiatric Associateships at National Institute of Mental Health

The National Institute of Mental Health Associate Program at the National Institutes of Health offers Associateships for two or three years of intensive training in virtually all areas of clinical and basic psychobiological research in adult and child psychiatry. Excellent facilities are available at the Clinical Center on the 306 acre NIH campus in Bethesda, Maryland, and at nearby St. Elizabeths Hospital in Washington, D.C. During the first year, Clinical Associates usually are assigned clinical responsibility for adults or children who are admitted for both therapeutic and research purposes; in the second and subsequent years they usually work full time on clinical or research projects, according to their interests. Research Associates engage in fulltime laboratory research under the preceptorship of one of the senior staff. All associates may participate in tutorial seminars and other teaching programs.

Clinical Associates usually begin their appointments at the completion of residency training. Exceptionally well qualified individuals, however, can be appointed earlier in their training. The Clinical Center is approved for 1 year of psychiatric residency training at the PGY4 level. Since selections for these positions will be made in the spring of 1981, PGY2 or PGY3 psychiatric residents may now apply.

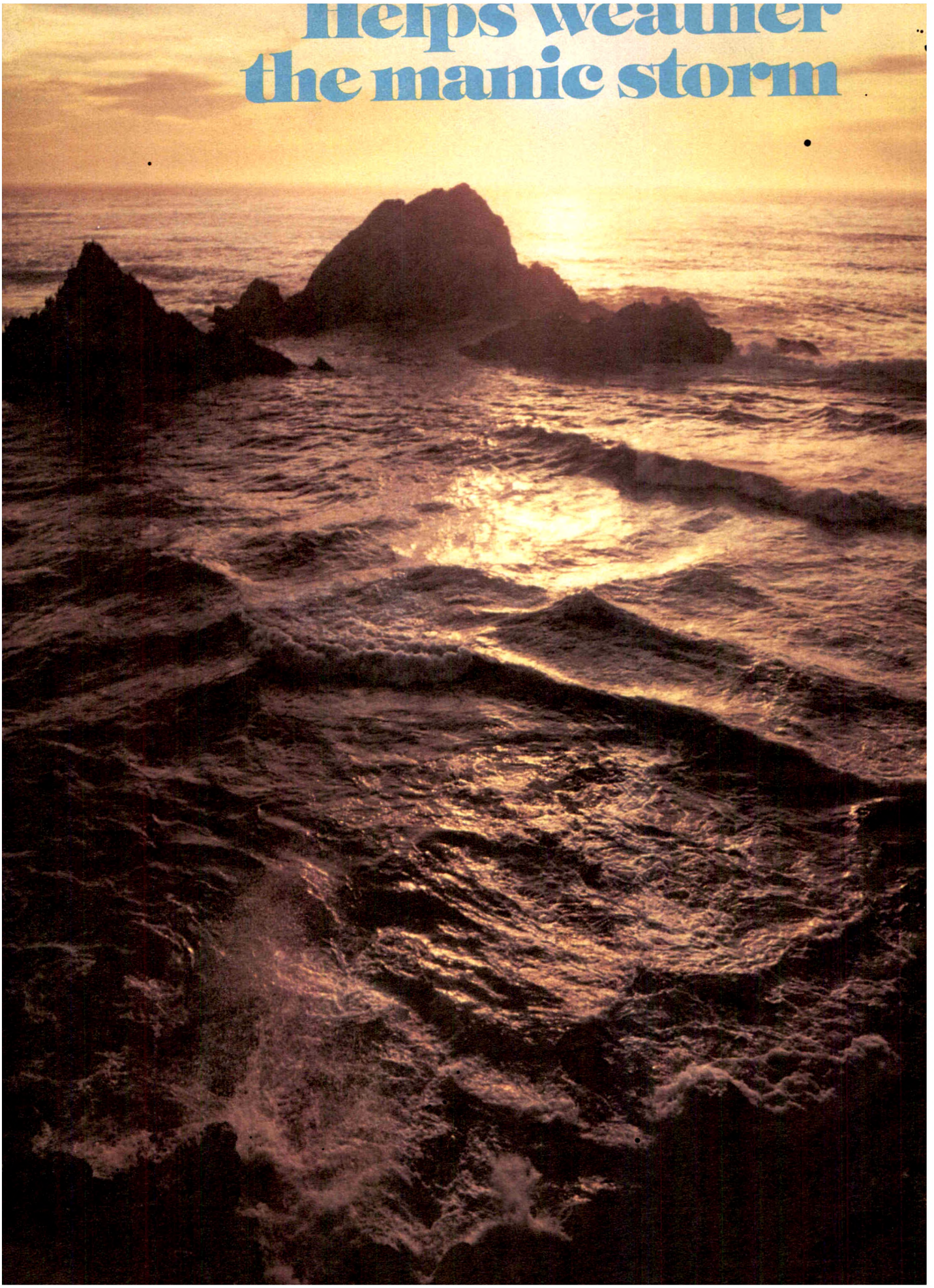
Applications are due by January 30, 1981 for appointments which begin July 1982. Both U.S. citizens and noncitizens can apply.

A competitive salary is available for candidates qualifying for appointment in the Commissioned Corps, United States Public Health Service. A portion of the salary is tax exempt. Moving and travel expenses are paid and health care is free. Appointment as a Staff Fellow or a Visiting Associate is also available.

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In manic-depressive patients with a history of mania, 'Eskalith'

- **prevents or diminishes the intensity of future episodes**
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- **helps patients function within normal limits**

Before prescribing, see complete prescribing information in SK&F literature or PDR. The following is a brief summary.

WARNING
Lithium toxicity is closely related to serum lithium levels, and can occur at doses close to therapeutic levels. Facilities for prompt and accurate serum lithium determinations should be available before initiating therapy.

Indications: Treatment of manic episodes of manic-depressive illness. Maintenance therapy prevents or diminishes the intensity of subsequent episodes in manic-depressive patients with a history of mania.

Warnings: Lithium should generally not be given to patients with significant renal or cardiovascular disease, severe debilitation or dehydration, sodium depletion, or to patients receiving diuretics.

Chronic lithium therapy may be associated with diminution of renal concentrating ability; such patients should be carefully managed to avoid dehydration with resulting lithium retention and toxicity. Morphologic changes with glomerular and interstitial fibrosis and nephron atrophy have been reported. When renal function is assessed, progressive or sudden changes in function indicate the need for reevaluation of treatment.

Caution patient and family to watch for diarrhea, vomiting, tremor, mild ataxia, drowsiness, or muscular weakness as signs of lithium toxicity, and to discontinue therapy and contact a physician should they occur. Patients receiving combined therapy with lithium and an antipsychotic should be monitored closely for early evidence of neurologic toxicity and treatment discontinued promptly if such signs appear. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery).

Lithium may prolong the effects of neuromuscular blocking agents. Such agents should be given with caution to patients receiving lithium.

Lithium may cause fetal developmental anomalies, especially involving the cardiovascular system. If lithium is used during pregnancy, or if a patient becomes pregnant while taking it, she should be apprised of the potential hazard to the fetus. Except in rare and unusual circumstances, nursing should not be undertaken while a patient is on lithium therapy, since lithium is excreted in human milk.

Not recommended for children under 12.

Precautions: Lithium tolerance is greater during the acute manic phase and decreases when manic symptoms subside.

Lithium therapy may lead to sodium depletion. Normal diet (including salt) and adequate fluid intake (2500-3000 ml.) must be maintained, at least during initial stabilization period. Protracted sweating or diarrhea can decrease tolerance; in such cases, administer supplemental fluid and salt.

Sweating, diarrhea, and concomitant infection with elevated temperatures may require temporary reduction or cessation of dosage.

Where hypothyroidism exists, thyroid function should be monitored during lithium stabilization and maintenance; where hypothyroidism occurs during stabilization and maintenance, supplemental thyroid treatment may be used.

Adverse Reactions: Mild to moderate toxic reactions may occur at serum lithium levels from 1.5 to 2.5 mEq./1., and moderate to severe reactions at levels from 2.0 to 2.5 mEq./1. Fine hand tremor, polyuria, and mild thirst may occur during initial therapy and persist. Transient and mild nausea and general discomfort may also appear during initial therapy. These effects usually subside with continued treatment or temporary reduction or cessation of dosage. If persistent, discontinue dosage. Diarrhea, vomiting, drowsiness, muscular weakness, and lack of coordination may be early signs of toxicity and may occur at levels below 2.0 mEq./1. At higher levels, ataxia, giddiness, tinnitus, blurred vision, and a large output of dilute urine may be seen. Serum levels above 3.0 mEq./1. may produce a complex clinical picture, involving multiple organs and systems. Serum levels should not exceed 2.0 mEq./1. during acute phase.

The following reactions appear to be related to serum lithium levels, including levels within the therapeutic range: **Neuromuscular**—tremor, muscle hyperirritability (fasciculations, twitching, clonic movements of whole limbs), ataxia, choreo-athetotic movements, hyperactive deep tendon reflex; **Central Nervous System**—blackout spells, epileptiform seizures, slurred speech, dizziness, vertigo, incontinence of urine or feces, somnolence, psychomotor retardation, restlessness, confusion, stupor, coma; **Cardiovascular**—cardiac arrhythmia, hypotension, peripheral circulatory collapse; **Gastrointestinal**—anorexia, nausea, vomiting, diarrhea; **Genitourinary**—albuminuria, oliguria, polyuria, glycosuria; **Dermatologic**—drying and thinning of hair, alopecia, anesthesia of skin, chronic folliculitis, exacerbation of psoriasis, xerosis cutis; **Autonomic**—blurred vision, dry mouth; **Thyroid Abnormalities**—euthyroid goiter and/or hypothyroidism (including myxedema) with lower T_3 and T_4. I^{131} uptake may be elevated; rare cases of hyperthyroidism; **EEG Changes**—diffuse slowing, widening of the frequency spectrum, potentiation and disorganization of background rhythm; **EKG Changes**—reversible flattening, isoelectricity or inversion of T-waves; **Miscellaneous**—fatigue, lethargy, transient scotomata, dehydration, weight loss, tendency to sleep.

Reactions unrelated to dosage include: transient EEG and EKG changes, leukocytosis, headache, diffuse nontoxic goiter with or without hypothyroidism, transient hyperglycemia, generalized pruritus with or without rash, cutaneous ulcers, albuminuria, worsening of organic brain syndromes, excessive weight gain, edematous swelling of ankles or wrists, thirst or polyuria, sometimes resembling diabetes insipidus, and metallic taste. A single case of a syndrome resembling Raynaud's has been reported.

How Supplied: 300 mg. capsules in bottles of 100.
300 mg. scored tablets in bottles of 100.

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Brief Summary of Prescribing Information

Indications and Usage: Symptomatic relief of anxiety, tension, agitation, irritability and insomnia associated with anxiety neuroses and transient situational disturbances; anxiety associated with depressive symptoms and as a treatment of symptoms of anxiety if such symptoms are a significant feature of functional or organic disorders, particularly gastrointestinal or cardiovascular.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients on lorazepam not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation.

Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions.

Observe usual precautions with impaired renal or hepatic function.

Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component.

Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day. No effect dose was 1.25mg/kg/day (approximately 6 times the maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatric patients requires caution and frequent monitoring for symptoms of upper G.I. disease.

Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug.

In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Overdosage: In management of overdosage with any drug, bear in mind that multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring of vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

Ativan® IV for Anxiety (lorazepam)

Dosage: **Individualize for maximum beneficial effects. Increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; insomnia due to anxiety or transient situational stress, 2-4mg h.s.**

How Supplied: **0.5, 1.0 and 2.0mg tablets.**

Wyeth Laboratories
Philadelphia, PA 19101

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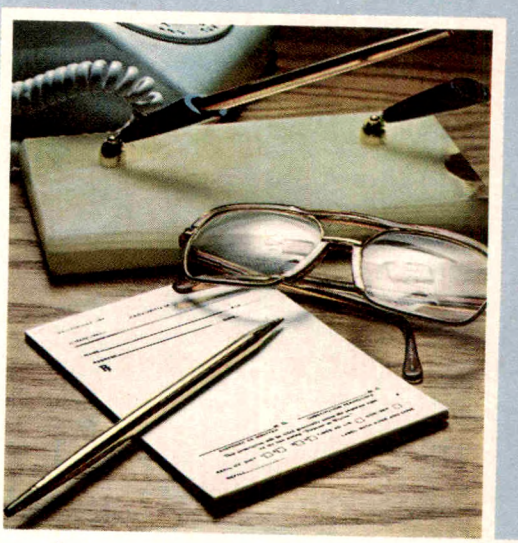
Why one benzodiazepine and not another?

Are you concerned about long-acting metabolites? Many clinicians, as well as pharmacologists, are beginning to draw attention to this problem (see New England Journal of Medicine, April 5, 1979).

In contrast to some older benzodiazepines, Ativan (lorazepam) does not give rise to long-lasting active metabolites. As with all benzodiazepines, you should follow the usual precautions concerning co-administration with other CNS depressants and warn your patients against operating dangerous machinery and motor vehicles.

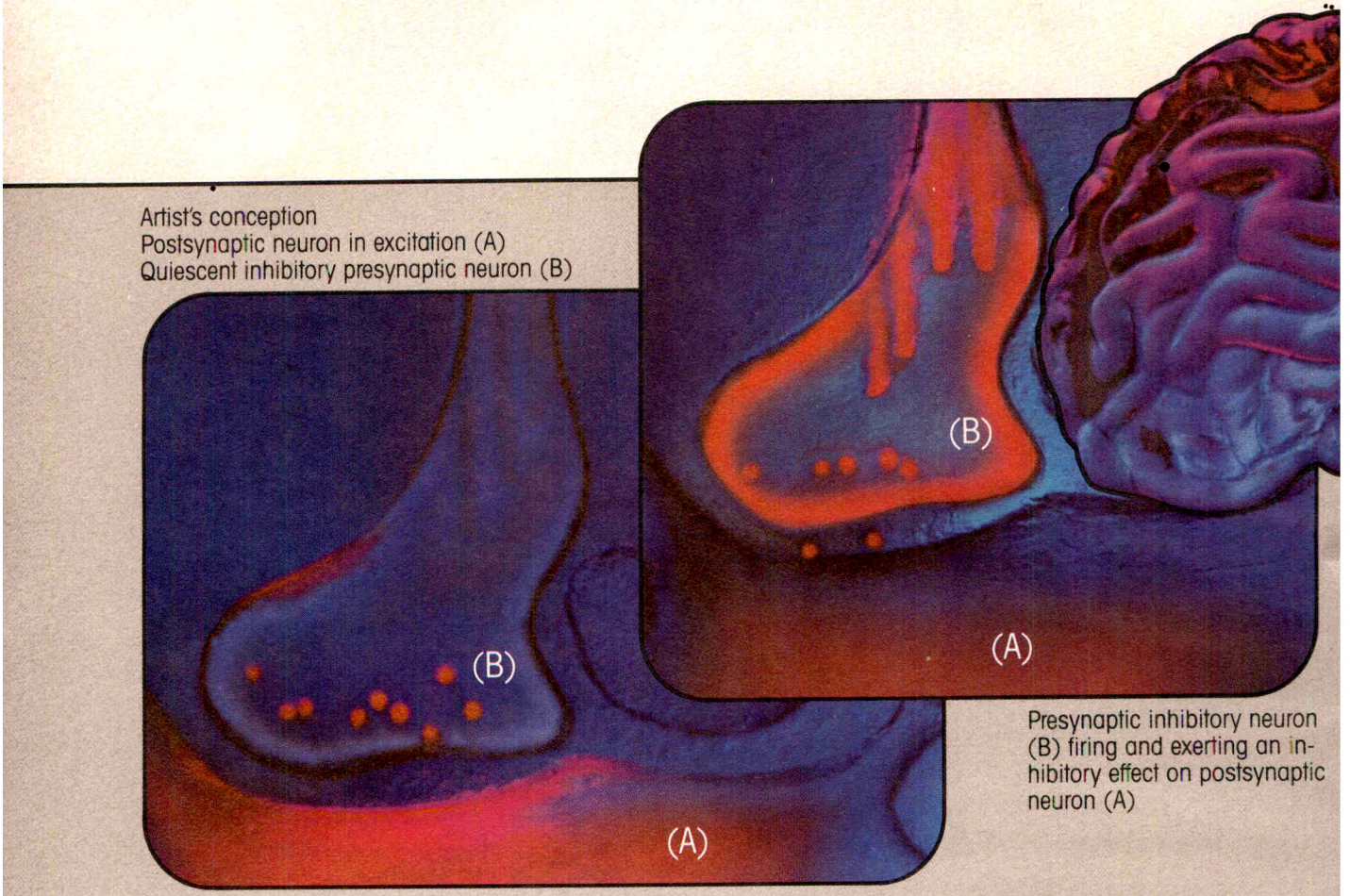
However, it is noteworthy that Ativan showed no clinical evidence of accumulation even when given in high doses over periods up to 6 months. The half-life of free lorazepam is about 12 hours; steady-state serum levels are attained in 2-3 days. Comparable data for diazepam: 20-50 hours and at least 7-10 days. (The pharmacokinetic profile of a drug can define such characteristics as absorption, distribution, metabolism and elimination but cannot, at present, be directly related to its therapeutic effectiveness.)

Ativan has a convenient b.i.d. or t.i.d. dosage schedule; it is compatible with a long list of other medications and, of course, it is a highly effective anxiolytic agent, as established in numerous nationwide, double-blind, controlled evaluations in thousands of patients.



See important information on preceding page.

Ativan® (lorazepam) **for Anxiety**



Artist's conception
Postsynaptic neuron in excitation (A)
Quiescent inhibitory presynaptic neuron (B)

Presynaptic inhibitory neuron (B) firing and exerting an inhibitory effect on postsynaptic neuron (A)

Research in Progress

points to the role of specific CNS binding sites for the benzodiazepines — often considered the most effective anxiolytic agents[1]

Although the clinical effectiveness of the benzodiazepines is widely accepted by the medical profession, their mechanism of action is as yet unknown. Inability to define a neurophysiological or biochemical basis of anxiety has hindered the search.[2]

Current research has, however, identified specific benzodiazepine binding sites in the CNS. Various characteristics of these sites suggest that they may represent the actual sites of action of these agents.[3,4]

Specific tenacity of benzodiazepines to CNS binding sites

In equilibrium binding studies on rat and human brain tissue, benzodiazepines demonstrate a high affinity for specific sites found primarily in the synaptic membrane fraction. Of the compounds tested, only benzodiazepines compete for these binding sites. Furthermore, each benzodiazepine displays a characteristic degree of affinity which closely parallels its clinical and pharmacological potency.[3,4]

A significant correlation has also been found between the binding affinity of a particular benzodiazepine and its anxiolytic, anticonvulsant and muscle relaxant effects in certain animal tests.[4]

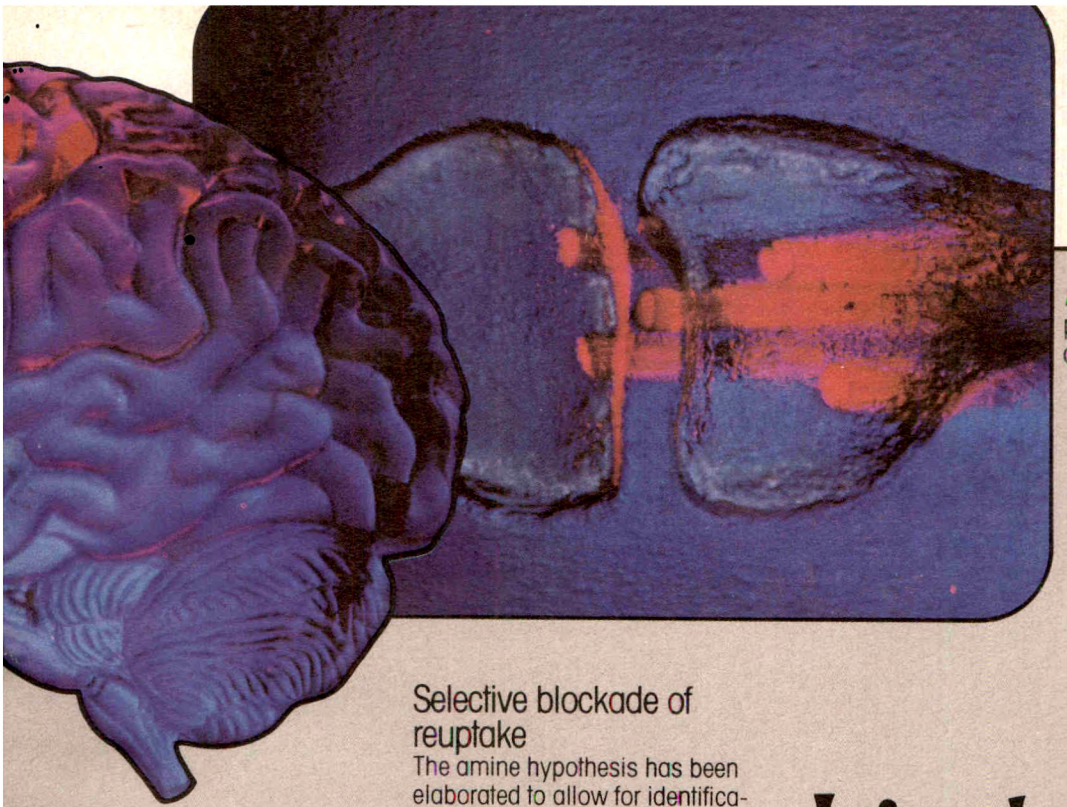
Although these *in vitro* animal and human tissue studies do not necessarily suggest clinical significance, further investigation of specific benzodiazepine binding sites may provide insight into the mechanism of action of these clinically effective anxiolytic agents.

References: 1. Rickels K: Drug treatment of anxiety, in *Psychopharmacology in the Practice of Medicine*, edited by Jarvik ME. New York, Appleton-Century-Crofts, 1977, p. 312. **2.** Byck R: Drugs and the treatment of psychiatric disorders, chap. 12, in *The Pharmacological Basis of Therapeutics*, ed. 5, edited by Goodman LS, Gilman A. New York, Macmillan Publishing Co., Inc., 1975, p. 190. **3.** Möhler H, Okada T: *Life Sci 20* (12): 2101-2110, 1977. **4.** Möhler H, *et al: Life Sci 22* (11): 985-996, 1978. **5.** Hollister LE: *N Engl J Med 299:* 1106-1109, Nov 16, 1978.

While *in vitro* studies cannot be interpreted to have clinical significance, these represent an attempt to explain

THE LIMBITROL
in the treatment of moderate

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Artist's conception of propagation of impulse across synaptic cleft.

and to the role of tricyclic antidepressants in blocking the presynaptic reuptake of neurotransmitters — believed important in the treatment of depression

The amine hypothesis, which holds that depression is associated with a deficiency of functional neurotransmitter at the synapse, is currently the most viable theory of depression. It is supported by studies on the pharmacologic action of various clinically effective antidepressants. The tricyclics, for instance, have been shown to block reuptake by the presynaptic terminals, permitting greater availability and prolonged action of neurotransmitters at receptor sites. This may compensate for the relative deficit of functional neurotransmitters found in depression.[5]

Selective blockade of reuptake

The amine hypothesis has been elaborated to allow for identification of subtypes of depression based on specific neurotransmitter deficiencies. It has been proposed that measurement of the metabolite 3-methoxy-4-hydroxy-phenylglycol (MHPG) in urine may permit such a differentiation; low MHPG levels indicating a deficit of norepinephrine, and normal or high MHPG suggesting a deficit of another neurotransmitter, probably serotonin. Current pharmacological studies indicate that certain tricyclics may be more efficient in blocking norepinephrine reuptake, while others more selectively blockade serotonin.[5] Further investigation may lead to eventual selection of subtype-specific antidepressants on the basis of biochemical tests.

Limbitrol® 5/12.5 10/25

Tablets 5-12.5 each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)
Tablets 10-25 each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt)

Dual action specific for dual symptomatology

Limbitrol contains both a benzodiazepine — specific for the symptoms of anxiety, and amitriptyline — specific for depressive symptoms. It therefore provides the agents most appropriate to relieve both symptom syndromes in the nonpsychotic patient with mixed anxiety and depression. What better rationale for prescribing Limbitrol for patients who need pharmacologic intervention in support of their psychotherapy.

CLINICAL AFFINITY
o severe depression and anxiety

Please see summary of complete product information on following page.

LIMBITROL® TABLETS Tranquilizer—Antidepressant

Before prescribing, please consult complete product information, a summary of which follows:
Indications: Relief of moderate to severe depression associated with moderate to severe anxiety.
Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants. Do not use with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Contraindicated during acute recovery phase following myocardial infarction.
Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs. Closely supervise cardiovascular patients. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses. Myocardial infarction and stroke reported with use of this class of drugs.)

***Usage in Pregnancy:* Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage; withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline; symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).
Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Periodic liver function tests and blood counts are recommended during prolonged treatment. Amitriptyline component may block action of guanethidine or similar antihypertensives. Concomitant use with other psychotropic drugs has not been evaluated: sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Limbitrol should not be taken during the nursing period. Not recommended in children under 12.
In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects.
Adverse Reactions: Most frequently reported are those associated with either component alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline. Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely.
This list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs.
Cardiovascular: Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke.
Psychiatric: Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.
Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns.
Anticholinergic: Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract.
Allergic: Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus.
Hematologic: Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia.
Gastrointestinal: Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.
Endocrine: Testicular swelling and gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female and elevation and lowering of blood sugar levels.
Other: Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.
Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. I.V. administration of 1 to 3 mg physostigmine salicylate has been reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.
Dosage: Individualize according to symptom severity and patient response. Reduce to smallest effective dosage when satisfactory response is obtained. Larger portion of daily dose may be taken at bedtime. Single h.s. dose may suffice for some patients. Lower dosages are recommended for the elderly.
Limbitrol 10-25, initial dosage of three to four tablets daily in divided doses, increased up to six tablets or decreased to two tablets daily as required. Limbitrol 5-12.5, initial dosage of three to four tablets daily in divided doses, for patients who do not tolerate higher doses.
How Supplied: White, film-coated tablets, each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) and blue, film-coated tablets, each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)—bottles of 100 and 500; Tel-E-Dose ® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50.

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How to initiate and maintain therapy

Select dosage strength appropriate for each patient
☐ Limbitrol 5-12.5 is recommended to minimize drowsiness and for elderly patients
☐ Limbitrol 10-25 may be indicated for patients who tolerate medication without undue side effects

Specify daily dosage based on symptom severity
☐ An initial dosage of three tablets is recommended
☐ Dosage may be increased to six tablets or decreased to two tablets daily as necessary
☐ Once a satisfactory response is obtained, patients should be continued on the smallest dose required to maintain the desired effect

Utilize dosage options to best accommodate individual patient needs
☐ T.I.D. or Q.I.D., familiar regimens most suited for patients who tolerate medication without undue drowsiness
☐ Two tablets one hour before bedtime and one tablet midday may minimize daytime drowsiness and help relieve a common target symptom—insomnia
☐ Entire dosage h.s. to take maximum advantage of the sedative effect

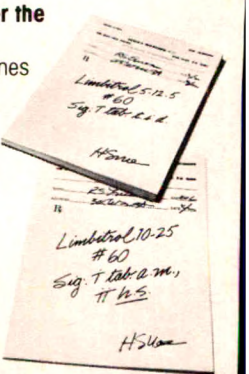
Your guide to patient management... when you decide medication is needed

How to make each patient an informed patient

1. Discuss with patients the probability that they will experience drowsiness, especially during the first week.
2. Reassure your patients that drowsiness is one indication that the medication is working and that it may help alleviate their insomnia.
3. Encourage patients to report if drowsiness becomes troublesome so that, if necessary, dosage schedule can be adjusted.
4. Caution patients about the combined effects with alcohol or other CNS depressants. Let them know that the additive effects may produce a harmful level of sedation and CNS depression.
5. Caution patients about activities requiring complete mental alertness, such as operating machinery or driving a car.
6. Warn pregnant patients and patients of childbearing age that the safety of Limbitrol in pregnancy has not yet been established.

Please see complete product disclosure for other pertinent information.

Limbitrol should not be used under the following circumstances:
1. Hypersensitivity to benzodiazepines or tricyclic antidepressants.
2. Concomitantly with an MAO inhibitor. To replace an MAO inhibitor with Limbitrol, discontinue MAO inhibitor for a minimum of 14 days before cautiously initiating Limbitrol therapy.
3. During the acute recovery phase following myocardial infarction.



In moderate to severe depression and anxiety

Limbitrol®

Relief without a phenothiazine

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NOVEMBER 1980

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Just Published . . .

The Rape Victim

by Elaine Hilberman, M.D.

Mythologies about rape are legion. However, in recent years behavioral scientists have accumulated a body of information about rape and about an unfolding series of reactions experienced by the victim after rape. The aim of this concise, tightly reasoned book is to summarize what is now known about the needs and experiences of the victim and her family, and to provide a framework in which the clinician can more knowledgeably supply assistance and support. This document, published by the American Psychiatric Association, is the result of a project of the Association's Committee on Women.

Pointing out that the profound impact of rape stress is best understood in the context of rape as "a crime against the person and not the hymen," Dr. Hilberman sees rape as the ultimate violation of the self short of homicide, as an act of violence and humiliation in which not only is the victim's very existence threatened, and her inner and most private space invaded, but her autonomy and control are totally demolished. Dr. Hilberman shows that if and when the victim reports the crime, she is confronted with a complex series of yet additional stresses growing out of her contact with the hospital, police, courts, media and community opinion. Additionally, her crisis differs from other crises in that her usual social support system is likely to be disrupted. Her immediate needs are for empathy and safety, and a sense of control over what is happening in her dealings with the several agencies. Dr. Hilberman shows that in the absence of sensitivity to these needs, the experience of reporting becomes another assault.

This book presents a professional, sobering, and balanced picture of the problems of the rape victim and details what the clinician must know—about local hospital policy, criminal justice procedure, rape statutes, and community attitudes and services—in order to be able to treat victims with an understanding of the larger context in which rape occurs.

ELAINE HILBERMAN, M.D., *is in the Department of Psychiatry at the University of North Carolina School of Medicine.*

Paperback edition—$5.00 each, may be ordered from the AMERICAN PSYCHIATRIC ASSOCIATION, Publication Sales, 1700 18th St., N.W., Washington, D.C. 20009.

Hardback edition—$7.95 may be ordered from Basic Books, Inc., 10 East 53rd Street, New York, New York 10022.

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Take it from Andy Machak, automotive electrical plant foreman: "I'm alive today because a fellow employee was trained in CPR.

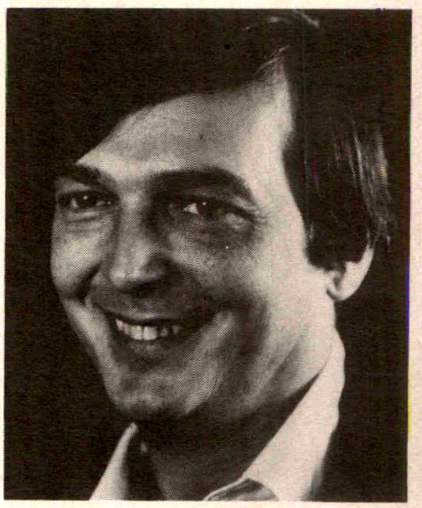
"Cardiopulmonary resuscitation training was one valuable asset that day. I got caught in some heavy machinery at the plant and blacked out.

"They told me later it took six guys to free me. I wasn't breathing. My heart stopped. One of the guys, Don Guarino, said, 'I gotta try anyway.'

"Thanks to his CPR training, he revived me – saved my life.

"Since that happened I realize that no employee is immune from heart attacks or accidents, and a few CPR-trained employees – maybe one for every 50 people – can make the difference between life and death."

Call your Red Cross Chapter and find out about CPR training for your plant or office. CPR training is a valuable asset for any business.





It takes steady, trained hands to perform CPR.

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Help cure cancer write now.

Thanks to your help, the tide is beginning to turn. The past few years have brought new discoveries in chemotherapy.

And new diagnostic techniques that combine the "eyes" of X-ray machines with the "brains" of computers. And successful new programs of combination therapies.

And there are promising reports coming in from research laboratories all over the world.

We now have everything we need to save about half the people who get cancer.

Please don't quit on us now.

We're halfway there.

American Cancer Society

We want to cure cancer in your lifetime.

This space contributed by the publisher as a public service.

Brief Summary of Prescribing Information

Indications and Usage: Symptomatic relief of anxiety, tension, agitation, irritability and insomnia associated with anxiety neuroses and transient situational disturbances; anxiety associated with depressive symptoms and as a treatment of symptoms of anxiety if such symptoms are a significant feature of functional or organic disorders, particularly gastrointestinal or cardiovascular.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients on lorazepam not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation.

Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions.

Observe usual precautions with impaired renal or hepatic function.

Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component.

Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg kg day. No effect dose was 1.25mg/kg/day (approximately 6 times the maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatric patients requires caution and frequent monitoring for symptoms of upper G.I. disease.

Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug.

In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Overdosage: In management of overdosage with any drug, bear in mind that multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring of vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

Ativan® IV for Anxiety (lorazepam)

Dosage: **Individualize for maximum beneficial effects. Increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; insomnia due to anxiety or transient situational stress, 2-4mg h.s.**

How Supplied: **0.5, 1.0 and 2.0mg tablets.**

Wyeth Laboratories
Philadelphia, PA 19101

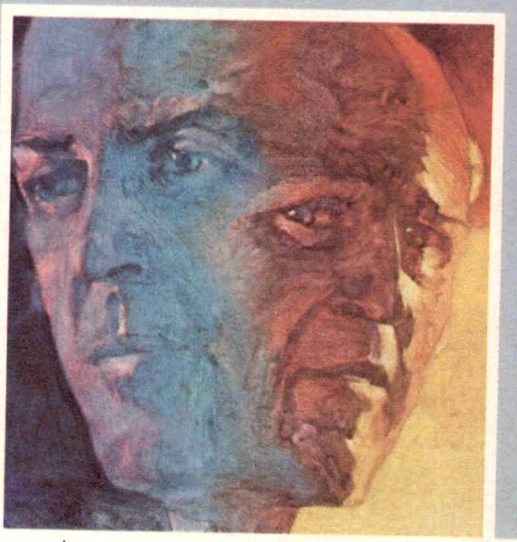
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A mixed syndrome. When drug intervention is indicated in patients whose anxiety is accompanied by depressive symptoms, the choice, essentially, is among three basic strategies: (1) an antidepressant; (2) a tranquilizer; (3) a combination of (1) and (2).

With respect to strategy (2), you should know that Ativan (lorazepam) has been shown to be statistically and clinically significantly effective in alleviating the anxiety component of this mixed syndrome, in common protocol controlled studies of 653 patients (337 on Ativan).

Since Ativan was also found to be compatible with tricyclics, if you opt for strategy (3), your patient has the benefit of rapid anxiolytic action, absence of long-acting metabolites and rapid clearance when you decide to discontinue Ativan. All benzodiazepines, however, including Ativan, produce added CNS depressant effects when administered with alcohol and other CNS depressants.

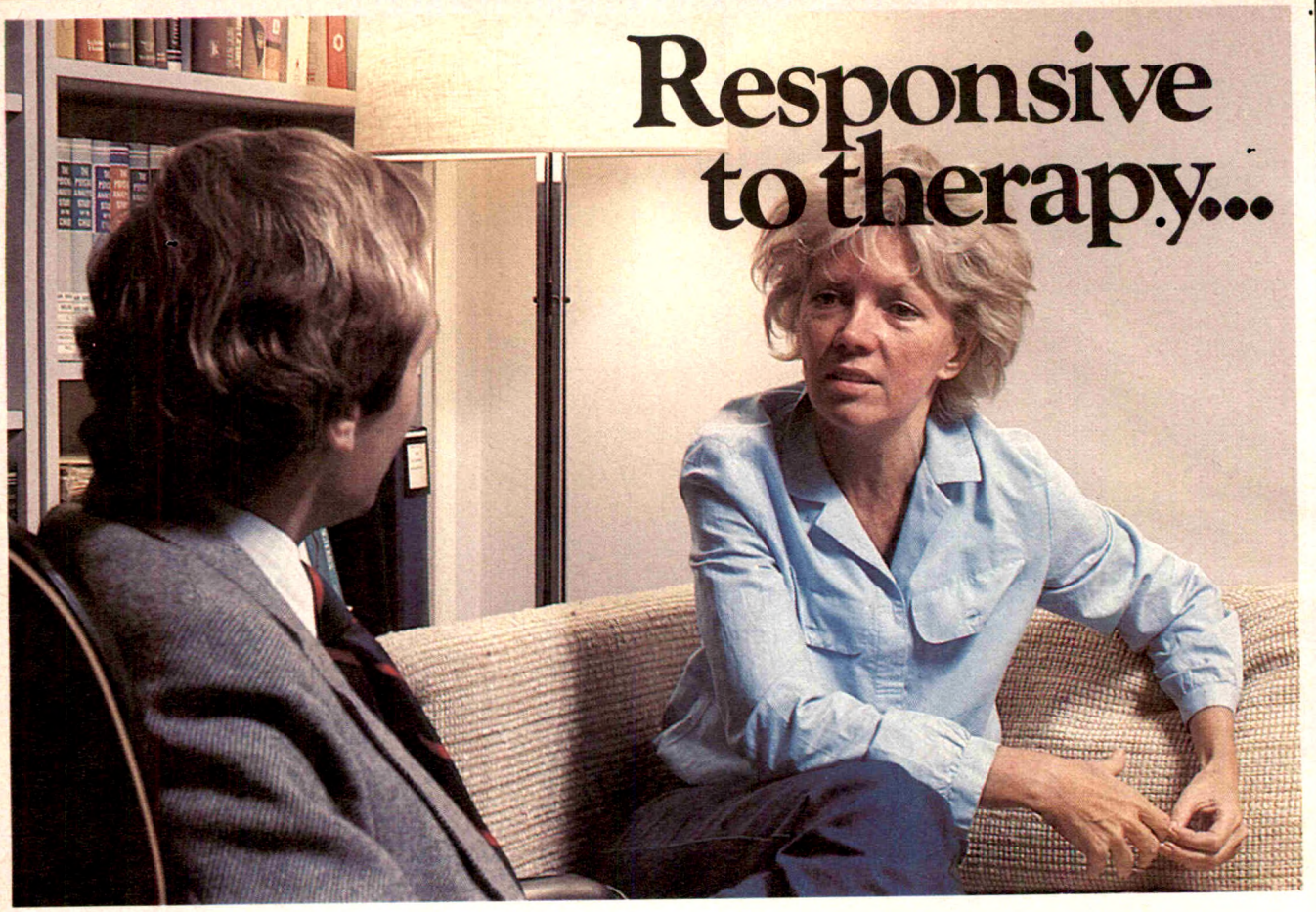
In either case, Ativan offers you the convenience of a b.i.d. schedule and a flexibility not possible with any fixed combination.



See important information on preceding page.

Ativan® (lorazepam) for Anxiety

associated with depressive symptoms


Responsive
to therapy...


alert
on the job.*
© McNEILAB, Inc. 1980

Haldol®
(haloperidol)
tablets/concentrate/injection

Promptly controls psychotic symptoms... usually leaving the patient alert and productive

Highly effective in a wide range of both acute and chronic psychotic disorders,[1-7] such as hallucinations, delusions, suspiciousness, hostility, mania.

Permits aggressive titration to effective dosage levels for optimal response.[1,4]

Facilitates prompt initiation of other therapeutic efforts... and often helps avoid hospitalization.[1-3]

Minimal risk of hypotension, oversedation, or troublesome anticholinergic effects.[2,4-6,8,9] Transient hypotension occurs rarely; severe orthostatic hypotension has not been reported. Although some instances of drowsiness have been reported, marked sedation is rare.

Common side effects easily controlled.[1,2,4-7] Although extrapyramidal symptoms (EPS) have been reported frequently, they are usually dose-related and readily controlled with dose adjustment or antiparkinson drugs. EPS often diminish spontaneously with continued use of HALDOL haloperidol.

References: 1. Man, P.L.: Dis. Nerv. Syst. *34*:113 (Feb.) 1973. 2. Ayd, F.J., Jr.: Med. Sci. *18*:55 (Oct.) 1967. 3. Rapp, M.S.: Can. Psychiatr. Assoc. J. *15*:73 (Feb.) 1970. 4. Howard, J.S.: Dis. Nerv. Syst. *35*:458 (Oct.) 1974. 5. Gerle, B.: Clin. Trials J. *3*:380 (Feb.) 1966. 6. Abuzzahab, F.S., Sr.: Psychosomatics *11*:188 (May-June) 1970. 7. Darling, H.F.: Dis. Nerv. Syst. *34*:364 (Oct.-Nov.) 1973. 8. Snyder, S.H., et al: Science *184*:1243 (June 21) 1974. 9. Stimmel, C.L.: Dis. Nerv. Syst. *34*:219 (Apr.) 1976.

*Not an actual case history, this situation illustrates the action of HALDOL haloperidol as reported in various clinical studies (available on request).

Please turn page for summary of prescribing information.

concentrate

A tasteless, odorless, colorless liquid concentrate for better patient compliance: 2 mg per ml.

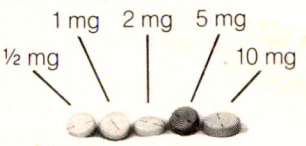
injection

A rapid-acting injection for psychiatric emergencies: 5 mg per ml,* with 1.8 mg methylparaben and 0.2 mg propylparaben per ml, and lactic acid for pH adjustment to 3.4 ± 0.2.



tablets

5 tablet strengths for convenience in individualizing dosage:



HALDOL® (haloperidol)

tablets / concentrate / injection

A dosage form for every therapeutic need

Summary of Prescribing Information

Contraindications: Severe depression, coma, CNS depression due to centrally-acting depressants, Parkinson's disease, hypersensitivity to the drug.

Warnings: ***Usage in Pregnancy:*** Safe use in pregnancy or in women likely to become pregnant has not been established; use only if benefit clearly justifies potential hazards. Infants should not be nursed during drug treatment.

Usage in Children: Safety and effectiveness not established; not recommended in pediatric age group.

Combined Use With Lithium: Patients receiving lithium plus haloperidol should be monitored closely for early evidence of neurological toxicity.

General: Bronchopneumonia, sometimes fatal, has followed use of major tranquilizers, including haloperidol. Prompt remedial therapy should be instituted if dehydration, hemoconcentration or reduced pulmonary ventilation occurs, especially in the elderly. Decreased serum cholesterol and/or cutaneous and ocular changes have been reported with chemically-related drugs, although not with haloperidol. Mental and/or physical abilities required for hazardous tasks or driving may be impaired. Alcohol should be avoided due to possible additive effects and hypotension.

Precautions: Administer cautiously to patients: (1) with severe cardiovascular disorders, due to the possibility of transient hypotension and/or precipitation of anginal pain (if a vasopressor is required, epinephrine should not be used since HALDOL haloperidol may block its vasopressor activity and paradoxical further lowering of blood pressure may occur); (2) receiving anticonvulsant medication since HALDOL haloperidol may lower the convulsive threshold; (3) with known allergies or a history of allergic reactions to drugs; (4) receiving anticoagulants. Concomitant antiparkinson medication, if required, may have to be continued after HALDOL haloperidol is discontinued because of different excretion rates; if both are discontinued simultaneously, extrapyramidal symptoms may occur. Intraocular pressure may increase when anticholinergic drugs, including antiparkinson drugs, are administered concomitantly with HALDOL haloperidol. When HALDOL haloperidol is used for mania in cyclic disorders, there may be a rapid mood swing to depression. Severe neurotoxicity may occur in patients with thyrotoxicosis receiving antipsychotic medication, including HALDOL haloperidol.

Adverse Reactions: ***CNS Effects:*** *Extrapyramidal Reactions:* Neuromuscular (extrapyramidal) reactions have been reported frequently, often during the first few days of treatment. Generally they involved Parkinson-like symptoms which were usually mild to moderately severe and usually reversible. Other types of neuromuscular reactions (motor restlessness, dystonia, akathisia, hyperreflexia, opisthotonos, oculogyric crises) have been reported far less frequently, but were often more severe. Severe extrapyramidal reactions have been reported at relatively low doses. Generally, extrapyramidal symptoms are dose-related since they occur at relatively high doses and disappear or become less severe when the dose is reduced. Antiparkinson drugs may be required. Persistent extrapyramidal reactions have been reported and the drug may have to be discontinued in such cases.

Withdrawal Emergent Neurological Signs: Abrupt discontinuation of short-term antipsychotic therapy is generally uneventful. However, some patients on maintenance treatment experience transient dyskinetic signs after abrupt withdrawal. In certain cases these are indistinguishable from "Persistent Tardive Dyskinesia" except for duration. It is unknown whether gradual withdrawal will reduce the occurrence of these signs, but until further evidence is available haloperidol should be gradually withdrawn.

Persistent Tardive Dyskinesia: Although rarely reported with HALDOL haloperidol, tardive dyskinesia may appear during or after long-term therapy. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and sometimes appear irreversible; there is no known effective treatment and all antipsychotic agents should be discontinued. The syndrome may be masked by reinstitution of drug, increasing dosage, or switching to a different antipsychotic agent.

Other CNS Effects: Insomnia, restlessness, anxiety, euphoria, agitation, drowsiness, depression, lethargy, headache, confusion, vertigo, grand mal seizures, and exacerbation of psychotic symptoms.

Cardiovascular Effects: Tachycardia and hypotension. ***Hematologic Effects:*** Reports of mild, usually transient leukopenia and leukocytosis, minimal decreases in red blood cell counts, anemia, or a tendency toward lymphomonocytosis; agranulocytosis rarely reported and only in association with other medication. ***Liver Effects:*** Impaired liver function and/or jaundice reported. ***Dermatologic Reactions:*** Maculopapular and acneiform reactions, isolated cases of photosensitivity, loss of hair. ***Endocrine Disorders:*** Lactation, breast engorgement, mastalgia, menstrual irregularities, gynecomastia, impotence, increased libido, hyperglycemia and hypoglycemia. ***Gastrointestinal Effects:*** Anorexia, constipation, diarrhea, hypersalivation, dyspepsia, nausea and vomiting. ***Autonomic Reactions:*** Dry mouth, blurred vision, urinary retention and diaphoresis. ***Respiratory Effects:*** Laryngospasm, bronchospasm and increased depth of respiration.

The injectable form is intended only for acutely agitated psychotic patients with moderately severe to very severe symptoms.

Caution: Federal law prohibits dispensing without prescription.

Full directions for use should be read before HALDOL haloperidol is administered or prescribed.

HALDOL tablets are manufactured by McNeil Laboratories Co., Dorado, Puerto Rico 00646.

* haloperidol present as the lactate

McNEIL

McNeil Laboratories, McNEILAB, Inc.
Fort Washington, PA 19034

Brief Summary

INDICATIONS — For management of anxiety disorders or short-term relief of symptoms of anxiety. Anxiety or tension associated with stress of everyday life usually does not require treatment with an anxiolytic.

Effectiveness in long-term use (over 4 months) not assessed by systematic clinical studies. Physician should periodically reassess usefulness for each patient.

For symptomatic relief of acute alcohol withdrawal.

CONTRAINDICATIONS — Known hypersensitivity to the drug. Acute narrow angle glaucoma.

WARNINGS — Not for use in depressive neuroses or psychotic reactions. Caution patients against hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles. Advise against simultaneous use of other CNS depressants, and caution patients that effects of alcohol may be increased. Not recommended for patients under 18. Nervousness, insomnia, irritability, diarrhea, muscle aches, and memory impairment have followed abrupt withdrawal from long-term high dosage. Withdrawal symptoms were reported after abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months. Use caution in patients having psychological potential for drug dependence (dependence has been observed in dogs and rabbits).

Pregnancy and Lactation: Minor tranquilizers should almost always be avoided first trimester. Consider possibility of pregnancy before initiating therapy. Patient should consult physician about discontinuation if she becomes pregnant or plans pregnancy. Do not give to nursing mothers.

PRECAUTIONS — Observe usual precautions in depression accompanying anxiety, or in patients with suicidal tendency, or those with impaired renal or hepatic function. Do periodic blood counts and liver function tests during prolonged therapy. Use small doses and gradual increments in the elderly or debilitated.

ADVERSE REACTIONS — Drowsiness, dizziness, various g.i. complaints, nervousness, blurred vision, dry mouth, headache, mental confusion, insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression, slurred speech, abnormal liver and kidney function, decreased hematocrit, decreased systolic blood pressure.

DOSAGE — ANXIETY — Usual daily dose 30 mg or less (start the elderly or debilitated at 7.5-15 mg). Adjust gradually within 15-60 mg daily range. Capsules and scored tablets: divided doses; or once daily h.s. (start patient at 15 mg). Single dose tablets, 22.5 mg (for patients stabilized on 7.5 mg t.i.d.) or 11.25 mg; once daily at any hour. ALCOHOL WITHDRAWAL— In divided doses: 1st day 30 mg initially, then 30-60 mg; 2nd day 45-90 mg; 3rd day 22.5-45 mg; 4th day 15-30 mg. Then taper to 15-7.5 mg daily, and discontinue as soon as stable.

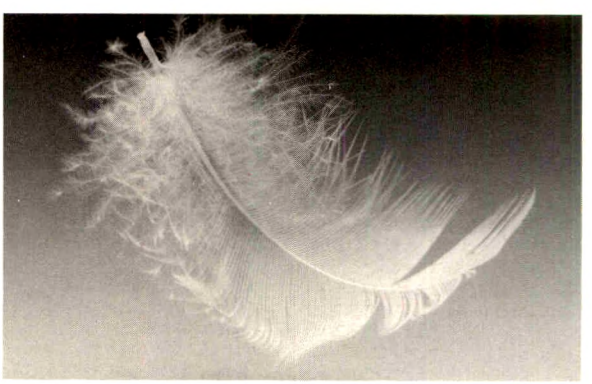
INTERACTIONS — Potentiation may occur with ethyl alcohol, hypnotics, barbiturates, narcotics, phenothiazines, MAO inhibitors, other antidepressants. In bioavailability studies with normal subjects, concurrent administration of antacids at therapeutic levels did not significantly influence bioavailability of TRANXENE.

OVERDOSAGE — Take general measures as for any CNS depressant.

SUPPLIED — Tranxene (clorazepate dipotassium) 3.75, 7.5, and 15 mg capsules and scored tablets. Tranxene-SD Half Strength 11.25 and Tranxene-SD 22.5 mg single dose tablets.

REFERENCES — 1. The elimination kinetics of an agent can be closely defined, but cannot at present be directly related to therapeutic or adverse effects. 2. Sedation reported in normal volunteers 30 minutes after dosing. TRANXENE Drug Monograph 97-0185, 1979, p. 9.
3. Op cit., p 15.

How therapy for anxiety stops is as important as how it starts.



Tranxene® (IV)

clorazepate dipotassium

Starts promptly... stops gently

Abbott Laboratories/
Abbott Pharmaceuticals, Inc.
North Chicago, IL 60064


How therapy for anxiety stops is as important as how it starts.
When treatment with TRANXENE® *concludes*, nordiazepam levels taper gently.[1] This built-in tapering effect may give patients time to adjust to post-therapy — an important consideration when choosing an agent for short-term management of anxiety.
Yet, TRANXENE *starts* fast. Pharmacologic effects are observed in just half an hour.[2] Peak serum levels are reached in 1-2 hours.
TRANXENE offers predictable calming action.[3] In 25 short-term studies, positive therapeutic response ranging from 83% to 90% was obtained. TRANXENE. A logical first choice for short-term therapy.
Tranxene® IV
clorazepate dipotassium
Starts promptly... stops gently
See overleaf for brief summary.

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By Maria Kovacs

The Ethics of Psychotherapy
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'STELAZINE' HELPS FIND A WAY THROUGH

The schizophrenic patient is often withdrawn, aloof and emotionally unresponsive. For such patients, 'Stelazine' provides effective, unsurpassed control of these and other psychotic symptoms.

Also important, 'Stelazine' can help patients participate in your therapeutic programs. 'Stelazine' usually does not cause excessive sedation and offers the convenience of b.i.d. dosage.

In fact, no other antipsychotic agent has demonstrated significantly greater overall effectiveness and significantly fewer adverse effects than 'Stelazine'.

'Stelazine'. A first choice for therapy.

Before prescribing, see complete prescribing information in SK&F Co. literature or PDR. The following is a brief summary.

Indications

Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:
Effective: For the management of the manifestations of psychotic disorders.
Possibly effective: To control excessive anxiety, tension and agitation as seen in neuroses or associated with somatic conditions.
'Stelazine' has not been shown effective in the management of behavioral complications in patients with mental retardation.
Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose or greatly depressed states due to C.N.S. depressants; blood dyscrasias; bone marrow depression; liver damage.

Warnings: Generally avoid using in patients hypersensitive (e.g., blood dyscrasias, jaundice) to any phenothiazine. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery), especially during the first few days' therapy.

Additive effect is possible with other C.N.S. depressants, including alcohol.

Use in pregnancy only when necessary for patient's welfare.

Precautions: Use cautiously in angina. Avoid high doses and parenteral administration when cardiovascular system is impaired. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Prolonged administration of high doses may result in cumulative effects with severe C.N.S. or vasomotor symptoms. If retinal changes occur, discontinue drug. Agranulocytosis, thrombocytopenia, pancytopenia, anemia, cholestatic jaundice, liver damage have been reported.

Patients with a history of long-term therapy with 'Stelazine' and/or other neuroleptics should be evaluated periodically for possible adjustment or discontinuance of drug therapy. Neuroleptic drugs cause elevated prolactin levels that persist during chronic administration. Since approximately one-third of human breast cancers are prolactin-dependent in vitro, this elevation is of potential importance if neuroleptic drug administration is contemplated in a patient with a previously detected breast cancer. Neither clinical nor epidemiologic studies to date, however, have shown an association between the chronic administration of neuroleptic drugs and mammary tumorigenesis.

Adverse Reactions: Drowsiness, dizziness, skin reactions, rash, dry mouth, insomnia, amenorrhea, fatigue, muscular weakness, anorexia, lactation, blurred vision. Neuromuscular (extrapyramidal) reactions: motor restlessness, dystonias, pseudoparkinsonism, persistent tardive dyskinesia.

Other adverse reactions reported with Stelazine (trifluoperazine HCl, SK&F) or other phenothiazines: Some adverse effects are more frequent or intense in specific disorders (e.g., mitral insufficiency or pheochromocytoma).

Grand mal convulsions; altered cerebrospinal fluid proteins; cerebral edema; prolongation and intensification of the action of C.N.S. depressants, atropine, heat, and organophosphorus insecticides; nasal congestion, headache, nausea, constipation, obstipation, adynamic ileus, inhibition of ejaculation; reactivation of psychotic processes, catatonic-like states; hypotension (sometimes fatal); cardiac arrest; leukopenia, eosinophilia, pancytopenia, agranulocytosis, thrombocytopenic purpura; jaundice; biliary stasis; menstrual irregularities, galactorrhea, gynecomastia, false positive pregnancy tests; photosensitivity, itching, erythema, urticaria, eczema up to exfoliative dermatitis; asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions, peripheral edema; reversed epinephrine effect; hyperpyrexia; a systemic lupus erythematosus-like syndrome; pigmentary retinopathy; with prolonged administration of substantial doses, skin pigmentation, epithelial keratopathy, and lenticular and corneal deposits. EKG changes have been reported, but relationship to myocardial damage is not confirmed. Discontinue long-term, high-dose therapy gradually. NOTE: Sudden death in patients taking phenothiazines (apparently due to cardiac arrest or asphyxia due to failure of cough reflex) has been reported; but no causal relationship has been established.

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As she sleeps... her antidepressant goes to work.

Promptly improves sleep patterns.
In a recent multicenter study among 89 depressed outpatients, a substantial majority had improved sleep patterns in the first week. Peak antidepressant effects occurred at 3 to 4 weeks.[1]



Relieves fatigue and anxiety.

Improved sleep generally lessens daytime fatigue. And the prompt anxiety-reducing effect of Surmontil[2] can help patients function better in the first weeks of therapy.

Low incidence of side effects.[3-5]

Effective in single nighttime dose after initial dosage titration.[2]

Before prescribing, please see complete product information, a summary of which follows:

CONTRAINDICATIONS: Contraindicated in cases of known hypersensitivity to the drug, and during the acute recovery period after myocardial infarction. The possibility of cross-sensitivity to other dibenzazepine compounds should be kept in mind. Surmontil should not be given in conjunction with drugs of the monoamine oxidase inhibitor (MAOI) class. At least two weeks should elapse between cessation of therapy with an MAOI and institution of therapy with Surmontil (trimipramine maleate).

WARNINGS: Children: This drug is not recommended for use in children, since safety and effectiveness in the pediatric age group have not been established. **Adults:** Use extreme caution in giving the drug to patients with evidence of cardiovascular disease. Caution is advised in patients with: increased intraocular pressure, history of urinary retention, narrow-angle glaucoma, seizure disorder, hyperthyroidism, a need for thyroid medication. In patients receiving guanethidine or similar agents, Surmontil may block the pharmacologic effects of these drugs. Warn patients that the drug may impair the mental or physical abilities required for driving or performing other potentially hazardous tasks.

PRECAUTIONS: Because of an inherently serious suicide potential, the nonhospitalized severely depressed patient should be given the smallest drug amount feasible. In schizophrenic patients, activation of the psychosis may occur and require reduction of dosage or the addition of a major tranquilizer to the medication schedule. Manic or hypomanic episodes may occur, especially in patients with cyclic-type disorders; Surmontil may have to be discontinued until the episode is relieved and reinstituted, if required, at lower dosage. Limit concurrent administration of Surmontil and electroconvulsive therapy to those patients for whom it is essential. When possible, discontinue the drug for several days prior to elective surgery. The use of alcoholic drinks during therapy may provoke exaggerated response. Potentiation of effects has been reported when tricyclic antidepressants were administered with sympathomimetic amines, local decongestants, local anesthetics containing epinephrine, atropine, or drugs with an anticholinergic effect. Drugs having a parasympathetic effect, including tricyclic antidepressants, may alter ejaculatory response.

Usage in pregnancy: Pregnancy Category C. Surmontil has shown evidence of embryotoxicity and/or increased incidence of major anomalies in rats or rabbits at doses 20 times the human dose. There are no adequate and well-controlled studies in pregnant women. Surmontil should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

ADVERSE REACTIONS: When tricyclic antidepressants are used, each of the following adverse reactions must be considered, although some have not in fact been reported with Surmontil.

Cardiovascular—Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric—Confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurologic—Numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures, alterations in EEG patterns; tinnitus.

Anticholinergic—Dry mouth and, rarely, associated sublingual adenitis; blurred vision, disturbances of accommodation, mydriasis, constipation, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic—Skin rash, petechiae, urticaria, itching, photosensitization, edema of face and tongue.

Hematologic—Bone-marrow depression including agranulocytosis, eosinophilia; purpura; thrombocytopenia. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathologic neutrophil depression.

Gastrointestinal—Nausea and vomiting, anorexia, epigastric distress, diarrhea, peculiar taste, stomatitis, abdominal cramps, black tongue.

Endocrine—Gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido, impotence; testicular swelling; elevation or depression of blood-sugar levels.

Other—Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency; drowsiness, dizziness, weakness, and fatigue; headache; parotid swelling; alopecia.

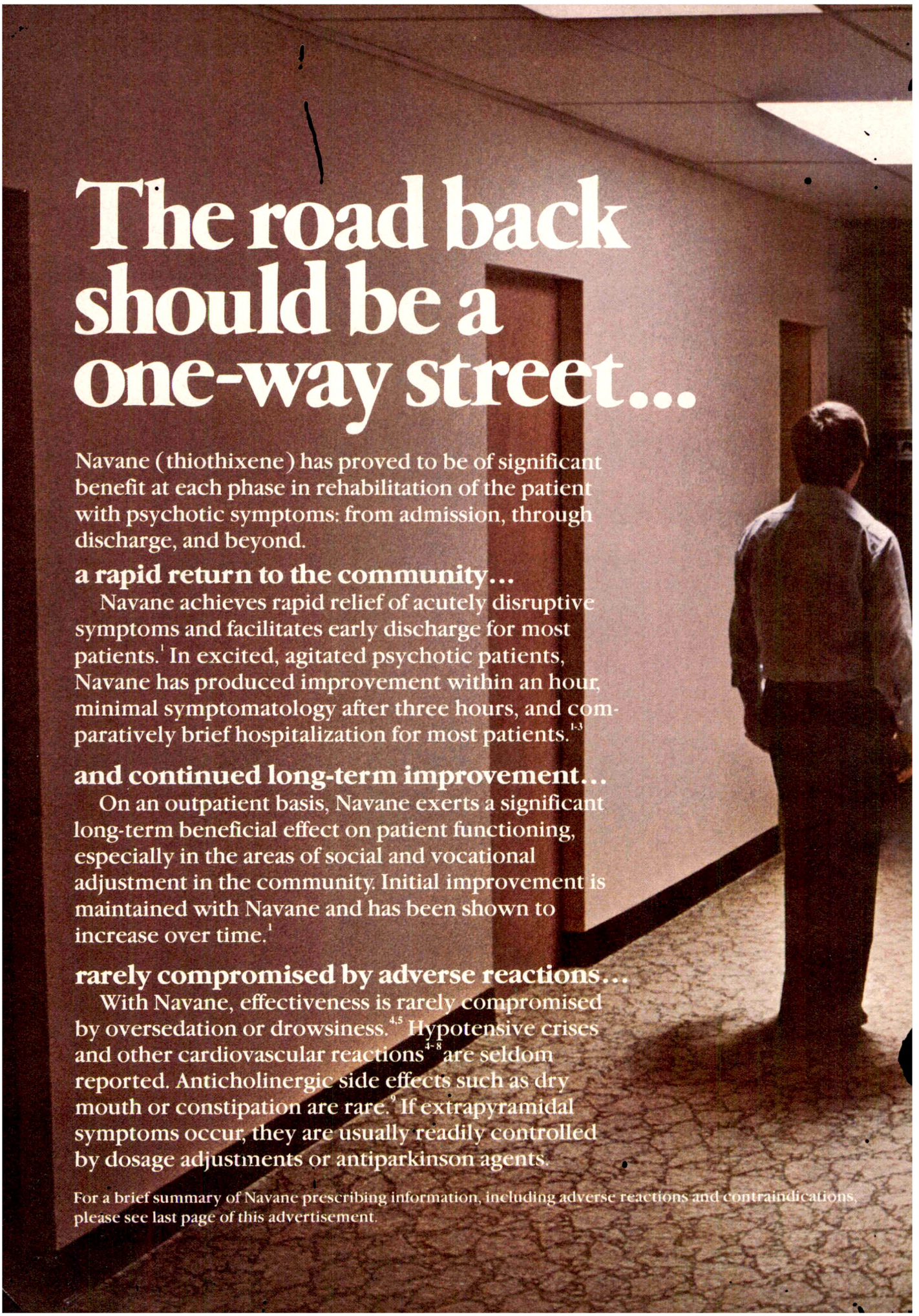
Withdrawal Symptoms—Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache, and malaise.

SUPPLIED: 25 mg in bottles of 100 opaque blue and yellow capsules, 50 mg in bottles of 100 opaque blue and orange capsules.

References: 1. Goulton J, Baker PG, Wilkinson MA: A multicentre general practice study of 'Surmontil' (trimipramine maleate) in the treatment of endogenous depression with associated sleep disturbances. *Br J Clin Pract* 32:323-325, 1978. **2.** Pecknold JC, Ananth J: Trimipramine in the treatment of anxious-depressed out-patients. *Curr Ther Res* 25:94-100, 1978. **3.** Lean TH, Sidhu MS: Comparative study of imipramine (Tofranil) and trimipramine (Surmontil) in depression associated with gynaecological conditions. *Proc Obstet Gynaecol Soc* 3:222-228, 1972. **4.** Evans JI, et al: General practitioner clinical trials: two new psychotropic drugs. *Practitioner* 198 (27):135-139, 1967. **5.** Salzmann MM: A controlled trial with trimipramine, a new anti-depressant drug. *Br J Psychiatry* 111:1105-1106, 1965.

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Navane (thiothixene) has proved to be of significant benefit at each phase in rehabilitation of the patient with psychotic symptoms: from admission, through discharge, and beyond.

a rapid return to the community...

Navane achieves rapid relief of acutely disruptive symptoms and facilitates early discharge for most patients.[1] In excited, agitated psychotic patients, Navane has produced improvement within an hour, minimal symptomatology after three hours, and comparatively brief hospitalization for most patients.[1-3]

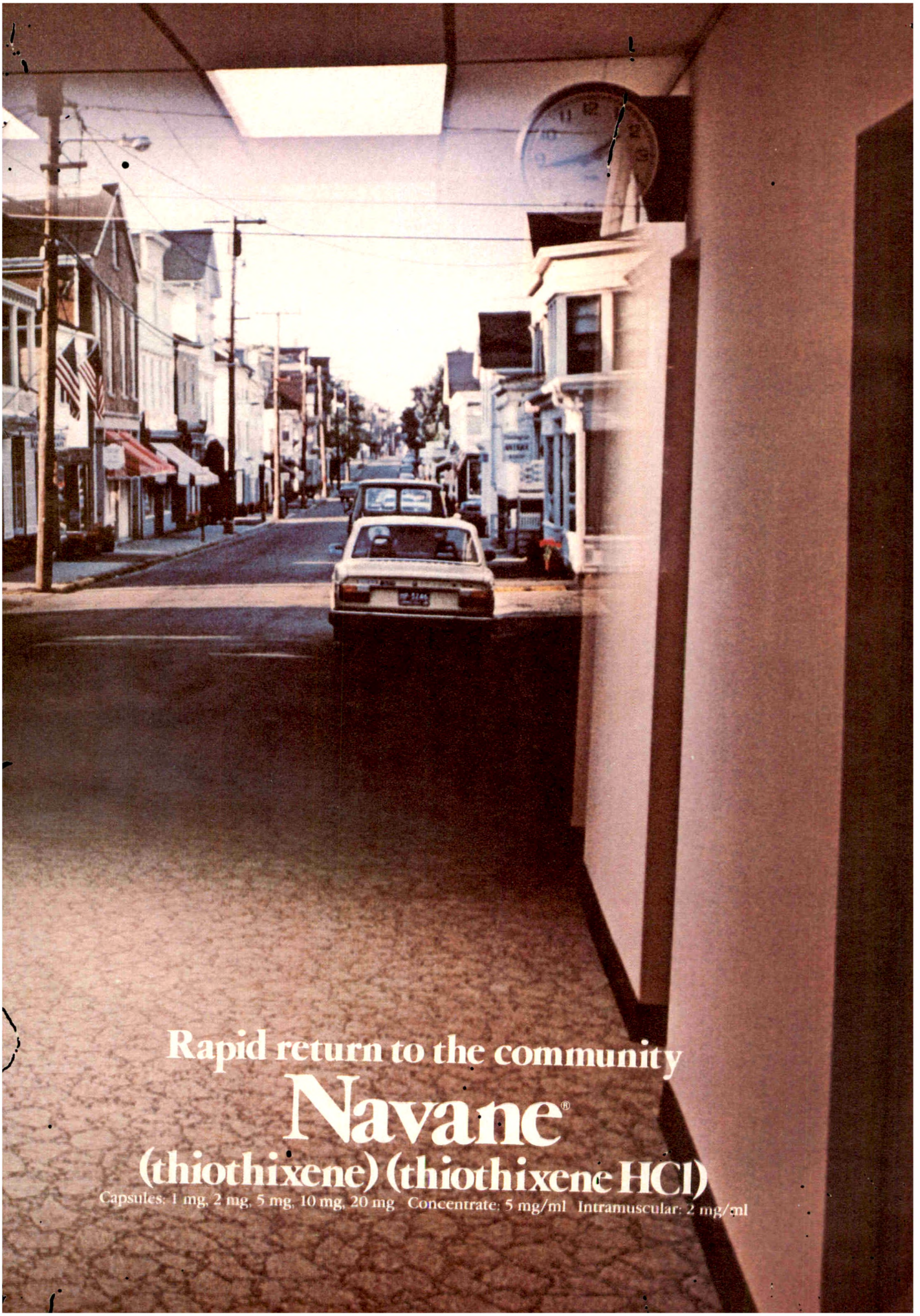
and continued long-term improvement...

On an outpatient basis, Navane exerts a significant long-term beneficial effect on patient functioning, especially in the areas of social and vocational adjustment in the community. Initial improvement is maintained with Navane and has been shown to increase over time.[1]

rarely compromised by adverse reactions...

With Navane, effectiveness is rarely compromised by oversedation or drowsiness.[4,5] Hypotensive crises and other cardiovascular reactions[4-8] are seldom reported. Anticholinergic side effects such as dry mouth or constipation are rare.[9] If extrapyramidal symptoms occur, they are usually readily controlled by dosage adjustments or antiparkinson agents.

For a brief summary of Navane prescribing information, including adverse reactions and contraindications, please see last page of this advertisement.


Rapid return to the community
Navane®
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Rapid return to the community

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Contraindications. Navane (thiothixene) is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy*– Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration of Navane to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children– The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration– As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane (thiothixene). Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudo-parkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leukocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution–For Intramuscular Use Only. Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane (thiothixene) Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate– In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Stimulants that may cause convulsions (e.g. picrotoxin or pentylenetetrazol) should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg of thiothixene in bottles of 100, 1,000, and unit-dose pack of 100 (10 x 10's). Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100, 500, and unit-dose pack of 100 (10 x 10's).

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg, and in 30 ml (1 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, and 5 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10 vials. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

References: 1. Rickels K, Norstad N, Downing R: The acutely decompensated schizophrenic patient, presented as a scientific exhibit at the American Psychiatric Association 28th Institute on Hospital and Community Psychiatry, September 20-22, 1976, Atlanta, Ga. 2. Stotsky BA: Relative efficacy of parenteral haloperidol and thiothixene for the emergency treatment of acutely excited and agitated patients. *Dis Nerv Syst* 38:967-973, Dec 1977. 3. Sloan RB, Razani J, Maloney MP, et al: Premorbid adjustment, cognition and outcome in schizophrenia, presented as a scientific exhibit at the 128th Annual Meeting of the American Psychiatric Association, May 5-9, 1975, Anaheim, California. 4. Engelhardt DM, Rudorfer L, Rosen B: Haloperidol and thiothixene in the long-term treatment of chronic schizophrenic outpatients in an urban community: Social and vocational adjustment. *J Clin Psychiatry* 39:834-840, Dec 1978. 5. Itil TM, Unverdi C, Wohlrabe J, et al: Drug therapy of psychosis associated with organic brain syndrome, presented as a scientific exhibit at the American Public Health Association Centennial, Atlantic City, New Jersey, Nov 12-16, 1972. 6. Brauzer B, Goldstein BJ: Comparative effects of intramuscular thiothixene and trifluoperazine in psychotic patients: *J Clin Pharmacol* 8:400-403, Nov-Dec 1968. 7. Dillenkoffer RL, Gallant DM, George RB, et al: Electrocardiographic evaluations of schizophrenic patients, presented as a scientific exhibit at the 125th Annual Meeting of the American Psychiatric Association, Dallas, Texas, May 1-4, 1972. 8. Data on file at Roerig. 9. Goldstein B, Weiner D, Banas R: Clinical evaluation of thiothixene in chronic ambulatory schizophrenic patients, in Lehmann HE, Ban TA (eds): *Modern Problems in Pharmacopsychiatry.* Basel, S Karger, 1969, vol 2, pp 45-52.

For additional information on Navane, please consult your Roerig representative or write to: Roerig Medical Department, 235 East 42nd Street, New York, NY 10017.

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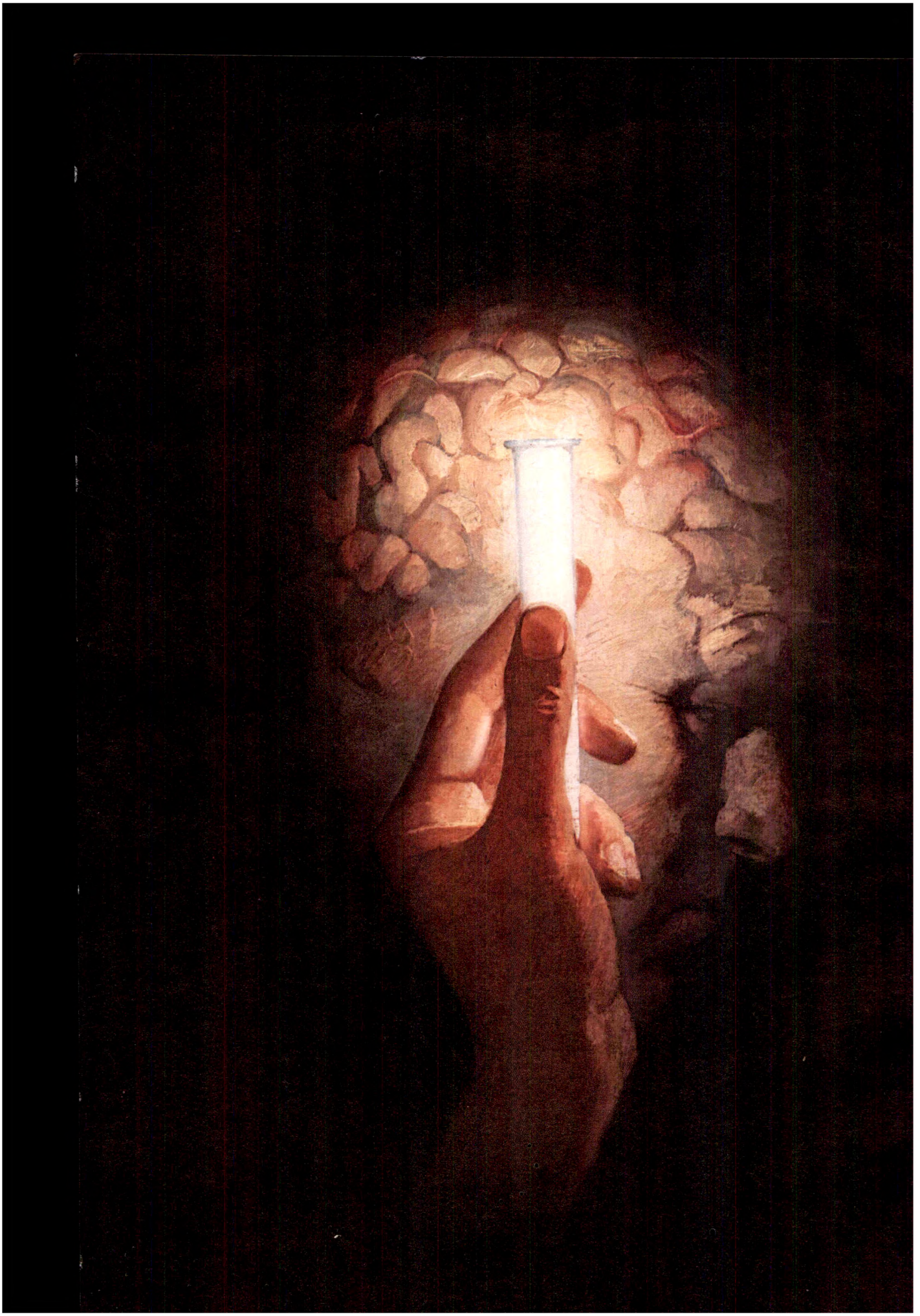
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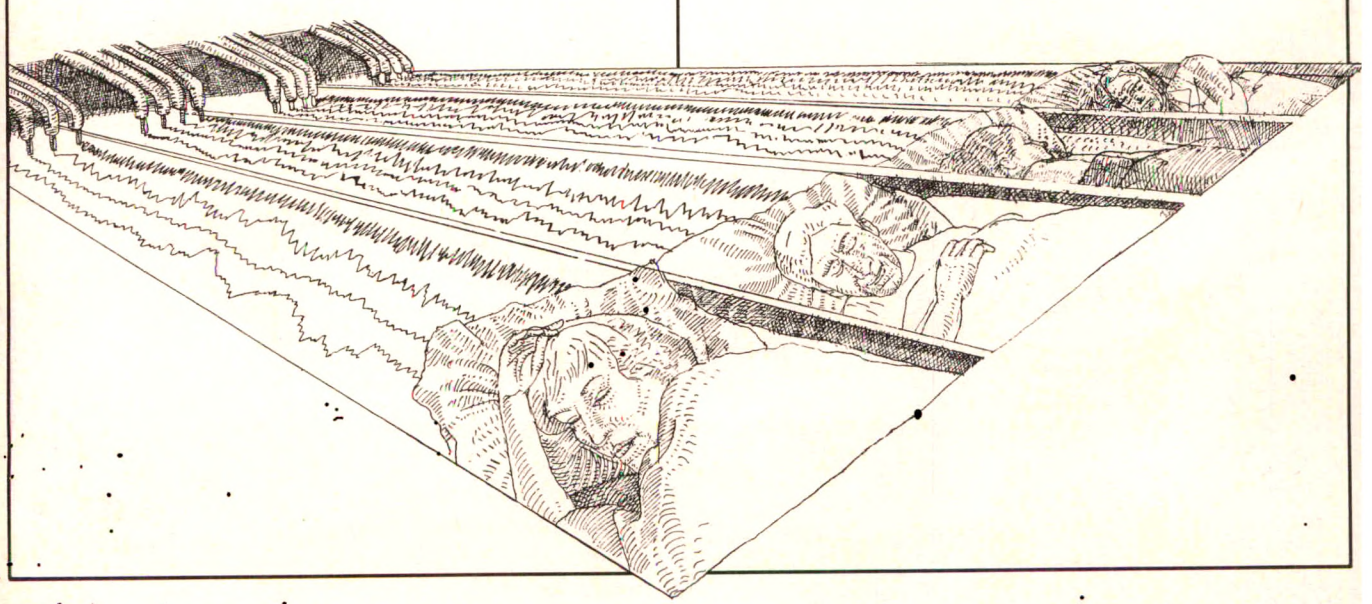
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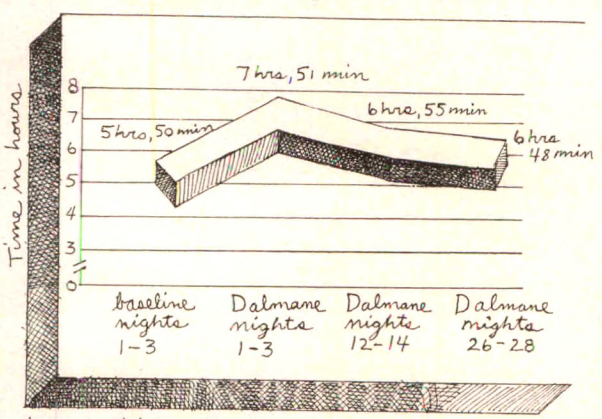
Dalmane (flurazepam HCl/Roche) is the only sleep medication objectively proven effective through 28 consecutive nights of administration in two separate studies in the sleep research laboratory.[2-4] The prolonged use of Dalmane is seldom necessary; should it be, appropriate patient evaluations such as periodic blood counts and liver and kidney function tests are recommended.



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Dalmane® (flurazepam HCl/Roche) improved sleep during short, intermediate and longer-term use



Improvement shown represents mean improvement, in hours, of total sleep time. n=5

Adapted from Dement WC, *et al: Behav Med* 5:25-31, Oct 1978

Since most insomnia is transitory in nature, initiate therapy at the lowest effective dose for the shortest time advisable to achieve a predetermined therapeutic goal.

References:

1. Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley NJ
2. Kales A, *et al: Clin Pharmacol Ther* 19: 576-583, May 1976
3. Dement WC, *et al: Behav Med* 5: 25-31, Oct 1978
4. Kales A, *et al: Clin Pharmacol Ther* 18: 356-363, Sep 1975

Clinical documentation of efficacy in 4950 patients with insomnia[1]

Dalmane (flurazepam HCl/Roche) efficacy was evaluated in 18 clinical studies. These included paired-night, double-blind crossover, double-blind comparison and patient preference studies. Results of these studies demonstrated and confirmed the effectiveness of Dalmane.

Periodic evaluation of patient's sleep habits may help prevent subsequent insomnia

Should insomnia recur, the patient may need guidance in setting up a regular sleep program. This may include establishment of a regular bedtime hour, taking a warm bath prior to retiring and avoidance of alcohol, stimulants or heavy meals late in the evening.

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Protocol for insomnia management

☐ Treat physical and/or psychological factors that may underlie an inability to sleep.

☐ Thoroughly question and evaluate new patients who specifically request sleep medications.

☐ Consider other medications the patient may be taking (including alcoholic beverages) and be aware of possible drug interactions.

☐ Select a sleep medication with objectively *documented* proof of efficacy and a wide margin of safety.

☐ Initiate treatment at the lowest effective dose for the period of time deemed necessary *to achieve a reasonable therapeutic goal.*

☐ Monitor response to therapy and, based upon patient reassessment, determine need for continuing medication on an individual basis.

☐ Advise patients who ingest alcoholic beverages or other CNS depressants against doing so while on sleep medication. CNS stimulants such as caffeine should also be discontinued.

☐ Caution patients against engaging in hazardous occupations requiring complete mental alertness such as operating machinery or driving a motor vehicle shortly after ingesting the drug. In addition, as with all CNS depressants, patients may have reduced motor performance while on hypnotic therapy and should be advised accordingly.

☐ Avoid prescribing for suicidal or dependency-prone individuals whose histories indicate an inability to handle any psychoactive substances including alcohol.

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DA-5T

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One 30-mg capsule *h.s.*—usual adult dosage
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Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Objective sleep laboratory data have shown effectiveness for at least 28 consecutive nights of administration. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended.
Contraindications: Known hypersensitivity to flurazepam HCl.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).
Usage in Pregnancy: **Several studies of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) suggest increased risk of congenital malformations during the first trimester of pregnancy. Dalmane, a benzodiazepine, has not been studied adequately to determine whether it may be associated with such an increased risk. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.
Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies, or with impaired renal or hepatic function. Periodic blood counts and liver and kidney function tests are advised during repeated therapy.
Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase.
Dosage: Individualize for maximum beneficial effect.
Adults: 30 mg usual dosage; 15 mg may suffice in some patients.
Elderly or debilitated patients: 15 mg recommended initially until response is determined.
Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

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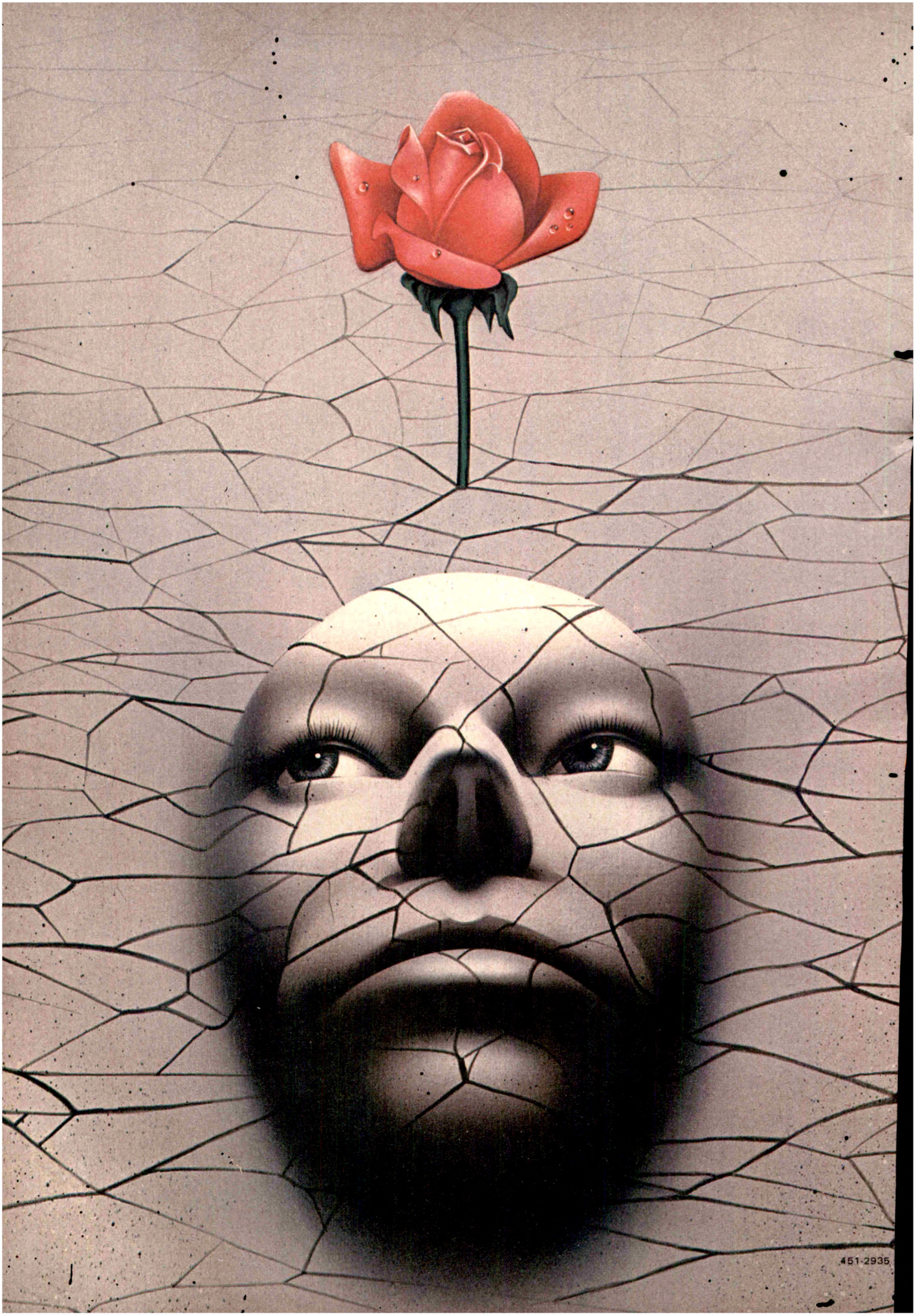
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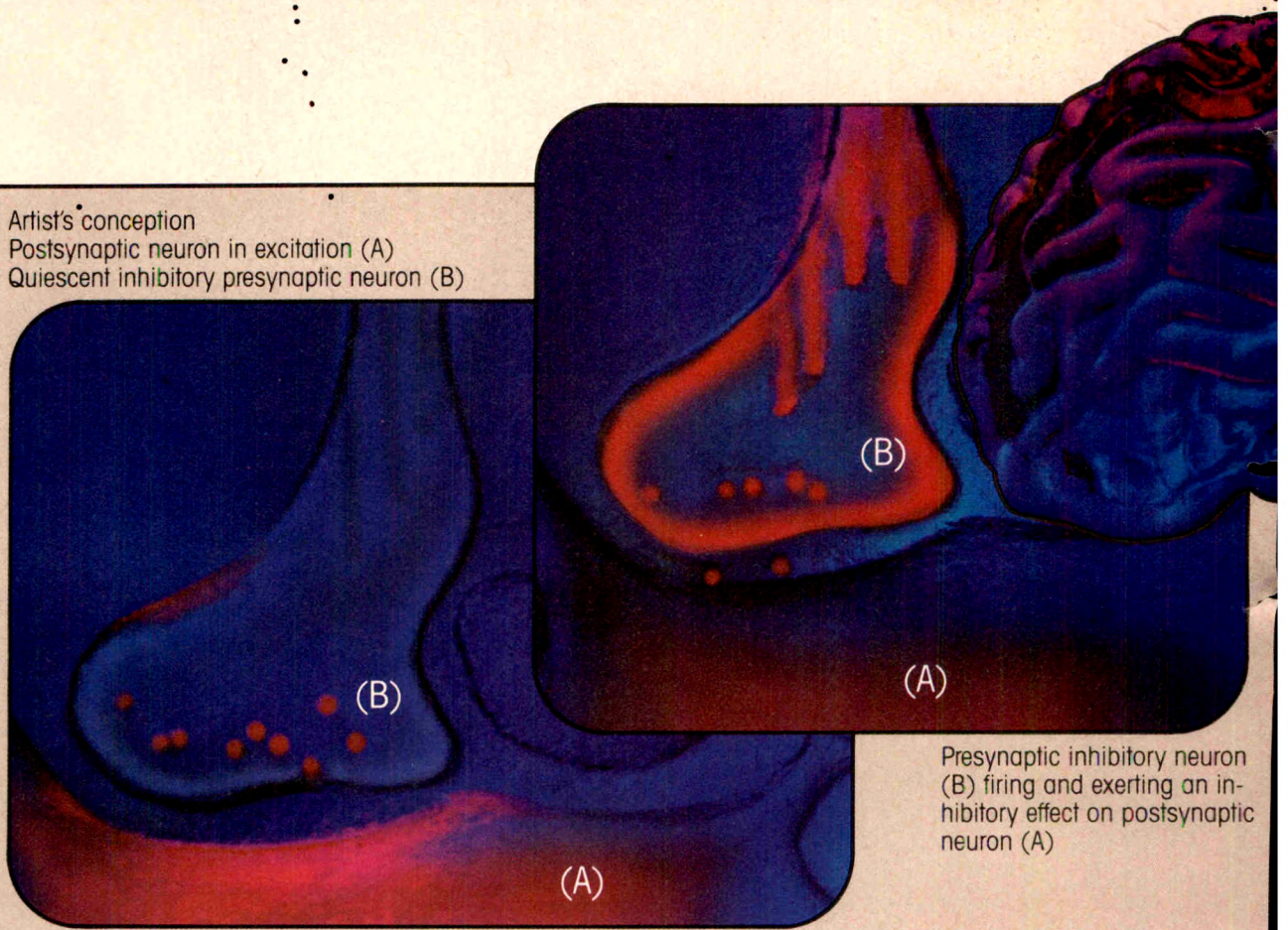
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Artist's conception
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Quiescent inhibitory presynaptic neuron (B)

Presynaptic inhibitory neuron (B) firing and exerting an inhibitory effect on postsynaptic neuron (A)

Research in Progress

points to the role of specific CNS binding sites for the benzodiazepines — often considered the most effective anxiolytic agents[1]

Although the clinical effectiveness of the benzodiazepines is widely accepted by the medical profession, their mechanism of action is as yet unknown. Inability to define a neurophysiological or biochemical basis of anxiety has hindered the search.[2]

Current research has, however, identified specific benzodiazepine binding sites in the CNS. Various characteristics of these sites suggest that they may represent the actual sites of action of these agents.[3,4]

Specific tenacity of benzodiazepines to CNS binding sites

In equilibrium binding studies on rat and human brain tissue, benzodiazepines demonstrate a high affinity for specific sites found primarily in the synaptic membrane fraction. Of the compounds tested, only benzodiazepines compete for these binding sites. Furthermore, each benzodiazepine displays a characteristic degree of affinity which closely parallels its clinical and pharmacological potency.[3,4]

A significant correlation has also been found between the binding affinity of a particular benzodiazepine and its anxiolytic, anticonvulsant and muscle relaxant effects in certain animal tests.[4]

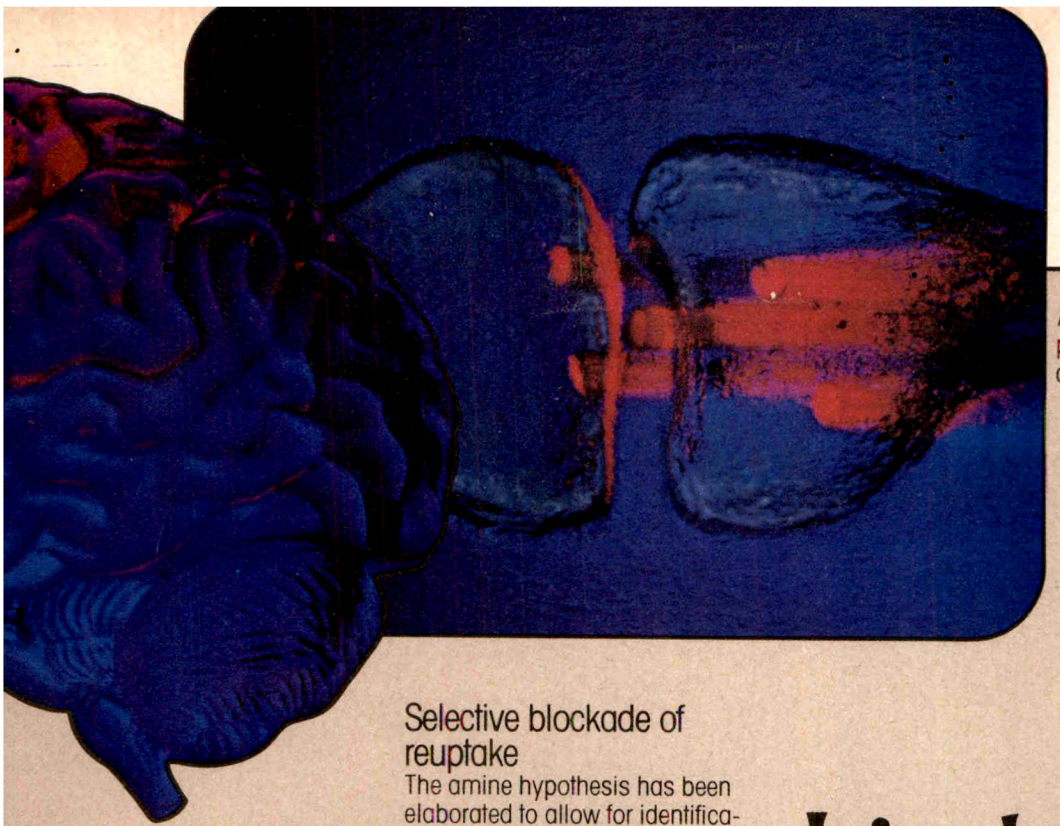
Although these *in vitro* animal and human tissue studies do not necessarily suggest clinical significance, further investigation of specific benzodiazepine binding sites may provide insight into the mechanism of action of these clinically effective anxiolytic agents.

References: 1. Rickels K: Drug treatment of anxiety, in *Psychopharmacology in the Practice of Medicine*, edited by Jarvik ME. New York, Appleton-Century-Crofts, 1977, p. 312. **2.** Byck R: Drugs and the treatment of psychiatric disorders, chap. 12, in *The Pharmacological Basis of Therapeutics*, ed. 5, edited by Goodman LS, Gilman A. New York, Macmillan Publishing Co., Inc., 1975, p. 190. **3.** Möhler H, Okada T: *Life Sci 20* (12): 2101-2110, 1977. **4.** Möhler H, *et al*: *Life Sci 22* (11): 985-996, 1978. **5.** Hollister LE: *N Engl J Med 299*: 1106-1109, Nov 16, 1978.

While *in vitro* studies cannot be interpreted to have clinical significance, these represent an attempt to explain

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Artist's conception of propagation of impulse across synaptic cleft.

and to the role of tricyclic antidepressants in blocking the presynaptic reuptake of neurotransmitters — believed important in the treatment of depression

The amine hypothesis, which holds that depression is associated with a deficiency of functional neurotransmitter at the synapse, is currently the most viable theory of depression. It is supported by studies on the pharmacologic action of various clinically effective antidepressants. The tricyclics, for instance, have been shown to block reuptake by the presynaptic terminals, permitting greater availability and prolonged action of neurotransmitters at receptor sites. This may compensate for the relative deficit of functional neurotransmitters found in depression.[5]

Selective blockade of reuptake

The amine hypothesis has been elaborated to allow for identification of subtypes of depression based on specific neurotransmitter deficiencies. It has been proposed that measurement of the metabolite 3-methoxy-4-hydroxy-phenylglycol (MHPG) in urine may permit such a differentiation; low MHPG levels indicating a deficit of norepinephrine, and normal or high MHPG suggesting a deficit of another neurotransmitter, probably serotonin. Current pharmacological studies indicate that certain tricyclics may be more efficient in blocking norepinephrine reuptake, while others more selectively blockade serotonin.[5] Further investigation may lead to eventual selection of subtype-specific antidepressants on the basis of biochemical tests.

Limbitrol® 5-12.5 10-25

Tablets 5-12.5 each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)
Tablets 10-25 each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt)

Dual action specific for dual symptomatology

Limbitrol contains both a benzodiazepine — specific for the symptoms of anxiety, and amitriptyline — specific for depressive symptoms. It therefore provides the agents most appropriate to relieve both symptom syndromes in the nonpsychotic patient with mixed anxiety and depression. What better rationale for prescribing Limbitrol for patients who need pharmacologic intervention in support of their psychotherapy.

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LIMBITROL® TABLETS Tranquilizer—Antidepressant

Before prescribing, please consult complete product information, a summary of which follows:
Indications: Relief of moderate to severe depression associated with moderate to severe anxiety.
Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants. Do not use with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Contraindicated during acute recovery phase following myocardial infarction.
Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs. Closely supervise cardiovascular patients. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses. Myocardial infarction and stroke reported with use of this class of drugs.)
Usage in Pregnancy: **Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage; withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline; symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).
Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Periodic liver function tests and blood counts are recommended during prolonged treatment. Amitriptyline component may block action of guanethidine or similar antihypertensives. Concomitant use with other psychotropic drugs has not been evaluated: sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Limbitrol should not be taken during the nursing period. Not recommended in children under 12.
In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects.
Adverse Reactions: Most frequently reported are those associated with either component alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline. Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely.
This list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs:
Cardiovascular: Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke.
Psychiatric: Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.
Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns.
Anticholinergic: Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract.
Allergic: Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus.
Hematologic: Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia.
Gastrointestinal: Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.
Endocrine: Testicular swelling and gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female and elevation and lowering of blood sugar levels.
Other: Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.
Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. I.V. administration of 1 to 3 mg physostigmine salicylate has been reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.
Dosage: Individualize according to symptom severity and patient response. Reduce to smallest effective dosage when satisfactory response is obtained. Larger portion of daily dose may be taken at bedtime. Single h.s. dose may suffice for some patients. Lower dosages are recommended for the elderly.
Limbitrol 10-25, initial dosage of three to four tablets daily in divided doses, increased up to six tablets or decreased to two tablets daily as required. Limbitrol 5-12.5, initial dosage of three to four tablets daily in divided doses, for patients who do not tolerate higher doses.
How Supplied: White, film-coated tablets, each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) and blue, film-coated tablets, each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50.

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☐ Limbitrol 5-12.5 is recommended to minimize drowsiness and for elderly patients
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☐ An initial dosage of three tablets is recommended
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☐ Once a satisfactory response is obtained, patients should be continued on the smallest dose required to maintain the desired effect

Utilize dosage options to best accommodate individual patient needs
☐ T.I.D. or Q.I.D., familiar regimens most suited for patients who tolerate medication without undue drowsiness
☐ Two tablets one hour before bedtime and one tablet midday may minimize daytime drowsiness and help relieve a common target symptom—insomnia
☐ Entire dosage h.s. to take maximum advantage of the sedative effect

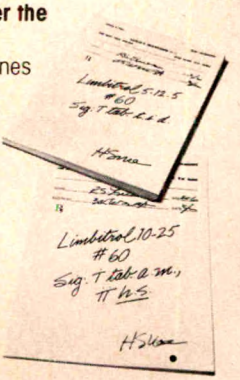
Your guide to patient management... when you decide medication is needed

How to make each patient an informed patient

1. Discuss with patients the probability that they will experience drowsiness, especially during the first week.
2. Reassure your patients that drowsiness is one indication that the medication is working and that it may help alleviate their insomnia.
3. Encourage patients to report if drowsiness becomes troublesome so that, if necessary, dosage schedule can be adjusted.
4. Caution patients about the combined effects with alcohol or other CNS depressants. Let them know that the additive effects may produce a harmful level of sedation and CNS depression.
5. Caution patients about activities requiring complete mental alertness, such as operating machinery or driving a car.
6. Warn pregnant patients and patients of childbearing age that the safety of Limbitrol in pregnancy has not yet been established.

Please see complete product disclosure for other pertinent information.

Limbitrol should not be used under the following circumstances:
1. Hypersensitivity to benzodiazepines or tricyclic antidepressants.
2. Concomitantly with an MAO inhibitor. To replace an MAO inhibitor with Limbitrol, discontinue MAO inhibitor for a minimum of 14 days before cautiously initiating Limbitrol therapy.
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Contraindications: Severe central nervous system depression, comatose states from any cause, hypertensive or hypotensive heart disease of extreme degree.

Warnings: Administer cautiously to patients who have previously exhibited a hypersensitivity reaction (e.g., blood dyscrasias, jaundice) to phenothiazines. Phenothiazines are capable of potentiating central nervous system depressants (e.g., anesthetics, opiates, alcohol, etc.) as well as atropine and phosphorus insecticides; carefully consider benefit versus risk in less severe disorders. During pregnancy, administer only when the potential benefits exceed the possible risks to mother and fetus.

Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy, observed primarily in patients receiving larger than recommended doses, is characterized by diminution of visual acuity, brownish coloring of vision, and impairment of night vision; the possibility of its occurrence may be reduced by remaining within recommended dosage limits. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving), and increase dosage gradually. Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension since phenothiazines may induce a reversed epinephrine effect on occasion.

Neuroleptic drugs elevate prolactin levels; the elevation persists during chronic administration. Tissue culture experiments indicate that approximately one-third of human breast cancers are prolactin dependent in vitro, a factor of potential importance if the prescription of these drugs is contemplated in a patient with a previously detected breast cancer. Although disturbances such as galactorrhea, amenorrhea, gynecomastia, and impotence have been reported, the clinical significance of elevated serum prolactin levels is unknown for most patients.

Daily doses in excess of 300 mg should be used only in severe neuropsychiatric conditions. FD&C Yellow No. 5 (tartrazine) in the 150-mg tablet may cause allergic-type reactions (including bronchial asthma), particularly in patients with aspirin hypersensitivity.

Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; rarely, nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—ECG changes (see *Cardiovascular Effects* below). *Other*—Rare cases described as parotid swelling.

It should be noted that efficacy, indications and untoward effects have varied with the different phenothiazines. It has been reported that old age lowers the tolerance for phenothiazines; the most common neurologic side effects are parkinsonism and akathisia, and the risk of agranulocytosis and leukopenia increases. The following reactions have occurred with phenothiazines and should be considered whenever one of these drugs is used.

Autonomic Reactions—Miosis, obstipation, anorexia, paralytic ileus. *Cutaneous Reactions*—Erythema, exfoliative dermatitis, contact dermatitis. *Blood Dyscrasias*—Agranulocytosis, leukopenia, eosinophilia, thrombocytopenia, anemia, aplastic anemia, pancytopenia. *Allergic Reactions*—Fever, laryngeal edema, angioneurotic edema, asthma. *Hepatotoxicity*—Jaundice, biliary stasis. *Cardiovascular Effects*—Changes in terminal portion of electrocardiogram, including prolongation of Q-T interval, lowering and inversion of T-wave, and appearance of a wave tentatively identified as a bifid T or a U wave have been observed with phenothiazines, including Mellaril (thioridazine); these appear to be reversible and due to altered repolarization, not myocardial damage. While there is no evidence of a causal relationship between these changes and significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients showing characteristic electrocardiographic changes while taking the drug. While proposed, periodic electrocardiograms are not regarded as predictive. Hypotension, rarely resulting in cardiac arrest. *Extrapyramidal Symptoms*—Akathisia, agitation, motor restlessness, dystonic reactions, trismus, torticollis, opisthotonus, oculogyric crises, tremor, muscular rigidity, and akinesia. *Persistent Tardive Dyskinesia*—Persistent and sometimes irreversible tardive dyskinesia, characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and sometimes of extremities may occur on long-term therapy or after discontinuation of therapy, the risk being greater in elderly patients on high-dose therapy, especially females; if symptoms appear, discontinue all antipsychotic agents. Syndrome may be masked if treatment is reinstituted, dosage is increased, or antipsychotic agent is switched. Fine vermicular movements of tongue may be an early sign, and syndrome may not develop if medication is stopped at that time. *Endocrine Disturbances*—Menstrual irregularities, altered libido, gynecomastia, lactation, weight gain, edema, false positive pregnancy tests. *Urinary Disturbances*—Retention, incontinence. *Others*—Hyperpyrexia; behavioral effects suggestive of a paradoxical reaction, including excitement, bizarre dreams, aggravation of psychoses, and toxic confusional states; following long-term treatment, a peculiar skin-eye syndrome marked by progressive pigmentation of skin or conjunctiva and/or accompanied by discoloration of exposed sclera and cornea; stellate or irregular opacities of anterior lens and cornea; systemic lupus erythematosus-like syndrome.

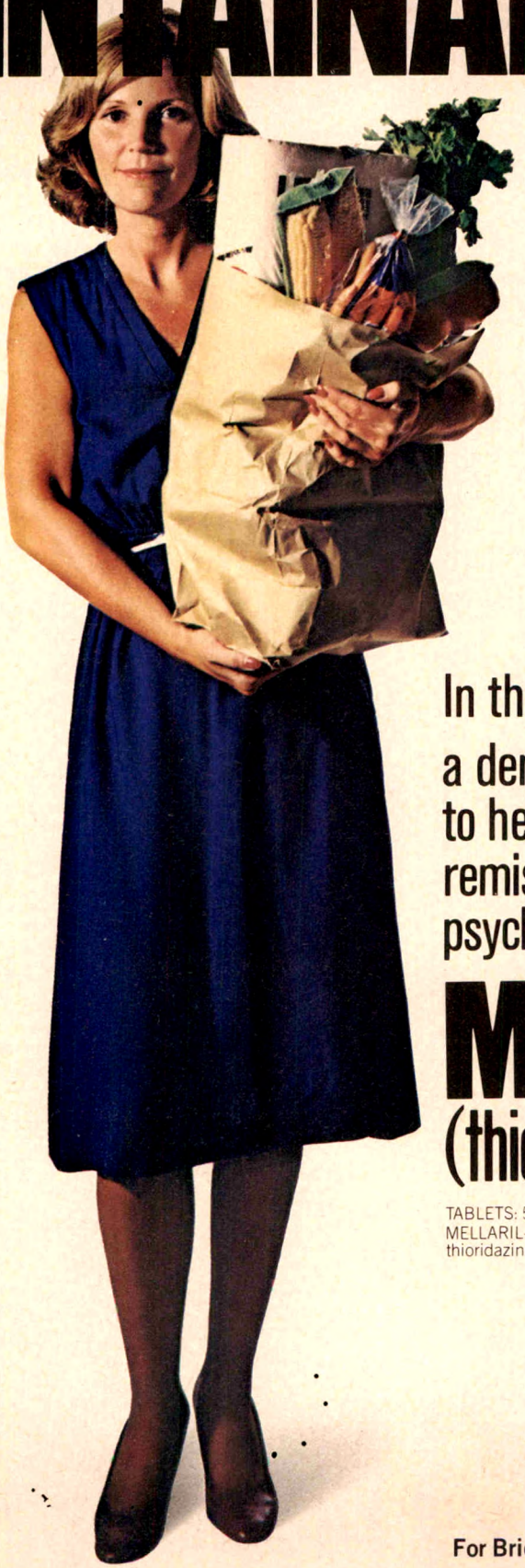
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THE AMERICAN JOURNAL OF PSYCHIATRY

The Efficacy of Cognitive and Behavior Therapies for Depression

BY MARIA KOVACS, PH.D.

This selective overview looks at the effectiveness of behavior therapy, cognitive-behavior therapy, and cognitive therapy for depressive disorders. The outcome of single-case studies, comparative treatment projects with volunteers, and clinical trials with outpatients are used to evaluate the current state of the art and to suggest future research directions.

A growing body of literature suggests that cognitive and behavior therapies are clinically useful and may be in fact superior to certain, more traditional treatments for a variety of disorders. The effectiveness of these interventions has been reported, for example, in the management of severe chronic tension headache (1, 2), test anxiety (3), chronic anger (4), stuttering (5), and "irrational thinking" in the elderly (6). Recent comprehensive reviews also testify to their increasing use in the treatment of nonpsychotic, unipolar depressions (7–10).

Evidence of the usefulness of cognitive and behavior therapies with ambulatory depressed patients is especially welcome, since in spite of the utility of antidepressant pharmacotherapy (11–14) there is a need for adjunct or alternate nonsomatic interventions. For example, recent reports suggest that from one-third to two-thirds of carefully selected nonpsychotic depressed outpatients show no clinically significant response to and/or drop out of pharmacotherapy (13, 15). It appears also that even after effective pharmacotherapy, eventual chronicity ensues for a notable minority of outpatients (16, 17). Finally, some depressed patients cannot or prefer not to be treated with pharmacological agents.

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THE THERAPIES AND THEIR EFFICACIES

"Cognitive-behavior therapy" is a term that has come to refer to a diverse group of treatment approaches (9). The interventions that fall under this label are generally directive, structured, goal-directed, and time-limited; involve the patient in an active collaboration with the therapist; use behavioral techniques derived from learning theory and/or the laboratory; use "homework assignments" and skills practice; and seek to increase the patient's adaptive problem-solving repertoire through teaching specific verbal and behavioral strategies.

Strictly speaking, however, since cognition encompasses perception, memory, judgment, language, problem-solving strategies, visual imagery, and so forth (18–20), every psychotherapeutic system has some cognitive basis (21). As far as the cognitive dimension is concerned, the differences among the various therapies reside in how cognitions are defined and in the importance attributed to them in the etiology and maintenance of psychopathological syndromes. For example, while many psychoanalytic models acknowledge the salience of conscious thoughts and attitudes (22–24), more fundamental and unconscious psychodynamic conflicts are presumed to be at the root of the patient's difficulties. In contrast, some behaviorists take the stance that while cognitions are covert behaviors, they nonetheless obey the same rules of learning and maintenance as other classes of behavior and ought to be just as amenable to change (25). In a slightly different vein, there are behaviorists who have postulated that cognitions are "moderating psycholog-

ical processes" that supplement simple stimulus-response behavior chains (26). According to the cognitive models (27–29), however, thinking and evaluation (as opposed to external contingencies) are *the* key to the development and understanding of psychopathology. Thus, the "purer" cognitive therapies reflect Kelly's theory of personal constructs (30) as well as a long tradition in academic cognitive psychology which holds that one's appraisal of a stimulus condition and the resultant emotion together determine the person's goal-directed behavior (31–34).

For the purposes of the present paper, the various cognitive-behavior treatment approaches are divided into three classes: the behavior therapies, the cognitive-behavior therapies, and the cognitive therapies. This three-fold division was based on considerations of historical development, underlying rationale, therapeutic goals, and explanations of the etiology of depression including the importance placed on cognitive variables.

Behavior Therapies

The "traditional" behavior therapies of depression are primarily documented by single-case reports or noncontrolled studies (see references 8 and 9 for reviews). The general emphasis is on the measurement of specific *overt* behaviors and on their modification through external contingencies (reinforcers). The theoretical rationale is that both adaptive and maladaptive behaviors are learned and are maintained through reinforcement schedules that can be analyzed, specified, and manipulated. Common treatment targets encompass increasing the frequency of socially desirable behaviors such as self-grooming, while decreasing the rate of undesirable or socially aversive ones such as crying and avoiding eye contact.

Given the influence of stimulus-response learning paradigms, it is not surprising that the most frequently employed intervention is contingency management, for example, selectively rewarding the patient for any expression of the desirable behavior. Homme's extension of the Premack principle (25) is also employed, through which a desirable but low-occurrence behavior is increased by pairing it with a naturally and fairly frequently occurring behavior that the patient finds reinforcing (e.g., smoking).

Several studies have described the success of contingency management in the experimental control of isolated depressive signs and symptoms and socially inappropriate behaviors. For example, Hersen and associates (35) reported that appropriate nondepressive behaviors (talking, smiling) and general work activity were increased by means of an inpatient token economy program. Hanaway and Barlow (36) described the token economy treatment of a "depressed," recently blinded, deaf inpatient who exhibited various depressive and socially peculiar behaviors. Reisinger (37) also used a token economy system to increase smiling and to decrease crying in a 20-year old "depressed" woman who had been hospitalized for 6 years, while Robinson and Lewinsohn (38) reported success in altering a depressed man's reduced rate of speech. For a more comprehensive overview, see Rehm and Kornblith (9).

In spite of their positive results, studies such as the above preclude any definitive conclusions about the efficacy of traditional behavior therapy for depressive disorders. In most of the reports, the patients' diagnostic status was unclear, and the pre- and post-intervention measurements generally pertained only to the behaviors that were to be modified. There was little information about the vicissitudes of other signs and symptoms or about the global severity of the disorder. The follow-up data were somewhat unsystematic, and the successful use of reversal and extinction strategies suggests the transient nature of the treatment gains. Thus, the overall usefulness of the more traditional behavior therapies for the treatment of a depressive *disorder* is yet to be satisfactorily tested. A similar conclusion can be drawn from the findings of Shipley and Fazio (39), who reported that a functional problem-solving approach was superior to "support" only and to waiting-list control in decreasing their subjects' self-rated depressive symptomatology. Since depression was conceptualized as a reaction to loss of reinforcement, subjects were taught more effective environmental manipulations. However, their status (students in a college introductory psychology class) and the lack of diagnostic or clinical data preclude an assessment of that study's applicability to clinic populations.

Of the interventions that are conceptually closest to traditional behavior therapy, Lewinsohn's social skills model (40, 41) of depression is probably the best known. While this model assumes that the etiological factor in depression is a low rate of response-contingent positive reinforcement, it looks at a range of intrapersonal variables that is not addressed in the contingency management paradigms. It emphasizes the importance of environmental contingencies, the social skills of the organism, and the social reinforcement of deviant behavior. The critical variables and mechanisms are as follows:

1. A person with a poor social skills repertoire (relatively low verbal activity, passivity, problems in listening and in interpreting appropriately others' behavior, and lack of adequate skills in dispensing positive reinforcement to others, resulting in failure to elicit the available environmental reinforcers).
2. Events that are contingent on the person's behavior may not be reinforcing.
3. Events that have been reinforcing become unavailable. The emergent symptoms of depression are then strengthened by social attention.

As a logical consequence of this paradigm, social-skills programs with depressed individuals generally

start with homework assignments that encompass the recording and monitoring of problem situations. The patient uses the self-rated Pleasant Events Schedule (41) to assess the baseline frequency of reinforcing events and to increase gradually his or her participation and involvement in such activities. Home observations by treatment personnel serve to 1) identify faulty interpersonal communication and the reinforcement patterns that contribute to the patient's depression, and 2) assist in the specification of treatment goals. The patient's initial self-monitoring and the home observations are used to arrive at a behavioral analysis of the problem and to contract for goals. Thus, social skills training seeks to counteract behavior deficits by increasing the frequency of emitted behaviors that are associated with positive social reinforcement. The goal is accomplished through any number of treatment strategies, such as helping patients to become aware of and to alter double-bind communications (42) and assertiveness training by means of role-playing (43).

The publications of Lewinsohn and associates on the efficacy of social skills treatment of depression have been primarily case reports. Lewinsohn and Shaw (44), for example, described how home observations of marital interactions were used to identify the social-behavior problems of a 24-year-old depressed woman referred for treatment after a "fainting spell." The results of the marital social skills training were reported as changes on self-rated symptom checklists and observational ratings. A 38-year-old woman with a 6-year history of "depression" was also reported as successfully treated through social skills training (42).

The above two representative reports suggest the utility of home observations of social behavior in the identification of patients' problems. They also document that improved interpersonal skills and increased pleasant activities are associated with a diminished severity of a depressive syndrome as assessed by self-rated scales. However, further conclusions are precluded because the reports do not include clinical assessments, the patients' diagnostic status is questionable (44), and the actual interventions are not described (42). In addition, one of the studies to be cited below (45) reported that Lewinsohn's social skills training program was less effective than cognitive therapy in reducing depressive symptoms and that its effects were not distinguishable from the nondirective-control treatment conditions.

On the other hand, McLean's version of social skills therapy with couples was reportedly successful in two separate clinical samples. In one study (46), "doctor's choice," which included diverse pharmacological and psychological interventions, was compared with social skills couples treatment for 20 nonpsychotic patients who were referred because of "incapacitating depressive behaviors." According to self-ratings of mood, changes in predefined target behaviors, and analyses of taped home conversations, the social skills-treated group did much better than the rest of the patients. A subsequent study (47) was much strengthened by the use of Feighner and associates' research diagnostic criteria (48) to select 178 patients. They were treated with 10 sessions of social skills couples therapy, dynamically oriented psychotherapy, tricyclic pharmacotherapy, and progressive muscle relaxation (treatment control). Behavior therapy was associated with the lowest treatment dropout rate (5%) and the most favorable scores on all outcome variables. A three-month follow-up revealed a decrease in intergroup differences, although social skills behavior therapy remained superior on most measures.

These studies suggest the efficacy of McLean's social skills program in the treatment of outpatients who suffer from a primary depressive disorder and also suggest the apparent superiority of this intervention over some other nonsomatic therapies and tricyclic pharmacotherapy. However, in McLean and Hakstian's most recent study (47) the patients' final clinical status is difficult to assess due to the authors' use of computer-derived outcome measures rather than better known psychiatric or symptom rating scales.

Cognitive-Behavior Therapies

A number of behaviorists have elaborated on the traditional behavior paradigms by defining cognitions (that is, internal or covert behaviors) as processes that moderate or mediate between stimulus and response and also as appropriate goals for modification. In the present paper, such models are designated as cognitive-behavior therapies. One that has been specifically applied to depression is self-control behavior therapy.

Self-control behavior therapy addresses the role of cognitions in the maintenance of behavioral responses when there is an absence of immediate external reinforcement (26, 49). This model assumes that depression results from an inability to tolerate the loss or delay of reinforcement, which is, in turn, a function of repeated failure or deficiency in specific self-control (cognitive) processes. That is, in depression there is 1) selective monitoring of (paying attention to) negative events and focusing on the immediate rather than on the long-term consequences of behavior, 2) the setting of overly stringent performance criteria and inaccurate attribution of responsibility (deficient self-evaluation), and 3) insufficient reward and excessive punishment, which constitute faulty self-reinforcement (26, 50).

Self-control behavior programs have been generally administered in a six-week group format and involve didactics and the sequential reinstatement of self-control skills (51). Appropriate self-monitoring is taught by having the patients increase the frequency of pleasant events in their lives and by refocusing their attention on the long-term consequences of their behavior. Rehearsals in reattribution and in setting realistic goals seek to reinstate reasonable self-evaluation. To learn

proper self-reinforcement, patients practice the self-administration of positive rewards for increasing their own goal-related activities and experiences. Homework assignments facilitate discussions in the therapy sessions and enhance the patients' learning.

Two studies have reported the effectiveness of a self-control training program in the management of depression. The subjects were women volunteers who were moderately depressed as determined by cutoff scores on the Minnesota Multiphasic Personality Inventory and by interview criteria. Individuals with past psychiatric hospitalizations and current suicidal behavior were excluded. The clients' progress was assessed by self-report symptom scales, among others, an overt motor measure of depression, and by the number of pleasant events in which they engaged. After six weeks of group therapy, clients who received self-control behavior training scored significantly better on most of the outcome measures than those who were treated with nondirective therapy (discussion of past and present problems and the identification and clarification of feelings) and those who were on a waiting list. However, by six-week follow-up the previously significant differences between the groups had decreased to the point where there was only a tendency toward statistical significance. In a replication and extension of this study, Rehm and associates (52) compared self-control and social skills behavior therapy programs in the treatment of 24 women. After six weekly group sessions, self-control therapy was associated with the greatest improvements on self-rated and behavioral-observational measures of depression. However, the self-control group improved more on self-control measures, while the social skills-treated clients improved more on social skills measures. The six-week follow-up data were equivocal.

These two studies are notable for the systematic step-by-step application of a treatment program and the use of observational outcome measures. The findings indicate that self-control behavior therapy is efficacious in the treatment of moderately depressed women volunteers with fairly good premorbid histories and is more successful than nondirective or social skills therapy or no treatment at all. However, the specificity of the findings is somewhat obscured by the fact that the self-control and the social skills program used overlapping strategies (i.e., increasing pleasant events). Although, as the authors noted, the clients were psychometrically similar to clinical populations, the studies did not provide diagnostic information or clinical ratings of the subjects' symptom status. Thus, further studies are needed to determine whether similar findings can be obtained with clinical and diagnostically distinct depressed patients.

Cognitive Therapy

Both rational-emotive (29) and cognitive therapy (27, 28) postulate that man is primarily a thinking, judging, and inquiring being whose actions and affective experiences are determined by his or her *interpretation* of internal and external events. Thus; the cognitive view of behavior (27–30, 34) suggests that numerous psychopathological symptoms and signs can be understood and ameliorated by an analysis and modification of the patient's relevant modes of thinking. Since only Beck's cognitive therapy (28) has been empirically tested for the treatment of depression, the following discussion excludes rational-emotive therapy.

In taking a cross-section of depressive symptomatology, Beck's cognitive therapy focuses on and seeks to correct the depressed patient's characteristically negative thoughts and attitudes and the maladaptive ways in which the patient processes information. For example, the depressed patient's evaluations are distorted by a tendency to magnify the importance of personally relevant unfavorable events and to minimize the meaning of positive events or attributes. The patient selectively remembers unpleasant occurrences and persistently draws conclusions in the absence of, or even contrary to, evidence (53). It is assumed that most of the cardinal symptoms of depression, e.g., pervasive dysphoria, passivity, social withdrawal, and suicidal behaviors, are natural concomitants of the patient's pervasively negative and distorted self- and world view and are supported by a system of relevant beliefs or basic assumptions. It is furthermore assumed that the patient's basic assumptions are embedded in depressogenic schemata. Schemata, or cognitive templates, are characteristic ways of organizing, interpreting, or construing particular sets of experiences (27, 28, 54, 55). Thus, depressogenic schemata are ostensibly reactivated by individual-specific internal or external cues that the person perceives as signaling the loss of something vital for "survival" and/or self-esteem.

Cognitive therapy has been generally administered in a short-term (e.g., 12-week) individual-session format, as outlined in a treatment manual (56). Patients are given didactic explanations of the relationship among cognition, affect, and behavior and how these relate to the phenomenology of depression. Through examples from their own lives, the patients are taught how to observe and record their maladaptive cognitions. By learning semantic techniques such as "questioning the evidence," the patients come to examine whether their thoughts and conclusions represent a reasonable assessment of the situation at hand and to modify their cognitions as may be appropriate. Homework assignments in the course of daily living are used to help the patient test the validity of his or her notions and master relevant situations. Behavioral techniques (e.g., daily activity schedules, counting negative thoughts) are also employed to provide the patient with "mastery" experiences. While it is postulated that the patient's negative evaluations constitute the

first link in the chain of depressive symptoms, any depressive sign or symptom may be targeted for initial modification. The ultimate goal is, nevertheless, to elucidate and to modify as necessary the patient's depressogenic schemata.

In an uncontrolled trial, Rush and associates (57) reported the use of cognitive therapy with three moderately to severely depressed men who met research diagnostic criteria (48) for primary depressive disorder. The patients had had past depressive episodes and multiple somatic treatments, with either marginal or good but unsustained response. Cognitive therapy reportedly produced marked decreases in the patients' symptomatology, and the improvements were maintained at one-year follow-up.

In a subsequent study with 32 mildly to moderately depressed 18- to 25-year-old university health service clients (45), a 4-week group treatment format was used to compare cognitive therapy and Lewinsohn's social skills program with nondirective therapy and a waiting list control group. An interview was used to exclude individuals with major psychiatric illnesses and suicidal risk and to assure that depression was the primary presenting problem. Independent clinical ratings of the severity of depression (58) were also obtained. Cognitive therapy was found to be the most effective in ameliorating depressive symptomatology, according to both self-report and clinical ratings. The social skills treatment and nondirective therapy were similarly effective, according to self-ratings, while the clinical ratings revealed no differences among the social skills, nondirective, and waiting list groups. However, a one-month follow-up of the cognitive and the behavior modification groups revealed no significant symptomatic differences on any of the ratings.

In a recent study (16, 59), 44 nonbipolar, nonpsychotic outpatients suffering from moderate to severe depression were randomly assigned to individual treatment with cognitive therapy or imipramine hydrochloride. The patients met Feighner and associates' criteria (48) for primary depressive disorder, as well as predetermined cut-off scores on self- and clinician-rated symptom scales. The modal patient had been symptomatic for less than one year, had suicidal ideation, and had had previous depressive episodes. At the end of the 12-week clinical trial, cognitive therapy and imipramine administration were both found to be effective interventions for the patients in the study. Symptomatic improvement in both treatment conditions was documented on self-report scales and clinician- and therapist-completed measures. However, cognitive therapy resulted in significantly greater symptomatic improvement than did pharmacotherapy. Also, the dropout rate was significantly higher in the medication group (32%) than in the group that received cognitive therapy (5%).

One year later, the patients who had completed cognitive therapy in the study's clinical trial phase were still significantly less symptomatic on a self-rated depression scale than patients who were initially treated with and had completed their imipramine trial. However, no statistically significant differences were found on the available clinician-completed scales at one-year follow-up. Although those who had completed the pharmacotherapy had a twofold greater relapse rate than the cognitive therapy completers, the difference was not statistically significant.

The above studies are notable for their use of a treatment manual, the monitoring of therapist adherence to the manual, and the careful psychiatric screening of the patients. The findings indicate that cognitive therapy can be effectively applied to treat nonpsychotic, nonbipolar depressed outpatients and can exceed pharmacotherapy in both symptom reduction and treatment completion rate. However, the conclusions must remain tentative because in one case (16) the clinical evaluators were not fully blind to the patients' treatment assignment and since questions have been raised about the length and dose of the pharmacological regimen. Moreover, in two of the studies (16, 57) it is unclear to what degree the cognitive strategies, as opposed to the behavioral techniques, contributed to the patients' symptomatic improvement. Thus, further research is needed to replicate the above summarized work and to tease out the relative contributions of the various techniques in the amelioration of depressive signs and symptoms.

The study by Taylor and Marshall (60) provides some support for the efficacy of a combined cognitive and behavioral approach. Using 28 mildly to moderately depressed college students, the authors found that a six-session combined approach proved more effective than the cognitive or the behavioral treatment alone. However, as assessed by two self-rated depression measures, all three interventions led to significant decreases in symptomatology, compared with a waiting list condition. The results were essentially maintained at six-week follow-up.

CONCLUSIONS

Currently, only McLean's social skills behavior program and Beck's cognitive therapy have been tested in controlled clinical trials with psychiatrically screened depressed outpatients. In spite of differences in underlying theory and treatment techniques, these clinical trials suggest that 1) structured, time-limited, and directive interventions that seek to teach cognitive and behavior skills are beneficial in the treatment of nonpsychotic, nonbipolar outpatients suffering from moderate to severe depressive disorders, and 2) for such ambulatory patients, these cognitive and social skills interventions may even exceed pharmacotherapy in both symptom reduction and treatment completion rate.

However, the long-term efficacy of the treatments is more elusive. The available follow-up data suggest that their impact is relative, that is, these interventions seem to get depressed patients well in a shorter period than do several other therapies. While the maintenance of *differential* treatment effects is hard to evaluate because of events that impinge on the patient during follow-up, based on the theoretical rationales of the therapies in question, lasting well-being would be anticipated subsequent to successful intervention.

Thus, the studies on the efficacies of Beck's cognitive therapy and McLean's social skills behavior program need replication with an emphasis on remedying shortcomings of the existing research. Moreover, additional work is called for to determine what type of depressed patient is the best candidate for either of these interventions alone, or in combination with pharmacotherapy. Ways and means of improving the patients' long-term clinical status deserve special attention.

Outcome studies of the traditional behavior therapies that rely mostly on contingency management indicate that these interventions encompass techniques that are successful in the short-term control of isolated depressive signs and symptoms and functionally inappropriate behaviors. However, the stability of the behavioral gains across situations and over time still need to be demonstrated. It may be possible, for example, that changing environmental contingencies is not sufficiently potent by itself to bring about global clinical change. Thus, the efficacy of traditional behavior modification techniques in the amelioration of depressive disorders is yet to be subjected to a randomized clinical trial. Similarly, the self-control cognitive-behavior programs appear to have much promise. There is now a need to assess their effectiveness in the treatment of psychiatric patients who have had a comprehensive clinical evaluation.

Future studies could explore, for example, whether one of the cognitively oriented therapies subsequent to contingency management may stabilize therapeutic gains. Since it would lead to more intelligent treatment choices, another interesting topic of investigation would be the relationship among subtypes of or specific "deficits" in depression and the relative efficacies of the various behavior and cognitive interventions.

It is important to remember that the cognitive and behavioral therapies derive from a psychological/behavioral approach to the development and phenomenology of disturbed behavior. In this case, they represent only two global conceptualizations of how depressive illness comes about and what may be done to help individuals who suffer from it. They do not preclude or replace biochemical, physiological, or other etiologic explanations. Rather, the various perspectives highlight different although interconnected aspects of these clinical conditions. For example, while the cognitive model of depression can provide a sensible and meaningful explanation of the depressed patient's wishes to commit suicide, the catecholamine model's explanatory strength is more in the domain of noncognitive depressive signs and symptoms. Until the mode of action of any intervention is more fully understood, however, the utility of a treatment must rest on its empirically demonstrable results rather than on its conceptual attractiveness.

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The Ethics of Psychotherapy

BY TOKSOZ B. KARASU, M.D.

The author addresses the ethics of psychotherapy in terms of the interface between science and ethics, the goals of treatment, the therapeutic relationship, and special issues of confidentiality and therapist-patient sex. He considers the problems of multiple therapeutic modalities, dual allegiance of the therapist, the therapeutic use (and abuse) of power, and issues of dependency and suggests ways to maximize the clinician's exercise of ethical choices. Ethical dilemmas in psychotherapy are not entirely soluble; ultimately, the therapist, guided by his or her profession as a group, will be able to find answers to the complex problems that inevitably arise.

In 1978 Spiegel (1) suggested that we have evolved from an Age of Anxiety to an Age of Ethical Crises. Progressive loss of faith in traditional institutions and the erosion of authority are now being met with the increasing challenge of existing standards and widespread concern for safeguarding human values and rights. Today's growing climate of antiestablishment, antiprofessional, and antirational sentiment has direct implications for the roles and responsibilities of the psychotherapist. Growing skepticism about the sanctity of science, medicine, and psychiatry means that these fields are no longer above rebuke or exempt from active moral review by their recipients, professional peers, and others outside of their practice, such as third-party payers. The psychotherapist, once left relatively undisturbed in the private confines of his or her office, has now been besieged from within (2) and without (3). The siege from within reflects psychiatry's own members and critics, who extol widely different models and criteria of mental illness and its treatment, which are confusing and divisive to the field and its future. The siege from without reflects the public voice and confusion regarding the functions, procedures, and powers of the psychotherapist. There is an increasing expectation and demand for accountability, i.e., that the patient be granted, indeed is owed, health as a right and greater participation in determining and assessing the goals and activities as well as the outcome of treatment. Simultaneously, social, political, and personal pressures oblige the psychotherapist (under the prospect of national health insurance) to assess and review his or her practices. Michels (4) attributed the failure of the profession itself to stave off this challenge and criticism to four factors: 1) a fundamental disappointment with the limitations of science and reason in answering the problems of mankind, 2) an antielitism that aims to mitigate the power of professionals as symbolic representations of the inequitable distribution of resources in society, 3) the compounding of the antiprofessional stance by members of the profession who have failed and then have turned against it in response to exclusion from membership, and, most relevantly for the morality issue in psychotherapy, 4) the fear of overgeneralization of the authority of the professional from scientific to ethical arenas.

THE INTERFACE BETWEEN SCIENCE AND ETHICS IN PSYCHOTHERAPY

The issue of the relationship of science to ethics in psychotherapy may be considered the conceptual heart of the matter. *Webster's New World Dictionary* defines "ethics" as "the system or code of morality of a particular person, religion, group or profession," "morals" as "relating to, dealing with, or capable of making the distinction between right and wrong in conduct," and "science," which psychotherapy presumes to be, as "a branch of knowledge or study concerned with establishing or systemizing facts." Theoretically, science and ethics have been viewed as two distinct and separate entities, almost antithetical: science as descriptive, ethics as prescriptive; science as resting on validation, ethics as relying on judgment; and science as concerned solely with "what is," ethics as addressing "what ought to be" (5). The lines become less sharply drawn, however, when the complexities of social reality are brought into the picture, when the psychotherapist is obliged to act as a "double agent" (6) to accommodate conflicts of interest posed between patient and therapist and by third parties to whom the therapist holds allegiance, such as family members and school, hospital, and military authorities (7). The lines become even less clear as the

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therapist confronts the ambiguity between the science and the art of psychotherapy, dual attitudes of the psychotherapist's identity that differ in degree and quality (8). In fact, there is still a question of whether psychotherapy is a science at all, in that it deals with hermeneutics rather than explanation, is humanistic rather than mechanistic, seeks private rather than public knowledge—in all, in that it is not a science but a body of knowledge with a special status, which frees it from obligations that other sciences share (9).

In addition, although the distinction between the principles of science and those of ethics may more readily hold for the researcher inside his or her laboratory, it is simply less applicable to the clinician in daily practice (10). Lifton (6) highlighted this point in describing his work with Viet Nam veterans, which required him to combine sufficient detachment to make psychological evaluations with involvement that expressed his own personal commitments and moral passions. (He had taken an active antiwar advocacy position during the Viet Nam conflict and expressed profound interest and concern for Hiroshima survivors.) Lifton aptly concluded, "I believe that we [therapists] always function within this *dialectic between ethical involvement and intellectual rigour.*" He went even farther to recommend that "bringing our advocacy 'out front' and articulating it makes us more, rather than less, scientific. Indeed, our scientific accuracy is likely to suffer when we hide our ethical beliefs behind the claims of neutrality and that we are nothing but 'neutral screens.' "

With the above in mind, we may say that it is inevitable for the boundaries between science and ethics to become blurred. To the extent that the therapist implicitly, if not explicitly, cares about "what ought to be" as well as "what is," ethical issues will inhere in virtually all of his or her work. In the broadest terms, then, there has been a growing recognition that subjective commitment (unconscious as well as conscious) places inevitable constraints on the presumed purity and verity of objective treatment. More specifically, the idyllic notion of psychotherapy (and the psychotherapist) as value-free is now widely accepted as a fallacy (11). This is supported by current research demonstrating many contradictions in expressed belief versus reported practices in psychotherapy (12). Buckley and associates (12) hypothesized the presence of a "two-tier" system: one, the ideal or correct (i.e., value-free, nonsuggestive); the other, the practical or applied view (i.e., direct suggestions, encouragement of specific goals), which loosens the adherence to a value-free frame of reference. In brief, when actually applied, psychotherapy represents neither pure science nor pure ethics but a branch of the healing profession that resides somewhere in between. Therefore, Erikson (13) concluded that psychotherapy can find its ethical place only by locating a legitimate and unique area between the two ideological extremes of being an objectively applied true science and representing an ideology of healthy conduct.

Concern with the interface of science and ethics or healthy conduct and with the place of values in treatment is certainly not new to the field of psychotherapy. A major controversy has long pivoted on the degree to which psychoanalysis inherently propounds any particular value system. Some believe it does not do so (14). Others imply that it is political and repressive by definition (15, 16), that it favors particular cultural values, especially biases toward certain social classes over others (17), and that its basic tenets are inherently biased against women (18). Still others, although they accept that psychotherapy cannot be value-free and even believe that in its overall goals it should not be (19), see the fundamental issue as whether the imposition of such values are "deliberate and avowed" or "unrecognized and unavowed" (13).

On a more concrete level, Freud can be credited with the discovery and longtime recognition of the unique power and intensity of the transferential relationship (both to cure and to be resisted) and the inevitable influence of the therapist and his or her values in treatment. This has been classically dealt with in great depth in analytic attempts to maintain the purity of the therapeutic relationship (transference) through the technical neutrality of the therapist, and, when inadvertently violated, the full exploration and understanding of the therapist's countertransference. Freud certainly held an ethical ideal about the conduct of psychotherapy when he wrote, "One must not forget that the relationship between analyst and patient is based on a love of truth, that is, on the acknowledgment of reality, and that it precludes any kind of sham or deceit" (20, p. 248).

THE GOALS OF PSYCHOTHERAPY

The Principles of Medical Ethics of the American Medical Association (21), first adopted in 1847, set down the standards of practice for physicians. Although psychiatrists are assumed to have the same goals as all physicians, APA added to the *Principles Annotations Especially Applicable to Psychiatry* (22). The rationale for these annotations was that "there are special ethical problems in psychiatric practice that differ in coloring and degree from ethical problems in other branches of medical practice (22). These annotations made no alterations whatever in the original AMA standards.

Section 3 of the AMA *Principles* was the only one not annotated for psychiatry. It states that a physician (and therefore a psychiatrist) "should practice a method of healing founded on a scientific basis; and he should not voluntarily associate professionally with anyone who violates this principle." In the annotation to section 6 of the *Principles* the psychiatrist is advised

that "he/she should neither lend the endorsement of the psychiatric specialty nor refer patients to persons, groups, or treatment programs with which he/she is not familiar, especially if their work is based only on dogma and authority and not on scientific validation and replication."

Fisher and Greenberg (23) suggested that the question of the scientific credibility of Freud's theory and therapy, despite extensive exploration, has not been decisively settled in the minds of psychiatrists themselves, although their hearts may tell them otherwise. Moreover, the proliferation of well over 100 supposed schools of psychotherapy,[1] each with its own, albeit overlapping, theory of mental illness and health, therapeutic agents, overall goals, and specific practices (24), reflects the massive nature of investigating therapeutic efficacy and the complexity of establishing scientific guidelines. Given the stunning diversity of therapeutic forms now being offered to potential patients, how does one ethically equate the goals encompassed in a screaming cure (Janov's primal therapy), a reasoning cure (Ellis' rational therapy), a realism cure (Glasser's reality therapy), a decision cure (Greenwald's direct decision therapy), an orgasm cure (Reich's orgone therapy), a meaning cure (Frankl's logotherapy), and even a profound-rest cure (transcendental meditation)? For example, Leo (25) quoted Slavson's assessment of "feeling therapy, nude therapy, marathon therapy, and other new remedies of the ailing psyche." Slavson concluded that "these activities [are] untested, theoretically weak, and potentially very dangerous." More specifically, he asserted that "latent or borderline psychotics with tenuous ego controls and defenses may, under the stress of such groups and the complete giving up of defenses, jump the barrier between sanity and insanity." However, the data on the efficacy of these practices are not yet in. Does the psychotherapist have an ethical responsibility to force closure on these events, especially if his or her judgments are premature?

Such tremendous confusion in the state of the art has led to a virtual identity crisis for psychiatry and the psychotherapist (26–28), highlighted negatively (29) in the recent growth of the antipsychiatry movement, which makes it possible for a psychiatrist to be a psychiatrist by training, accreditation, affiliation, and status but at the same time an antipsychiatrist in ideology and action. (All this under one ethical psychiatric roof.) Indeed, the goals and responsibilities of the psychotherapist are now so broadly and vaguely defined that Raskin (30) said ours is a profession without a "role-specific function" and Vispo (31) said our practices run the gamut from "science to social revolution." Redlich and Mollica (7) described the quandary of whether the practical purpose of psychiatry and the psychotherapist as its agent is "to diagnose, treat, and prevent a relatively defined number of psychoses? to perform this task on a number of neurotics? to make unhappy and incompetent persons happy and competent? or to tackle poverty and civil and international strife?" Although these are not beyond the legitimate concern of psychotherapists in their quest to improve the psychological welfare of their patients, there is a question as to whether these goals are beyond their legitimate competence. Where does one draw the line?

Although broadly recognized goals can include Freud's love and work and variants of growth and maturation, self-realization, self-sufficiency, and security and freedom from anxiety—all of which may be noble aspirations—ethical issues inhere in the practices that are conducted in their name. Across a spectrum of possibilities have been posed questions of freedom to change or not to change versus coercion, helping and healing versus shaping and imposing the therapist's influence, and issues of "cure" of illness versus positive growth. Szasz (32), probably the most prolific and vocal propounder of an antipsychiatry position, views conventional therapy by definition as "social action, not healing" and as "a series of religious, rhetorical, and repressive acts." The basis of the controversy is the medical model's designation of "patient," which presupposes restrictive conceptualizations of normality and health.

One of the most fundamental ethical dilemmas directly related to the goal of therapy is whether to encourage the patient to rebel against a repressive environment or to adjust to his or her condition (33). This issue is illustrated by psychiatry's standard definition and traditional treatment of homosexuals. "Homosexuality" as a "sexual deviation" (*DSM-II*) and its implications have been ardently challenged by advocates for gay rights, and sufficient pressure was brought to bear on APA that the official designation was changed to "sexual orientation disturbance." A review of the psychotherapy and behavior therapy literature indicates that therapists generally regard homosexuality as undesirable, if not pathological (34). On the less theoretical plane of actual treatment goals, the therapist may be obliged to take a position, implicitly if not explicitly, as to whether a heterosexual orientation is a valid ultimate goal for the patient or the patient should maximize the quality of his or her life adjustment as a homosexual. The latter stance, often unpopular, was subject to debate when a behavior therapist treating a man who was sexually attracted to boys provided the patient with methods to transfer that attraction to men, not women (11, 35, 36). Obviously, definitions of goals not only vary between therapists but change with the times as definitions of normality evolve.

Another ethical dilemma in establishing therapeutic goals pertains to the dual allegiance of the therapist to

[1] M. Parloff: Twenty-five years of research in psychiatry. Department of Psychiatry, Albert Einstein College of Medicine, New York, N.Y., Oct. 17, 1975.

the patient and to society's representatives. This was placed in bold relief in the role of psychiatrists who were treating soldiers in Viet Nam (6). Lifton stated that in such instances the therapist is forced to tread the thin line between advocacy and corruption. With adjustment to the military as the goal of therapy, the psychiatrist most likely "helped" the troubled soldier to remain on military duty. This meant that the soldier had to continue the commission of war crimes, which is what he was expected to do in Viet Nam. Thus psychiatry served unsuspectingly "to erode whatever capacity [the soldiers] retained for moral revulsion and animating guilt" (6), and the goals of psychotherapists became inseparable from those of military authority.

Aside from conflictual goals in psychotherapy posed by the individual versus the larger society, Hadley and Strupp (37) found that a major area of negative effects of psychotherapy according to a survey of practitioners and researchers in the field was "undertaking unrealistic tasks or goals." These authors listed false assumptions concerning the scope and potency of therapy's purported goals first among tendencies in the conduct of treatment that have profound ethical implications. Misleading impressions may be imbued by therapists when their need to instill hope in the patient and the omniscience endowed them (by themselves and/or the patient) become intertwined. Although some degree of positive expectation or hope is regarded as requisite for producing therapeutic effects in all psychotherapies (38), the patient may get the erroneous impression that therapy and the therapist can solve everything. This can perpetuate unrealistic expectations and goals that are ultimately deleterious to the patient. Such tendencies are often compounded by the failure to discuss, describe, or even acknowledge the reality of goals during treatment or when the stated goals are too broad or obscure.

Special technical problems with ethical implications also arise when goals explicitly or implicitly exceed the patient's capabilities, which fosters false hopes of speedy progress that cannot be realized if the patient actually requires longer treatment. Another problem occurs when the patient has accomplished certain goals but the therapist alters the goals to prolong treatment because he or she is unwilling to terminate it. Greenson (39) aptly warned that any form or aspect of therapy that makes the patient an addict to therapy and to the therapist is undesirable.

Therapists are obliged to ask themselves, How ethical is it to have limited goals when the needs of the patient may change or evolve as the therapy progresses, or when the patient is in need of long-term treatment but either financial or administrative expediencies will not permit it? Can superficial interpretations, made in the interests of time, shortchange patients? Where does one draw the line in serving long-term goals? For example, psychotherapists tend not to prescribe medication for moderate insomnia, anxiety, or depression, the presumed justification being the potentially motivating aspect of discomfort and suffering for psychological work. If this is the case, has the patient been informed of the means and ends of the therapist?

In summary, the issue of the goals of psychotherapy is complex and subtle, including professional versus personal, long- versus short-term, nonspecific versus specific, and overt versus covert goals. It is the latter that is ethically the most problematic and the one most under the therapist's and the patient's control.

THE THERAPEUTIC RELATIONSHIP

The therapeutic relationship or special power relationship that exists between doctor and patient constitutes psychotherapy's strength as well as its weakness. The duality inheres in the concept of authority, which may be defined in many ways (40). In its purist sense, it refers to an individual who is a specialist in his or her field and is entitled to credit or acceptance on this basis; in another sense, it refers to power that requires submission. Different types of therapeutic relationships have been formed with different therapies (24) or at different times in the process of the same therapy (41). A common ethical problem for psychotherapy pivots on the degree to which the therapeutic relationship is authoritarian versus egalitarian, or, more specifically, to what extent the pervasive power of the therapeutic transference relationship, which offers the therapist a unique vehicle for exercising enormous influence over another human being, is balanced by a true "therapeutic alliance" (39) or "therapeutic partnership" (42). Redlich and Mollica (7) pointed out that in general psychiatry "the fiduciary system, in which a patient puts his trust in the physician's ability and willingness to make crucial decisions, is being replaced by a contractual system." This trend applies as well to the current psychotherapies.

An egalitarian therapeutic relationship is gaining in prominence and is considered more humanitarian and facilitative of free exchange between patient and therapist than the traditional medical model (i.e., doctor-to-patient) or the behavioral model (i.e., teacher-to-student) (24). Might some aspects of this model, however, have negative implications for the therapeutic endeavor in general? Parsons (43), in his analysis of social structure and the dynamic process, identified certain requirements of the doctor-patient relationship necessary for successful treatment. One of the most essential was the "social distance" between practitioner and client. In their study of human organization Burling and associates (44) concluded, "We are coming to understand that faith in the doctor is a necessary element in cure, [and] that he will not be able to exercise therapeutic leverage if we, as patients, regard him in too prosaic a light" (p. 71). These authors suggested

that the therapist's power to claim the patient's confidence and the therapist's effectiveness will be impaired by growing familiarity. Where, then, does one draw the line between the good use of power in the traditional model and its abuse? Where do the new boundaries of partnership end and those of "real" familiarity and friendship begin? More important for the times, will there be new ethical dilemmas in the egalitarian relationship between therapeutic partners?

Goldberg's exposition of the equitable "therapeutic partnership" (42) suggests that it is not only the nature of the therapeutic alliance (i.e., its power distribution) that is critical but the degree to which it is made explicit. Often within its nonexplicit nature lies the ethical rub of psychotherapy. Goldberg recommended that a therapeutic contract establish a mutually agreed on and explicitly articulated working plan (comparable to the medical model's treatment plan), the essence of which is how each agent will ultimately use or restrict his or her use of power. This should consist not only of agreed on goals but established means, evaluation of therapeutic work during its course, and methods of addressing dissatisfactions in the working alliance. Articulating in practice what one intends in theory, however, is an admirable but not easily accomplished task. In addition to goal ambiguity, noted earlier, the distinction between theory and practice has also been a criticism of newer modalities like encounter group therapy (45, 46). Despite appearances of therapeutic virtues like openness, autonomy, and mutuality, encounter group therapy often gives little attention to the specificity of individual participants' psychological needs (45), and treatment is often started without defining what the patient wants or expects to be the result of the encounter (46).

How the therapeutic relationship is manifested also relates to one of the most prominent negative effects of the traditional therapeutic relationship, according to a 1976 survey (37): its insufficient regard for the patient's intentionality or will. This can be exacerbated in many ways. For example, in the analyst's fervent search for unconscious determinants the therapy may soon become an end in itself and the therapist may assume priority over all other people in the patient's life. This can occur in an even more extreme form in the newer, "spiritual" therapies, which encourage pious belief in the therapist that overrides the realities of life itself (witness the tragedy of Guyana). In either event, the therapist's power is greatly exaggerated for reasons that may have more to do with the therapist's needs than those of the patient.

Dependency, nonetheless, is one of the most common characteristics of all patients and allows for the early establishment of the helper-helpee relationship. Other relationships (e.g., transference and a working alliance) that help to play out that dependence and ultimately aid the therapist in encouraging independence in the patient develop in the process of treatment. Some patients, however, need lifelong supervision of their lives even after much of the psychotherapy work is done; often they may take action themselves and terminate treatment. It is also possible that a therapist, because of lack of experience or for less benign reasons (e.g., financial or pathological needs) perpetuates the dependency and unresolved transference of the patient. Although most often viewed within the arena of a technical problem of treatment, the question remains, When does this technical problem in therapy become an ethical issue as well?

CONFIDENTIALITY

The issues of confidentiality and privileged communication in psychotherapy have had national implications in the last decade, exemplified by the Watergate scandal and the attempt to steal Daniel Ellsberg's records from his psychiatrist's office, but these issues are generally more subtle in everyday psychotherapeutic practice. Although psychotherapists need be concerned here with the ethical rather than the legal aspects of confidentiality, the subject is often complicated by the specter of legal sanction.

The sanctity of confidentiality for the psychotherapeutic endeavor is crucial because of the inherently personal nature of its communications, which plumb the depths of the patient's innermost thoughts, fantasies, and feelings. A number of states, in fact, have statutes that grant the relationship between psychiatrist and patient the same absolute protections from public policy as are accorded to the relationship between husband and wife and attorney and client, to grand jurors, and to secrets of state (47). Indeed, the most elaborate clause of APA's *Annotations* to *The Principles of Medical Ethics* (section 9) (22) relates to this subject. It calls for extreme care in matters of both written and verbal communications, especially in instances of consultation with other professionals, with clinical notes and records, and in case presentations and other use or dissemination of confidential teaching materials. In addition, Redlich and Mollica (7) warned against the more insidious dangers of "gossip" between therapists that might inadvertently prove detrimental to the patient and the therapeutic relationship.

Having to meet current demands for detailed record-keeping in anticipation of the requirements and wishes of third-party payers and peer review organizations means that the confidential boundaries of the traditional dyadic relationship between therapist and patient have greatly enlarged and eroded. Both the domain for potential communication (i.e., who is privy to confidential information) and the risk of violation of confidentiality have geometrically increased. Plaut (48) has said that an escalating conflict exists between the right to secrecy and the right to information because of 1) increasing government involvement in areas that

were previously considered private affairs, 2) the electronic revolution in data collection, storage, and retrieval, and 3) the prevailing atmosphere of high suspiciousness between individuals and government authorities, for whom knowledge has always meant power.

In view of these new outside pressures, the major questions of confidentiality in psychotherapy are, Whose agent is the psychotherapist (the patient's? the family's? society's? the law's?)? What are the goals of divulging confidential material (better treatment? evaluation? consensual validation? support?)? What are the risks? (Will the therapeutic relationship be jeopardized? Will the patient terminate treatment?) Conversely, will rigid adherence to a rule of confidentiality between therapist and patient blind the therapist to worse fates for the patient—the risk of danger to self or others?

Sometimes the confusion of the therapist's allegiance inheres in the nature of the psychotherapy he or she is conducting—individual-, family-, or society-oriented. Although individual-oriented psychotherapy may limit itself to the more orthodox dyadic goals within the private framework of the patient's inner thoughts and feelings, other goals may be more society-oriented and may use the information communicated between therapist and patient to influence the patient's social milieu. A serious question that often arises is the therapist's role and responsibility to the family of the patient. In general, the less healthy the patient, the more important this issue can become. With relatively stable and independent individuals, there is usually no need to contact family members, nor would the therapist encourage any communication from them. Should the latter occur, it is usually in the patient's best interests that he or she be promptly informed of the family contact and that the contact become material for the sessions themselves, grist for the mill of the psychotherapeutic treatment. However, more disturbed patients may not only need the support of the family; they may involve family members with the therapist as an extension of their disturbance. In such circumstances, each communication with the family not only complicates the treatment but also raises serious ethical questions about breaches of confidentiality and whose interests the therapist is serving. In family therapy, whose purported goal is "to treat the family as the patient" (49), the situation may be reversed in that an individual's confidences may be subverted in the therapeutic service of the marital unit or family.

Thus whether a therapist adheres to an individual or a family orientation may determine not only the goals of treatment but also the nature of its treatment of the individual within each mode. How does the therapist determine whether it is in the individual patient's best interests to be seen alone or within the context of the family unit? How ethical is it to impose a systems approach in treatment (e.g., husband and wife, parents and children) because one views the problem as stemming from the family or marriage? On the other side of the coin are instances in which dynamic psychotherapists so strongly believe in utmost confidentiality and individual privacy of the dyadic relationship that they fail to divulge confidences or share information with family members that may prove vital to the welfare of the patient.

Indeed, the only exception to the overall rule of confidentiality between therapist and patient is that of "dangerousness" to others. This concept was legally stipulated by the famous *Tarasoff* decision (50), which enunciated the maxim that "protective privilege ends where public peril begins." According to this decision, therapists must warn authorities specified by law as well as potential victims of possible dangerous actions of their patients. (The legal case involved a young man's confidential announcement to his therapist that he intended to kill his girlfriend. After the therapist had consulted with two psychiatrists and notified the police that the man was dangerous, the young man was detained. However, he was released when he denied such violent intentions. Simultaneously, in response to his therapist's breach of confidence, he broke off treatment. Two months later he murdered his girlfriend. The therapist and his psychiatrist-supervisor were then sued by the woman's parents for failing to warn them of her peril.)

The case, while highly unusual, places in bold relief the dilemmas of therapists in balancing the rights to confidentiality of individuals with the protection of society from danger. Several psychotherapists (51, 52) have been antagonistic to the decision because of its conceptual and practical flaws and its negative implications for psychiatry. Roth and Meisel (51), for example, stated that the decision assumes a degree of expertise in predicting violence or danger that the psychiatrist simply does not possess; that the result will be a confusion and lowering of the threshold of dangerousness for issuing warnings to intended victims, which can compromise the patient's right to confidentiality and possibly his or her treatment; that the psychiatrist is liable not only if he or she fails to warn but also for invasion of privacy or defamation of the patient if the threat of harm does not materialize; and, finally, as actually happened in the *Tarasoff* case, the patient's dangerousness was probably increased because of his sense of betrayal at the therapist's commitment attempt and the patient's premature termination of the very treatment he needed. On a more conceptual level, Gurevitz (52) stated that the *Tarasoff* decision erroneously "defines and reenforces a social control function for psychiatry" by allying the psychotherapist "more with the goal of protecting society than with that of healing patients." It is not, Gurevitz pointed out, that psychiatrists reject the need to balance these functions; in fact, most psychiatrists at-

tempt to fulfill both responsibilities. However, they have done so with procedures that have not mandated them to routinely perform a duty that is "counter to their power of prescience."

Despite the *Tarasoff* decision, and no doubt because of it, alternative courses of action that can be taken by psychotherapists short of actual warning have been recommended. For example, due to their very strong convictions about the importance of confidentiality in the doctor-patient relationship, in no instance have Roth and Meisel (51) directly warned a potential victim without first obtaining the patient's permission. Since actual violence is relatively rare, they stated that it is prudent to "rely on odds and not warn." In addition, they suggested that therapists should inform the patient of the boundaries and limits of confidentiality. Even when danger seems imminent, the therapist should consider a number of social or environmental manipulations to reduce dangerousness before he or she makes the decision to compromise confidentiality. When confronted with a potentially violent patient, the therapist has options that include 1) continued therapeutic management of the patient, 2) involuntary hospitalization, 3) notifying the police, and 4) notifying the potential victims. Each of these choices of action places a differing weight on the competing values of confidentiality versus protection of the social order (or protection from "public peril").

There is also the question of the more private peril of the patient's danger to himself or herself. The following questions remain: Is the therapist able to predict danger to self any better than he or she can predict danger to others? If not, at what point does the duty to warn enter? Where does one draw the confidentiality line when the patient is a threat to himself or herself? Here issues of suicide and its active prevention may also ethically relate to the therapist's ideological position. Recently publicized cases of so-called "rational suicide," especially under the psychological and physical specter of terminal illness, along with the call for increased options for dealing with dying, suggest that the psychotherapist may be increasingly faced with the need to respect such choices by patients as part of his or her ethical responsibilities. In each such instance, the therapist and patient alike are obliged to confront and resolve their ethical dilemmas together.

THERAPIST-PATIENT SEX AND SEXISM

The Hippocratic Oath pledges that "with purity and holiness I will practice my art. . . . Into whatever houses I enter I will go into them for the benefit of the sick and will abstain from every voluntary act of mischief and corruption, and further from the seduction of females or males, of freemen and slaves" (53). The *Annotations Especially Applicable to Psychiatry* of *The Principles of Medical Ethics* (22) less eloquently but no less unequivocally upholds this moral tradition by stating simply, "Sexual activity with a patient is unethical." Indeed, it is the only specific activity deemed unethical between doctor and patient that is presented in so unambiguous a fashion. As part of the requirement that the physician "conduct himself with propriety in his profession and in all the actions of his life" (22), the dictum regarding sexual activity is especially important in the case of the psychiatrist because the patient tends to model his or her behavior after that of the therapist by identification. Further, the intensity of the therapeutic relationship may activate sexual feelings and fantasies on the part of both patient and therapist, while weakening the objectivity required for treatment. Insofar as it has earned a position of such priority, therapist-patient sex may be considered the ultimate expression of the overt misuse and exploitation of the transference relationship.

Nonetheless, sexual activities (including sexual intercourse and various forms of erotic contact between therapist and patient) have been increasingly reported in the literature; they have involved clinicians at all levels of training, from psychiatric resident to training analyst (54–61). Despite appearances, this is hardly a new problem for psychotherapists, who have reported erotic transferences and their vicissitudes since the dawn of psychoanalysis. Mesmer, Breuer, Janet, and Charcot, as well as Freud, have amply described the emergence of strong sexual feelings in treatment and the inevitable problems wrought by their presence. Although affairs between therapist and patient were never sanctioned by Freud or his followers, they did occur. However, therapists were often saved from moral indictment, as they still are, by marriage to the patient (and, of course, the termination of treatment by the spouse-therapist). Such a "shotgun" resolution between therapist and patient may be a rather extreme and limited option for dealing with sexual acting-out deriving from the therapeutic relationship. The question still remains, What are the ethical options if the therapist does not marry the patient?

In a current assessment of the legal and professional alternatives in deterring, disciplining, or punishing sexual activity between therapist and patient, Stone (62) cited four possible avenues of approach: 1) criminal law (e.g., charges of rape by fraud or coercion), 2) civil law (e.g., malpractice suits), 3) medical boards (e.g., revoked licensure), and 4) professional associations (e.g., pressure to limit referrals and threatened career opportunities). Each in its turn has been virtually ineffectual thus far in providing an effective system of control. In the instance of criminal law, rape charges, which were strongly recommended by Masters and Johnson (63), are rarely brought and rarely stick; most cases involve psychological coercion, not physical coercion. Both force and fraud appear to be required, and the prevailing judicial view is that if the patient consents and the therapist never claimed that

sexual activity was treatment there has been neither force nor fraud. In the instance of civil law damages or malpractice cases, again no legal course may be available if the therapist has not misidentified the sexual activity as treatment. In one recent publicized incident, two factors were held in the therapist's favor: the patient pressed charges a year and a half after the sexual relationship began, and the patient presumably did not have a normal transference. (Legally the therapist was found "negligent," and he continued to practice without his medical license.) Medical licensing boards are not consistent from state to state and often do not have a close relationship with members who are psychiatrists. Finally, professional associations generally have no subpoena power and little expertise in evidentiary investigation, either to protect the due process rights of the therapist charged or if the therapist sues them. Davidson (34) pointed out that going into treatment may be another way for the seductive psychiatrist to escape censure; treatment thus serves to sabotage efforts at discipline. Given the above failings of sanctions from without, Stone (62) was forced to conclude that in the end "patients must depend on the decent moral character of those entrusted to treat them."

What constitutes "decent moral character," however, may be changing with the times, at least according to the findings of some psychotherapists. We have not only Masters and Johnson's extreme stance on the matter (i.e., therapists who have sex with their patients should be charged criminally with rape, no matter who initiated the seduction) (63) but also attitudes on the other side of the scale. Evidence suggests that the sexual value system of psychiatric clinicians has evolved in the direction of increased sexual permissiveness in terms of what is acceptable for themselves and what is acceptable for others (64). In addition, there is the current overt endorsement of touching and so-called "nonerotic" physical behaviors, especially by advocates of the human potential movement (65, 66). Such activities, far from being regarded as unethical or harmful, are viewed as promoting personal growth and enhancing the therapeutic relationship.

The range of opinions and/or activities on the matter appears to reflect not only individual predilection but also theoretical orientation (67) and major medical specialty (57). Kardener and associates' survey of physicians' erotic and nonerotic physical contact with patients (57) may be relatively heartening in that it found psychiatrists among the lesser offenders in comparison with other medical specialists. Psychiatrists' reported rates of erotic contact—10%—and sexual intercourse with patients—5%—were lower than those of general practitioners, surgeons, and obstetrician/gynecologists. Moreover, substantial differences regarding therapist-patient sex were found between psychiatrists with "psychodynamic" and those with other theoretical orientations. For example, although 86% of the therapists with a psychodynamic orientation felt that erotic contact with the therapist would never benefit the opposite-sex patient, this figure was substantially lower for humanistic and behavioral therapists (71% and 61%, respectively). That there is widespread ambivalence on the subject even after the fact is reflected in Taylor and Wagner's review of cases of therapists who had actually had intercourse with their patients (68). Less than half (47%) reported that the experience had had negative effects on either patient or therapist, 32% reported that it had mixed effects, and 21% that it had positive effects. (The authors did not survey the patients.) Butler (58), however, found that 95% of therapists who had sex with their patients reported conflict, fear, and guilt; yet only 40% sought consultation for their problems.

In brief, available evidence suggests that erotic practices with patients do not conform, nor have they ever conformed, to medicine's ethical dictates; neither are any real risks taken by those who engage in them. Those therapists who may not themselves engage in sexual activity with patients have lent subtle support to the practice by protecting their errant colleagues with silence or with treatment (34). Several questions remain: Is such behavior unequivocally unethical regardless of outcome? If erotic contact is decidedly unethical, how ethical is the "nonerotic" kissing, hugging, and touching that more than 50% of a sample of psychiatrists said they engaged in with patients (57)? When, if ever, are these appropriate?

Others raise a more subtle issue: Are sexual relations with a patient (nearly always a female patient) simply the tip of the iceberg of the more pervasive practice of sexism in psychotherapy (69, 70)? The ethics of psychotherapy in relation to patient gender has implications not only for its specific abuses (e.g., sex between the male therapist and the female patient) but for other forms of sexual exploitation and discrimination. They may be manifested in psychotherapy's underlying theory, training practices, and doctor-patient relationship. Although it may well be difficult (even unethical) to do otherwise because of subtle and pervasive social pressures, therapeutic theories have more often supported than questioned stereotypical assumptions about sex roles. Broverman and associates (71) noted different standards of mental health for women and men, including tacit assumptions that dependency and passivity are normal for women whereas assertiveness and independence are normal for men. Women who are unhappy in their traditional role have often been considered psychopathological (69).

In addition to the unfortunate legacy of an "antifeminine Freudian position" (69), women may also be harmed by a "blame-the-mother" tradition, especially by therapists positing their potential as "schizophrenogenic" to their children (70). Such sex role biases are then often compounded during the therapist's pro-

fessional training, in which androcentrism (i.e., male chauvinism) is not likely to be corrected by a male supervisor (70). Indeed, female therapists are much less likely to have had supervisors of their own sex as role models during training; some even have had none (72). By far the most insidious issue of sexism in the practice of psychotherapy has been a function of the predominance of male therapists and the resultant tendency in psychotherapy to replicate within the dyadic relationship the "one-down" position in which women are typically placed. This may encourage the fantasy that an idealized relationship with a powerful other is a more desirable solution to life's problems than taking autonomous action (56, 70). Such a posture, in fact, may set the stage for the kind of sexual exploitation that occurs in instances of therapist-patient sex.

In the final analysis, ethical recommendations for dealing with both sex and sexism in psychotherapy would approximate the constellation of factors found in the new feminist therapies: a greater egalitarianism between therapist and patient in an active attempt to balance the therapist and patient's social power in the therapeutic endeavor and a conscious recognition of the necessity at all times to provide an ethical role model for the patient.

CONCLUSIONS

While ethical concerns have no doubt been with the field of psychiatry since its inception, *The Principles of Medical Ethics with Annotations Especially Applicable to Psychiatry* (22) provides a rather modest addendum of suggested standards for the profession. Unfortunately, some therapists feel that, however unprecedented its appearance and despite its noble aspirations, it has serious limitations in assisting them to make ethical decisions in their daily practice. It is understandably disappointing to those who confuse codes with covenants or who expect to magically produce morally scrupulous psychiatrists.

Aside from the merits or failings of a code of ethics itself, Zitrin and Klein (73) insisted that the more pressing problem is not the establishment of guidelines but their enforcement. The sentiment here is that a document can always be revised to better meet the psychiatrist's needs, but there are major problems in self or peer review processes and procedures. These problems include the voluntary nature of complaint investigations, the conflicting roles in which professional committees are required to act (as investigator, prosecutor, judge, and jury), inaccessibility and bureaucratic barriers to the system, inaccurate (and insufficient) case reporting, fear of liability by the professional reviewer, and overconcern with confidentiality, which often takes precedence over other ethical considerations and can be used as a rationalization to resist investigation (74). In actuality, however, it is exceedingly difficult to know what really occurs within the therapeutic relationship. Psychotherapists, like other professionals, are naturally reluctant to judge their colleagues, nor may they feel morally or technically equipped to do so. For example, Sullivan (75) reported that of more than 200 known charges of unethical conduct brought against psychiatrists within a 26-year period (1950–1976), only 8 psychiatrists were more than reprimanded.

The ethics not only of the profession but of peer review is also of concern (75). Here a major criticism is that the patient has been excluded from the design process and has been poorly informed about current procedures. Consequently, a prevailing view suggests that psychiatry at this time is simply unable to police itself (73) and that peer review, as currently constructed, is "bound to fail" (76). Nonetheless, we have made and continue to make positive headway in the peer review process (77).

The expectations held for a code of ethics and for peer review may be overendowed, especially by practitioners not directly involved in their development and implementation. For example, in his examination of the true roles and functions of a code of ethics, Moore (74) defined two major purposes as "structuring" and "sensitizing." The first has a basically preventive value and aims to hold back impulsive or unethical behavior; the latter has an essentially educative value and aims to raise one's ethical consciousness. However, a code of ethics can be misused by the moralist or therapist with personal motives that are essentially punitive, not educative or preventive. Moore made the point that a code of ethics should not be viewed as a vehicle for revenge, vindication, or private gain. Comparably, Newman and Luft (78) considered the primary purpose of the peer review process to be education, not control.

These authors suggested that an educational peer review system promoting cooperation among professionals would be of greater utility and more acceptable to clinicians than a bureaucratic system of control, which might tend to foster manipulation. Since the question remains as to how much authority should be vested in peer review committees, using such systems involves very powerful and delicate processes. Ultimately the need for continuing and remedial education for the practicing psychotherapist must be addressed, both for the clinician and for the selection, training, and evaluation of reviewers. More active development of codes of ethics as basic guidelines, not disciplinary instruments, can be used to help elucidate ethical conflicts and the part they play in the life of every psychotherapist.

In conclusion, as I have suggested throughout this paper, the problem of ethics in the practice of psychotherapy is not entirely soluble in that there is no single answer to the varied and complex dilemmas psychotherapists face in relation to the patient and to society.

Thus at this time we cannot completely rely on codified instruments—nor should we. Ideally, with the guidance of our professional peers, we must seek individual answers to the ethical problems that inevitably arise. I think the following suggestions would maximize the exercise of ethical choices by the psychotherapist:

1. Greater exploration of the philosophical foundations of therapeutic practice and the ethical assumptions on which psychotherapy is predicated.

2. Awareness of one's own self and personal commitments, for example, by constant examination and analysis of attitudes and behaviors within and outside of the therapeutic relationship.

3. Active development within the treatment endeavor of a "therapeutic alliance" or partnership in which there is equal power and participation by both parties toward mutual goals and responsibilities.

4. Greater allegiance to a code of ethics and its development to better sort out one's ethical choices and their implications for both patient and therapist.

5. Greater responsibility by the clinician for the maintenance of professional competence for himself or herself and others of the profession.

6. Openness to consultation with others and receptivity to outside opinions in making the best ethical decisions in treatment.

7. Greater understanding of human nature and morality from which timely and productive ethical alternatives can be derived.

As Bernal y del Rio (79) aptly put it, "By definition, ethical problems remain unresolved. By their unresolved quality, they provoke a continuous anxiety in the practicing psychiatrist and concomitantly a desire to search, to oppose, to think, and to research."

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Chronic Crisis Patients: A Discrete Clinical Group

BY ELLEN BASSUK, M.D., AND SAMUEL GERSON, PH.D.

Patients who made repeated visits to a psychiatric emergency ward and who were studied appeared to comprise a discrete clinical group characterized by a distinct symptom profile, common psychiatric history, and similar styles of interacting with caretakers. They generally did not differ from nonrepeater patients with regard to acute symptomatology but were hospitalized more frequently. They were generally negativistic, had a lengthy psychiatric history consisting of multiple inpatient hospitalizations and current outpatient treatments, and had difficulty establishing rapport with the emergency ward therapists, often evoking intense feelings of dislike. The authors offer an approach to the management of these patients based on an understanding of their cycle of seeking and rejecting help.

Psychiatric emergency services positioned at the interfaces between the community and the hospital and between inpatient and outpatient facilities sensitively mirror changes in the ideology and provision of mental health services. With the dramatic reduction in the population of state hospital inpatient units, ambulatory services have gradually assumed greater responsibility for the care of the chronically ill patient (1). This shift in the locus of care has been a major factor in the phenomenal expansion of psychiatric emergency services during the last two decades and will undoubtedly contribute to increasing demands on these services in the next decade (2). To effectively meet the challenge of providing acute management for the broad spectrum of psychiatric patients, it is becoming increasingly important to delineate subgroups of patients who share common characteristics and for whom specific treatment approaches can be developed.

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One such group of patients are those who, despite persistent efforts to engage them in ongoing treatment, keep returning to the emergency ward. Difficulties in providing continuity of care and preventing these patients, known as repeaters, from becoming part of the emergency ward revolving door is a major problem facing the clinician in this setting.

Previous studies of repeaters (3–7) have reported that 7%–17.9% return to the emergency ward within 6 months of the initial visit. Although one study (4) reported no demographic differences between repeaters and nonrepeaters, another (3) concluded that more than 90% of repeaters were women and 56% were divorced, widowed, or separated. In the latter study, repeaters were described as having a slightly different symptom profile characterized by a higher level of suicidal ideation, depression, psychomotor retardation, and anorexia.

In a study of repeaters to a 24- to 48-hour holding facility (6), the authors found that 13.1% were seen 2–7 times. An interesting observation was that multiple visits were related to a single episode of illness and generally occurred within days of each other. Most of these patients were women with chronic personality disorders, frequently accompanied by alcoholism and drug dependence. In contrast, most of the nonrepeaters were given the diagnosis of transient situational disorders.

To our knowledge, the only report in the literature discussing the clinical issues in the management of the repeater group is an anecdotal description of 15 patients who each made 7–30 visits to the emergency ward within one year (5). During at least one admission most were labeled as borderline, although 5 were also diagnosed as having chronic schizophrenia. This group returned repeatedly during recurrent crises with urgent demands for immediate relief of their distress related in a provocative, often help-rejecting style. Their manner of relating may significantly contribute to their difficulties in becoming engaged in a stable psychotherapeutic relationship and, therefore, to their frequent use of emergency services.

A rational treatment approach to the clinical management of these difficult patients demands an elaboration of their common distinguishing characteristics. Patients comprising this group represent a sizable proportion of most emergency ward populations and consume an even greater percentage of personnel time. To improve patient care and the efficient use of resources,

we designed a study to clarify our understanding of the repeater group and the reactions they elicit in their caretakers.

METHOD

This study was conducted at the Beth Israel Hospital in Boston, Mass., a private general hospital affiliated with a medical school. The psychiatric emergency service, located in the emergency unit, provides 24-hour care 7 days a week and is primarily staffed by first-year psychiatric residents and first-year psychology interns. Approximately 1,400 psychiatric patients are seen each year. The overall pattern of utilization is similar to that reported in the many descriptive studies in the literature of similar urban settings.

The total sample consisted of 307 patients treated by 18 therapists. Of these, the 50 patients (16.3%) who were seen in the psychiatric emergency service more than once during the previous 12 months were defined as repeaters. The choice of one year as the time frame for defining repeaters was based on the clinical assumption that most repeaters return for subsequent treatment within that period.

At the conclusion of the assessment and disposition of each patient, data on a variety of demographic and clinical variables were gathered. The tests used included the Brief Psychiatric Rating Scale (BPRS) (8), which measures the patient's clinical characteristics, and the Global Assessment Scale (GAS) (9), which measures the overall severity of the patient's functional impairment. In addition, the therapist's affective response to the patient was measured on a self-report scale consisting of nine items, each scored on a 7-point scale, rating rapport with, interest in, and emotional reactions to the patient. A total score of 63 is possible on this scale; scores of 23 or lower represent an intense negative response, and scores of 50 or higher represent a strong positive response to the patient (10).

To delineate patient characteristics associated with frequent use of psychiatric emergency services, the repeater group of patients was compared with the nonrepeaters on the following six dimensions: demographics, psychiatric treatment history, social supports, clinical symptomatology, therapist's affective response, and disposition.

RESULTS

Demographically, the 50 repeaters and 257 nonrepeaters differed significantly only on the age variable; there was a significantly greater likelihood of being a repeater if the patient was between 31 and 50 years of age ($\chi^2=24.91$, $p<.001$). Although there was a tendency for more women and housewives to be repeaters, the results were not significant. Race, marital status, and socioeconomic class did not differentiate the two groups.

TABLE 1
Psychiatric History of Patients with Repeated Visits and Patients with Single Visits to a Psychiatric Emergency Service

Variable	Percent of Repeaters	Percent of Nonrepeaters
Past inpatient treatment (N=307)[a]		
Yes	86	36.2
No	14	63.8
Number of previous hospitalizations (N=136)[b]		
1-2		
3-5	30.2	74.2
6 or more	23.3	18.3
Current and past psychotherapy (N=307)[c]	46.5	7.5
Never in therapy	18.0	50.6
Currently in therapy	60.0	29.2
Not currently but previously in therapy	22.0	20.2

[a] $\chi^2=40.10$, df=1, $p<.001$.
[b] $\chi^2=32.31$, df=2, $p<.001$.
[c] $\chi^2=21.50$, df=2, $p<.001$.

TABLE 2
Clinical Characteristics of 307 Patients Seen on a Psychiatric Emergency Service

Variable	t[a]
BPRS symptom	
Somatic concern	2.57[b]
Anxiety	−1.16
Emotional withdrawal	−1.08
Conceptual disorganization	1.71
Guilt feelings	−6.42[c]
Tension	− .87
Mannerisms and posturing	1.83
Grandiosity	1.16
Depressive mood	−1.22
Hostility	4.53[c]
Suspiciousness	2.57[b]
Hallucinatory behavior	1.89
Motor retardation	−2.04[d]
Uncooperativeness	4.29[c]
Unusual thought content	2.10[d]
Blunted affect	1.30
Total BPRS score	2.14[d]
Global Assessment Scale (GAS) score	6.10[c]
Degree of suicidal threat	.46
Degree of homicidal threat	.28

[a] Positive t values indicate that the symptom was more prevalent in repeaters.
[b] $p<.01$.
[c] $p<.001$.
[d] $p<.05$.

The current and past psychiatric treatment of the repeaters significantly differed from that of the rest of the emergency ward patients (see table 1). More of the repeaters were involved in ongoing psychotherapy, and more of them also had received previous inpatient treatment. Of the patients who had been previously hospitalized, the repeater group had a significantly greater number of inpatient episodes.

The network of social supports available to repeaters was significantly more sparse than that available to

nonrepeaters; 46% of the repeaters, but only 26.4% of the nonrepeaters, said they had one or no supports ($\chi^2=15.36$, $p<.001$). The scanty social supports of the repeaters was also evidenced in the finding that the great majority of repeaters (84%) arrived on the emergency ward unaccompanied by friend or family, compared with only 38.5% of the nonrepeaters ($p<.001$).

The major clinical difference between the two groups of patients was in their response to the emergency ward therapists. The repeaters were rated as significantly more hostile, suspicious, and uncooperative than the nonrepeaters (see table 2). The groups did not differ significantly on ratings reflecting the clinical symptoms of acute psychoses, affective disorders, or anxiety reactions. Overall ratings of psychopathology (total BPRS score) and functional impairment (GAS score) were significantly higher for the repeater patients.

Approximately one-half of the repeaters were considered to have character disorders and one-half psychoses, compared with approximately one-third of the nonrepeaters for each of these categories.

Measurements of the therapist-patient interaction revealed that the repeaters as a group tended to elicit a generally negative affective response from their emergency ward therapists. Thirty-two percent of the repeater group but only 4.3% of the nonrepeaters were found to be intensely disliked (therapists' scores of 12–23); only 8% of the repeaters, compared with 31.5% of the nonrepeaters, were found to engage in a highly positively toned interaction with the therapist (scores of 50–62) ($\chi^2=45.05$, $p<.001$).

Fifty-two percent of the repeaters and 33.9% of the nonrepeaters were referred for hospitalization ($\chi^2=16.78$, $p<.01$).

DISCUSSION

The data indicate that the repeaters represent a discrete clinical group characterized by a common symptom profile, a similar treatment history, and a typical manner of interacting with therapists. These three dimensions are interrelated, continuously reinforcing each other and resulting in a self-defeating style of seeking help and then rejecting it. The repeater group can be defined as chronic crisis patients; they share similar symptomatic and historical characteristics with a group of borderline patients described in a recent study conducted on an emergency ward (11). A cross-sectional analysis of their behavior illustrates the patterns that generate and maintain their status as repeaters.

Beginning with the symptom profile, these patients relate to caretakers in a generally negativistic manner and have been described as "entitled demanders" and "manipulative help rejecters" (12). Often they come to the emergency ward with the unrealistic expectation of immediate gratification of their wishes and instant relief of their discomfort. Their neediness is accentuated by their inability to effectively communicate reality-based wishes and their ambivalence about involvement in a potentially close relationship. They transmit intense anger and communicate in a provocative style. As our findings indicate, the therapist responds to this symptom profile with feelings of anger and dislike and has difficulty establishing a relationship.

The lack of rapport and the therapist's intense negative affect may contribute to the greater likelihood of hospitalizing such patients. The finding that repeaters and nonrepeaters did not differ significantly in the degree of acute psychotic, suicidal, homicidal, anxious, or depressive symptomatology suggests that other factors were involved in planning the disposition of the patient. The tendency to hospitalize repeaters more frequently than nonrepeaters may therefore be more of a reflection of the difficulty in establishing meaningful contact with the former group than of the clinically based need for inpatient care.

Further support for the decision to hospitalize may come from the striking paucity of the repeater's social network. Clinical experience and historical data suggest that the manner in which these patients communicate their needs often leaves other persons emotionally exhausted and depleted, not unlike the feelings that the emergency ward therapist experiences in attempting to manage this group. Over a period of time this is likely to result in a gradual recession of potential resources, a factor that has been shown to be a major determinant in dispositional decision making (13).

The psychiatric history of the repeater patient appears to be another nonclinical factor that affects decision making. For example, the finding that repeaters were diagnosed as psychotic more frequently than were nonrepeaters, even though the two groups did not generally differ in clinical ratings of psychotic symptomatology, may illustrate the influence of the patient's history. With regard to disposition, therapists may be overly influenced by the impressively large number of previous hospitalizations and may regard this as the treatment of choice. The resulting hospitalization only seems to perpetuate the cycle of rejection of help. A similar process may also occur in an outpatient treatment context.

To bypass this pattern the therapist should view the engagement and containment of these patients in any setting as a protracted process characterized by frequent flights from the helper. This disengagement is an expectable part of the behavior of some patients when involved in treatment. It partially reflects their incapacity to negotiate a comfortable distance without feeling isolated or a reasonable closeness without feeling engulfed. They are unable to tolerate the limits of a relationship and defend against it by seeking an alternative source of support, again repeating the cycle.

For the purpose of discussing an approach to the more effective emergency ward management of repeaters, they can be divided into two subgroups: those currently involved in a treatment program and those without therapeutic support. As the data indicate, many repeaters who visit the emergency ward are seeing an outside therapist and often have concerns and complaints about their treatment. This commonly is a manifestation of a transference bind and should be viewed as an inevitable part of the process of psychotherapy. Often when the patient cannot tolerate ambivalent feelings about the therapist, he or she may use the defense mechanism of splitting (14). The patient may view the therapist as the bad, devalued object and the emergency ward clinician as the good, overidealized object or vice versa. The emergency ward clinician should recognize this defensive operation and attempt to delineate the precipitant of the visit, focus on the therapeutic relationship, reality test some of the patient's distortions about the therapist, and encourage the patient to return and discuss his or her feelings with the therapist. In addition, and after discussion with the patient, the emergency ward clinician should notify the therapist of the patient's visit and develop an integrated approach to his or her management (15).

Treatment of the repeater who does not have a therapist is more difficult. As previously described, these patients may be repeatedly hospitalized because they lack a support network, tend to reject help, and have a history of psychiatric treatment. However, the same principles of management should be applied to this subgroup. Most importantly, an attempt should be made to engage these patients in ongoing treatment. For those few repeaters who have not been previously hospitalized or been in psychotherapy, strenuous efforts should be made to engage them in treatment. This may sometimes necessitate a short-term inpatient stay to complete a diagnostic assessment and initiate treatment. Such an inpatient stay should be weighed against the possibility of a severe regression, which often marks the clinical course of these patients.

Most repeaters have made abortive attempts to become involved in psychotherapy or in various treatment programs. As Raphling and Lion (5) have suggested, these patients should be managed on the emergency ward and their repeated visits should be viewed as opportunities to engage them in a therapeutic relationship. Consistent with the principles of aftercare, some of these patients will ultimately form a transference to the emergency ward that will help to effectively contain them at an optimal level of functioning (16). This approach might dilute the intensity of the transference, but it may sacrifice the possibility of working through aspects of the patient's problems with closeness in relationships. To successfully manage patients in this setting and to avoid further splitting, it is essential to have frequent and consistent communication with each member of the emergency ward staff who comes into contact with the patient. Most often the patient's previous behavior and history can offer clear indications for the most appropriate therapeutic stance.

What is most difficult in the management of these patients, however, is not the development of a dispositional approach, but rather the management of the therapist's feelings in response to the repeaters' provocative style. Because these patients are often intensely hostile, manipulative, uncooperative, and devaluing, the therapist experiences anxiety, helplessness, and even hate (11). To manage these feelings, the therapist must repeatedly remind himself or herself that the patient's style repeats aspects of the troubled early mother-child relationship. It is, in part, a learned response and has become a model for later relationships despite its distortions. An understanding of this will help the therapist remain empathetic in the face of the patient's provocativeness. The therapist should make every attempt to bypass the struggle while acknowledging to himself or herself the intensity and discomfort of these feelings. Often it is necessary to set limits on the patient's inappropriate behavior while simultaneously providing support and reassurance and clarifying aspects of the patient's help-rejecting stance.

CONCLUSIONS

Although the general hospital is gradually assuming some of the functions of the state hospital, it has not yet developed a comprehensive range of intermediate services such as partial hospitalization, halfway houses, and community residences. In addition, in many cases formal affiliation agreements that directly guide the flow of patients through the complex mental health system have not been defined. It is our clinical impression, supported by discussions with other emergency ward directors, that repeaters use multiple points of entry simultaneously, thereby establishing many opportunities for splitting and for sabotaging the existing therapeutic relationship or preventing one from developing. Communication through a committee of regional directors of emergency ward services can often help in the management of this difficult group of patients.

Identifying one facility as the major caretaker and notifying it of the patient's use of other settings will further improve the patient's care. Community mental health centers with many integrated and diverse treatment components might be more appropriate settings for the management of the repeater group because they offer a wider range of treatment alternatives within a centralized setting. However, if the patient has access to multiple psychiatric facilities within the geographic region, broader communication among these services must be established. The most important prin-

ciple in the care of these patients is that a single treatment facility, such as the emergency ward, be identified as the primary therapist and be responsible for planning a consistent treatment approach. This offers the patient the greatest possibility of containment, engagement in a treatment process, and eventual resolution of his or her problems.

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Relationship of Neuroleptic Drug Effects at Brain Dopamine, Serotonin, α-Adrenergic, and Histamine Receptors to Clinical Potency

BY STEPHEN J. PEROUTKA, M.D., AND SOLOMON H. SNYDER, M.D.

The authors examined the potencies of 22 neuroleptic drugs competing for binding sites associated with dopamine, serotonin, α-adrenergic, and histamine receptors in brain membranes. They found that although many neuroleptics are quite potent in competing at several of these receptor sites, the average antipsychotic clinical potency correlates closely only with the drug affinity for dopamine receptors labeled by ^{3}H-spiroperidol. At clinically effective doses, however, substantial occupancy of serotonin, α-adrenergic, and histamine receptors often occurs and may account for some of the auxiliary actions of neuroleptics.

Early pharmacological studies suggested that the antischizophrenic neuroleptic drugs exert their therapeutic actions by blocking dopamine receptors (1). Although phenothiazine neuroleptics are potent inhibitors of a dopamine-sensitive adenylate cyclase in brain tissues, the butyrophenone class of drugs, which includes the most potent neuroleptics, is comparatively weak at these receptors (2, 3). With the advent of techniques to label neurotransmitter receptors in the brain by binding procedures (4), it became possible to directly measure the recognition site of dopamine receptors through the binding of ^{3}H-neuroleptics such as ^{3}H-haloperidol (5, 6) and ^{3}H-spiroperidol (7, 8).

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Neuroleptics are potent inhibitors of ^{3}H-haloperidol binding to dopamine receptors, and their effects at these sites correlate closely with clinical potencies in humans and pharmacological activities in animals (9, 10). However, when techniques were developed to label other neurotransmitter receptors, it was found that neuroleptics are also quite potent at muscarinic cholinergic (11), α-adrenergic (12), serotonin (13–15), and histamine H_1 (16) receptors. In several instances neuroleptics are just as potent or even more potent at these other neurotransmitter sites than at dopamine receptors. For instance, clozapine is approximately 10 times as potent at certain α-adrenergic (12), serotonin (13, 15), and histamine (16) receptors than at dopamine receptors (6, 9, 17). Spiroperidol, on the other hand, has a similarly high affinity of 0.6×10^{-9} M at both dopamine and serotonin receptors.

Accordingly, it is conceivable that the antischizophrenic effects of neuroleptics may be associated with actions at sites other than dopamine receptors. To explore this possibility, we have evaluated a series of 22 neuroleptics for their effects at dopamine receptors labeled by ^{3}H-spiroperidol in rat caudate, serotonin receptors labeled by ^{3}H-spiroperidol in rat frontal cortex, α-adrenergic receptors labeled by ^{3}H-WB-4101 (2-([2′,6′-dimethoxy] phenoxyethylamino) methylbenzodioxane) and histamine receptors labeled by the antihistamine ^{3}H-mepyramine. We confirm and extend earlier findings (9, 10) that an extremely close correlation exists between average clinical potency and drug affinity for brain dopamine receptors. Additionally, we report here that no significant correlation was observed between clinical potency and neuroleptic affinity at other neurotransmitter receptors despite comparatively high drug affinity for these other sites.

METHOD

Radioreceptor binding assays were performed essentially as described previously (9, 12, 15, 16). Briefly, caudate and frontal cerebral cortex were dissected from freshly decapitated male Sprague-Dawley rats and homogenized with a Brinkmann Polytron in 50 mM Tris-HCl buffer (ph 7.7 at 37° C). The tissue was washed twice by resuspension and centrifugation. Fi-

TABLE 1
Average Clinical Daily Dose for Neuroleptics and In Vitro Potency at Neurotransmitter Receptors

Neuroleptic	Average Clinical Dose (mg/day)	Inhibition of ^{3}H-Ligand Binding K_i (nM)			
		^{3}H-Spiroperidol			
		At Dopamine Receptors (caudate)	At Serotonin Receptors (cortex)	^{3}H-WB-4101 at α-Adrenergic Receptors	^{3}H-Mepyramine at Histamine H_1 Receptors
Phenothiazines					
Fluphenazine	15	3.7	25	13	58
Trifluoperazine	25	4.4	24	67	135
Chlorpromazine	600	25	19	4.3	28
Thioridazine	625	63	16	7.1	25
Promazine	660	280	130	9.4	25
Butyrophenones					
Spiroperidol	1.5	0.68	0.59	14	550
Benperidol	1.6	1.4	3.7	7.1	200
Clofluperol	2.4	4.2	42	56	300
Trifluoperidol	3	2.7	6.8	8.4	2,100
Bromperidol	4	3.7	26	100	700
Droperidol	8	3.0	4.6	1.4	2,500
Haloperidol	12	4.4	45	14	2,600
Moperone	22	9.3	52	22	22,000
Fluanisone	100	16	36	1.8	150
Pipamperone	450	360	6.1	100	450
Others					
Fluspirilene	2	2.6	5.9	240	1,700
α-Flupenthixol	4	3.0	13	8.2	39
Pimozide	4	3.6	25	20	1,100
(+)-Butaclamol	6	3.8	2.6	20	470
cis-Thiothixene	30	4.5	36	11	37
Penfluridol	30	7.8	150	280	3,400
Clozapine	725	380	29	17	20

nal resuspensions were made in 0.1% ascorbic acid, 10 μM pargyline, 120 mM NaCl, 5 mM KCl, 2 mM $CaCl_2$, and 1 mM $MgCl_2$ for ^{3}H-spiroperidol studies. The tissue in the ^{3}H-WB-4101 experiments was resuspended in 50 mM Tris-HCl buffer, and the tissue used in ^{3}H-mepyramine experiments was resuspended in Na^+-K^+-phosphate buffer (50 mM, pH 7.5, at 37° C).

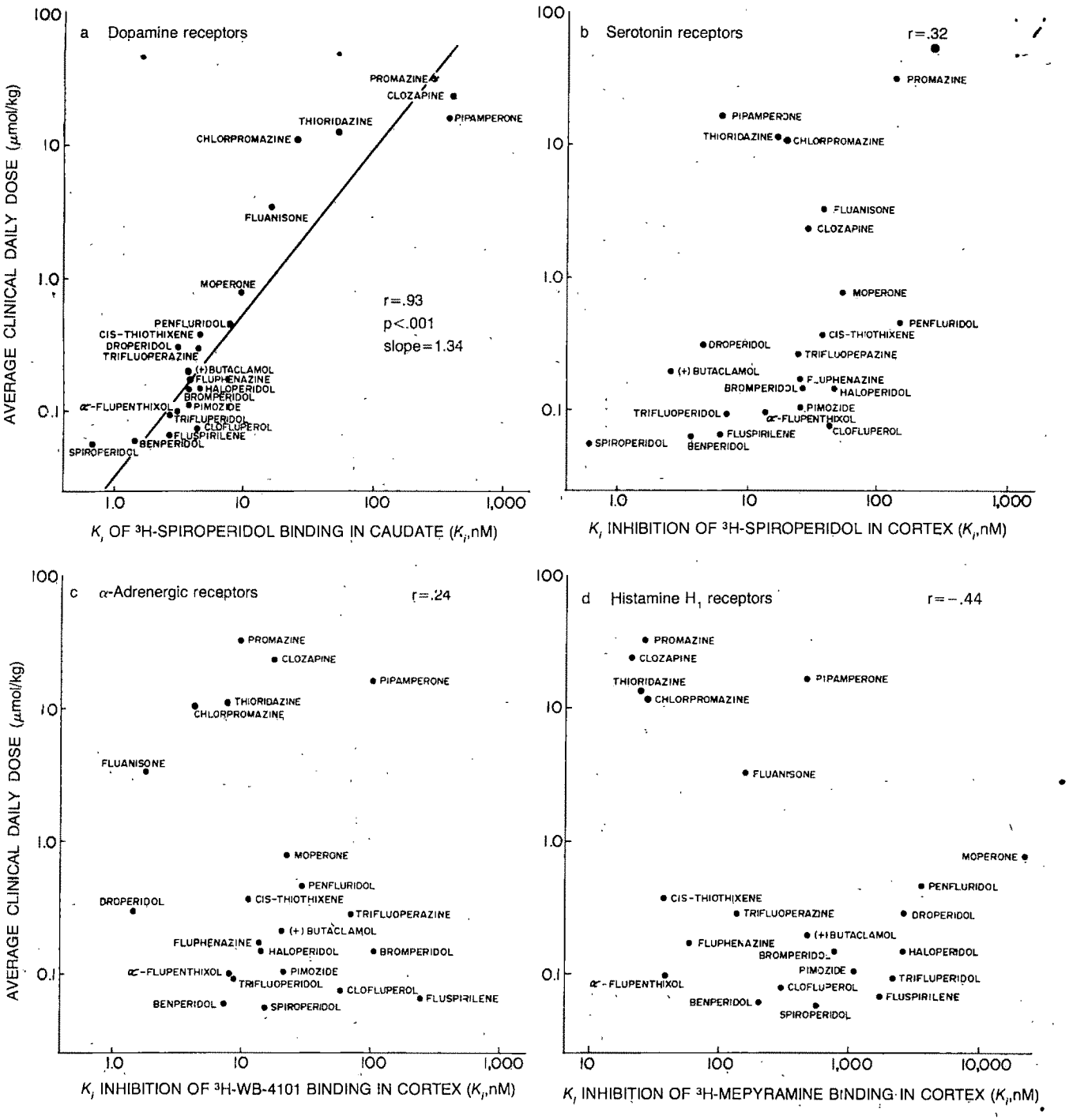
Each tube received 0.8 ml of tissue suspension, 0.1 ml of various concentrations of neuroleptic drugs, and 0.1 ml of ^{3}H-ligand. Final concentrations were 10 mg/ml wet weight of tissue and 0.25 nM ^{3}H-spiroperidol, 0.22 nM ^{3}H-WB-4101, or 2.0 nM ^{3}H-mepyramine. Triplicate tubes were incubated at 37° C for 15 minutes for ^{3}H-spiroperidol and at 25° C for 30 minutes for other ^{3}H-ligands and rapidly filtered under vacuum through Whatman GF/B filters with three 5-ml rinses of cold buffer. The filters were counted by liquid scintillation spectrometry.

Specific binding to dopamine receptors was defined as the excess over blanks taken in the presence of 1 μM (+)-butaclamol when ^{3}H-spiroperidol was assayed in the caudate. Serotonin receptors were labeled with ^{3}H-spiroperidol in the frontal cerebral cortex with specific binding defined as the excess over blanks taken in the presence of 1 μM *d*-LSD. Recent studies from several laboratories (13, 15, 18) have concluded that although ^{3}H-spiroperidol binding in the caudate labels only dopamine receptors, ^{3}H-spiroperidol binds almost exclusively to serotonin receptors in the frontal cerebral cortex where dopamine projections are much less evident.

Specific binding to α-adrenergic receptors was defined as the excess over blanks taken in the presence of 100 μM (−)-norepinephrine. Histamine H_1 receptors were labeled with ^{3}H-mepyramine with specific binding defined as the excess over blanks taken in the presence of 1 μM tripolidine.

For each drug, competition for the ^{3}H-ligands was measured at three or four concentrations of drug and the 50% inhibitory concentrations (IC_{50}) were derived from log-probit analysis. These values were converted to apparent K_is according to the equation $K_i = IC_{50}/(1+C/K_D)$ where C is the concentration of radioactive ligand and K_D is its dissociation constant. Each value reported is the mean of three or four separate experiments and is given in nanomolar concentration (10^{-9} M). Clinical potencies were derived from several studies (19–22). For each drug, the midpoint values of listed ranges of daily dose were averaged. The doses are presented in milligrams per day in table 1. In comparisons of clinical and receptor potency in figure 1, differences in molecular weight of the various drugs were taken into account by converting doses to micromoles. To facilitate comparisons with animal potencies (9), values were expressed as micromoles per kilogram, assuming a human weight of 70 kg.

FIGURE 1
Correlation of Average Clinical Dose with Neuroleptic Affinity for Dopamine Receptors (a), Serotonin Receptors (b), α-Adrenergic Receptors (c), and Histamine H_1 Receptors (d)



RESULTS

In confirmation of our earlier findings, we found that neuroleptics are potent inhibitors of ^{3}H-spiroperidol binding to dopamine receptors (table 1). Affinities for the receptor site labeled by ^{3}H-spiroperidol in the rat caudate are quite similar to values reported with ^{3}H-haloperidol binding (5, 6, 9, 10). As observed earlier, spiroperidol is the most potent agent at dopamine receptors labeled in this way, with a K_i value of approximately 0.7 nM. It is also the most potent neuroleptic reported in clinical studies. Benperidol and fluspirilene are similar to spiroperidol in that they are extremely potent neuroleptics and also have the second and third highest affinities for the dopamine receptor labeled by ^{3}H-spiroperidol. On the other hand, clozapine is clini-

cally the least potent neuroleptic of the 22 drugs studied; it also has low affinity for dopamine receptors, being less than 0.2% as potent as spiroperidol. A strikingly close correlation exists between average clinical potencies and affinities of the neuroleptics for dopamine receptors labeled by ^{3}H-spiroperidol ($r=.93$; $p<.001$) (figure 1a). In addition, the slope of the regression line is approximately unity.

In rat frontal cerebral cortex ^{3}H-spiroperidol predominantly labels serotonin receptors with negligible binding to dopamine receptors (18). Although many neuroleptics have high affinity for serotonin receptors (table 1), a different rank order of potency is observed when compared to relative potencies in blocking the ^{3}H-spiroperidol labeling of dopamine receptors in the caudate. For example, although spiroperidol has the same potency at the serotonin receptors in the cerebral cortex as at dopamine receptors in the caudate, clozapine is more than 10 times as potent at serotonin than at dopamine receptors. The most marked discrepancy between serotonin and dopamine receptors occurs with pipamperone, which is 60 times more active at serotonin than at dopamine sites. Conversely, agents such as clofluperol, haloperidol, and fluphenazine are substantially more potent at dopamine than at serotonin receptors. Therefore, although on the average neuroleptics have high affinity for serotonin receptors, we observed no statistically significant correlation between clinical dosages and potency at central serotonin receptors (figure 1b).

Earlier we showed that several neuroleptics are quite potent in competing for α-adenergic receptors labeled by ^{3}H-WB-4101 (12). The relative potencies of neuroleptics at α-adrenergic sites compared with those at dopamine receptors labeled by ^{3}H-haloperidol correlate closely with the sedative-hypotensive side effects of neuroleptics (12). In the present study we have confirmed and extended these observations (table 1). Neuroleptics with sedative properties such as promazine, droperidol, chlorpromazine, thioridazine, and fluanisone are among the most potent agents at these α-adrenergic sites. By contrast, bromperidol, clofluperol, spiroperidol, and trifluperazine are considerably more active at dopamine receptors than at α-adrenergic receptors and are not associated with sedative side effects. As was the case with serotonin receptors, no statistically significant correlation exists between clinical potency and affinity of neuroleptics for the α-adrenergic site labeled by ^{3}H-WB-4101 (figure 1c).

We found that promazine, thioridazine, and chlorpromazine are among the most potent neuroleptics at histamine H_1 receptors labeled by ^{3}H-mepyramine. This finding agrees with the pharmaceutical development of chlorpromazine and other neuroleptics as antihistamine agents (e.g., the widely used phenothiazine antihistamine, promethazine). In general, the phenothiazine neuroleptics are substantially more potent than the butyrophenones and other neuroleptics at histamine H_1 receptors. Again, however, we observed no correlation between the drug affinities and their clinical potencies (figure 1d).

DISCUSSION

The most striking finding of the present study is that many neuroleptics are extremely potent agents at serotonin, α-adrenergic, histamine, and dopamine receptors. We also found that clinical antipsychotic potency failed to correlate significantly with sedative effects at serotonin, α-adrenergic, and histamine sites, although we observed an extremely close and highly significant correlation between average clinical potency and drug affinity for dopamine receptors labeled by ^{3}H-spiroperidol. These results further strengthen the hypothesis that blockade of dopamine receptors may explain the therapeutic actions of neuroleptic drugs.

The correlation of effects on dopamine receptors with clinical potency is particularly striking considering the fact that clinical potency is determined by the absorption route, in vivo metabolism, and penetration into the central nervous system as well as by drug affinity for the target receptor. Presumably, these variations in bioavailability are averaged over the extensive series of neuroleptics evaluated here. In addition, reported clinical doses vary considerably (19–22), since it is difficult to complete well-controlled human studies with a large series of neuroleptics. Animal behavioral studies, however, have produced more precise in vivo data. Both apomorphine-induced (23, 24) and amphetamine-induced (23) stereotypy are behavioral correlates of dopamine overactivity in the brain. The ability of neuroleptics to block these behavioral syndromes is highly correlated ($r=.9$, $p<.001$) with their in vitro affinity for the dopamine receptor (9).

Our findings demonstrate that the correlation of clinical potency and affinity for dopamine receptors is highly selective, since no such correlation is observed with any of the other neurotransmitter receptors evaluated. This occurs despite the fact that the neuroleptics are often as potent or even more potent at serotonin, α-adrenergic, and histamine receptors than at dopamine receptors. Indeed, spiroperidol is currently the most potent drug known at both dopamine and serotonin receptors.

Because of the considerable potency of neuroleptics at multiple neurotransmitter sites, it is likely that at therapeutic doses these other neurotransmitter receptors are occupied and may account for certain side effects. For instance, the ability to block muscarinic cholinergic receptors correlated with a lowered incidence of the extrapyramidal side effects often associated with neuroleptics (11). The differential striatal and limbic actions of drugs may also be relevant (25). The observation of a relationship between the sedative-hypotensive effects of certain neuroleptics and their

ability to block α-adrenergic receptors (12) has been confirmed and extended by our study. The effect of serotonin blockade is less clear.

The antihistamine effect of neuroleptics appears to be limited to the phenothiazine derivatives, with all butyrophenones showing low affinity for central histamine receptors. It is unclear what behavioral effects would result from blocking central histamine H_1 receptors. The brain has a higher density of H_1 histamine receptors than any body organ examined (16), and some phenothiazine neuroleptics and tricyclic antidepressants are as potent at these sites as the most potent classical antihistamines (26, 27).

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Assessing Families in Their Own Homes

BY PETER STEINGLASS, M.D.

Assessment of the family in its own home provides important information about the family's style of regulating its internal environment. The author presents data from a study of families in which one of the spouses was alcoholic. These families were observed at home on nine occasions over a six-month period; the technique used, the Home Observation Assessment Method, measured dimensions of family behavior associated with routines of daily living. Dimensions of home behavior were highly correlated with traditional clinical measures of psychiatric symptomatology, severity of alcoholism, and family boundaries.

Although psychiatric clinicians have long been curious about family behavior in the home, home observation techniques have seldom been incorporated into clinically oriented research studies, and home visits are not usually included in the clinical assessment of individual or family patients. Despite enthusiastic reports of the benefits of home visits as a procedure allowing the clinician to evaluate the family on its own turf (1–4), practical considerations appear to have discouraged the large-scale use of home visits in clinical practice. A conceptual model clarifying how home observations might complement customary assessment procedures used in office and hospital settings has also been lacking.

In contrast to psychiatric clinicians, behavioral scientists, especially those interested in child development, have been generating a great deal of data about family behavior at home (5–8). However, although clinical inferences can often be drawn from these data, clinical issues are seldom central in the design of these studies (9, 10). As a result, clinicians have received little guidance about how to interpret these naturalistic data in relevant terms.

An interest in these issues and a desire to explore the relationships between a family's home behavior and its management of chronic psychiatric illness motivated me to undertake a clinical research study built around home observation. This decision stemmed from a conviction that the home setting is the best place to study an aspect of family life one might label the family's *regulation of its internal environment*. This aspect of family life is of particular interest to me because I suspect that family behavior related to control of its internal environment is a critically important factor determining the family's style of dealing with chronic illness. Since most psychiatric illnesses are chronic rather than acute in nature, a better understanding of such a relationship, if it in fact exists, would be of value to the psychiatric clinician.

In this particular study, alcoholism was the specific chronic condition examined. In another report (11) I dealt with aspects of home behavior that seem unique to alcoholism. In the present paper I will use data from the same study to focus on two issues related to the home assessment of family behavior: 1) Is it possible to objectively assess the family's regulation of its internal environment? and 2) What links can be drawn between data emerging from such an assessment and the more customary dimensions of behavior used by clinicians in assessing the family in an office or hospital setting?

METHOD

The Home Observation Assessment Method

The data to be presented come from a study in which 31 families that included one spouse who was alcoholic were observed in their homes on nine separate occasions over six months. Each family member signed an informed consent contract before data were collected. Each family was observed for six 4-hour time blocks (approximately 5 p.m. to 9 p.m.) during randomly selected weekday evenings and three 4-hour time blocks on weekend days (usually afternoon time blocks when the family was at home together).

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Data were collected using the Home Observation Assessment Method (HOAM), an instrument designed to allow two observers to make detailed observations of selected dimensions of interactional behavior over a 4-hour observation session (12). Each observer was assigned to either husband or wife and moved about the house as unobtrusively as possible. Although it sounds highly intrusive, the style of observation proved to be quite the opposite. Families quickly became accustomed to the observation process and felt that they behaved as they usually did when they were not being observed.

Coders recorded seven aspects of their subjects' behavior: 1) the subject's physical location in the home, 2) the identity of other people in the room with the subject, 3) the physical distance between the subject and others in the home when they were talking to each other, 4) basic interaction rates, both physical and verbal, 5) the content of selected verbal exchanges that involved decision making, 6) the affective level of the selected verbal exchanges coded, and 7) the outcome of the verbal exchanges coded.

Behavioral Indices and HOAM Factor Structure

After data had been collected, behavioral indices were created by applying two major questions to each of the seven dimensions of behavior coded: 1) How much of this behavior is occurring (its level of activity)? and 2) How variable is the behavior from observation to observation along this dimension (its variability)? Because we were interested primarily in family rather than individual properties, all indices were calculated as couple means. (Although the behavior of children is not separately coded in the HOAM system, the patterns of interaction between parents and children are accurately reflected in the couple data generated. It is therefore appropriate to think of these indices as family measures.)

In all, 25 indices of behavior were separately calculated for each of the families observed. Indices were approximately equally divided between activity measures and variability measures. The 25 indices were then subjected to a multivariate factor analysis using Varimax rotation (13). The most satisfactory factor structure yielded the five following factors:

1. Intrafamily engagement. Families scoring high on this factor interacted frequently with each other and at the same time tended to ignore the presence of the behavioral observers.

2. Distance regulation. This complex factor reflects a particular style of family interaction. Individuals in families scoring high on this factor came together in the same location only if they intended to talk to each other for some purpose. Although interaction occurred, family members did not appear to be comfortable with each other in the same location at home and maintained a considerable distance from each other. Families scoring low in this factor tended to "huddle" together in the home, rarely leaving each other's sight for independent projects.

3. Extrafamily engagement. Families scoring high on this factor were open to the presence of people who were not family members in the home and were more tolerant of individual differences in interactions with strangers.

4. Structural variability. Families scoring high on this factor demonstrated considerable variability of interactional behavior and physical movement from one coding session to another. Families scoring low on this factor most likely had a highly patterned and fixed style of interaction.

5. Content variability. Families scoring high on this factor manifested both high rates of decision-making behavior and variability of affect associated with verbal interaction. The direction of this variability indicates that such families had a relative tolerance for ambiguity, which allowed them to raise unsettled issues and to discuss them at length rather than quickly resolving them or making decisions as individuals without discussing them within the family.

Traditional Clinical Measures of Behavior

Traditional measures of behavior were obtained from three sources: 1) a self-administered symptom checklist, the SCL-90 (14), a measure of psychiatric symptomatology, 2) a self-administered alcoholism screening test, the SAAST (15), to assess the magnitude of alcoholism, and 3) a rating scale filled out by the observers reflecting their degree of comfort or discomfort in the family's home.

The SCL-90 is a widely used 90-item symptom checklist that develops index scores for degree of symptomatology along nine traditional psychiatric dimensions and combined ratings of "global health" reflected in the overall pattern of symptomatology. The instrument was administered individually to each spouse. Scores are based on sex-related normative data.

The SAAST is a 30-item instrument covering a wide range of physical and behavioral-social consequences of alcoholism as well as history of treatment contacts for alcoholism. Each family in our sample had one identified alcoholic who scored in the alcoholic range on this instrument. There was, however, a wide range in the magnitude of scores (low of 10 to high of 24). The SAAST scores, therefore, are an accurate reflection of the magnitude of the consequences of alcoholism experienced by the family.

Following the six-month data collection period, behavioral observers were asked to rate each family on a comfort-discomfort scale, a 10-point scale reflecting how comfortable or uncomfortable the family made the observer feel in the home. Observers demonstrated extremely high interrater reliability, and ratings reflected an even distribution of families along the scale.

FIGURE 1
Movements of Husband and Wife During an Evening In-Home Observation Session: A "Midrange" Family

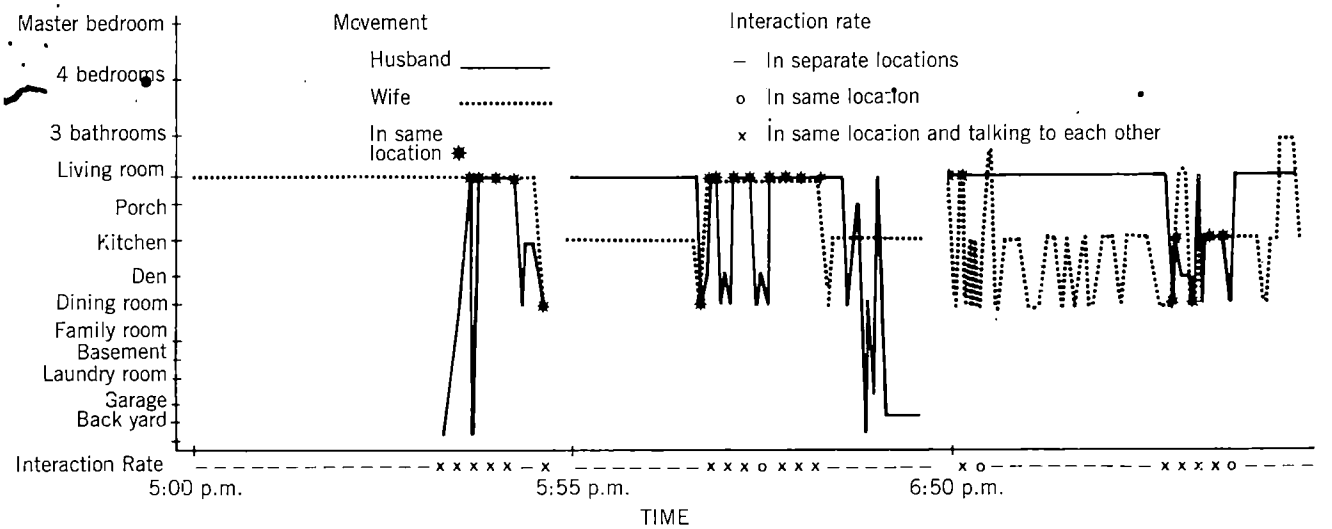
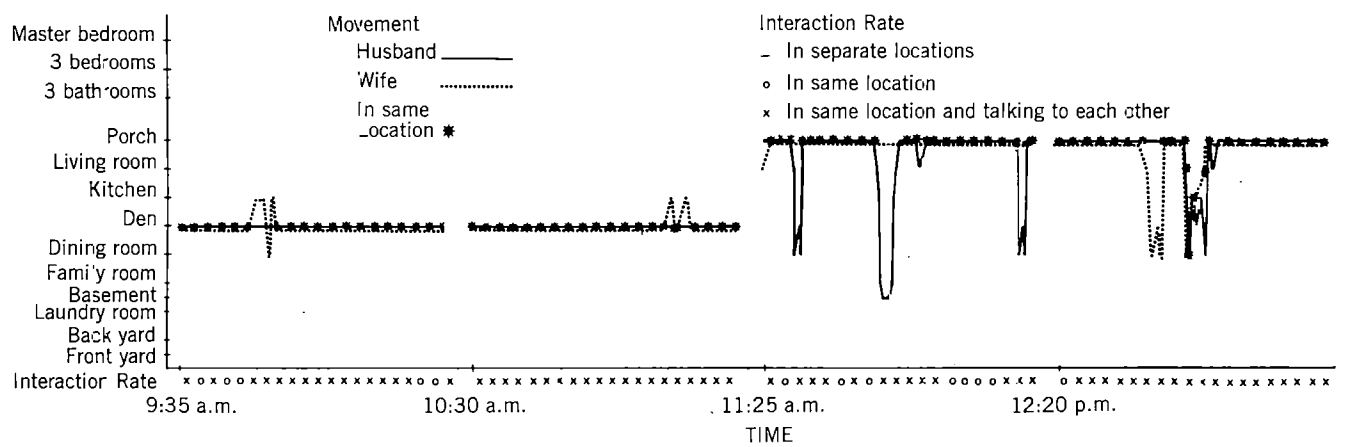


FIGURE 2
Movements of Husband and Wife During an Evening In-Home Observation Session: A "Huddling" Family



RESULTS

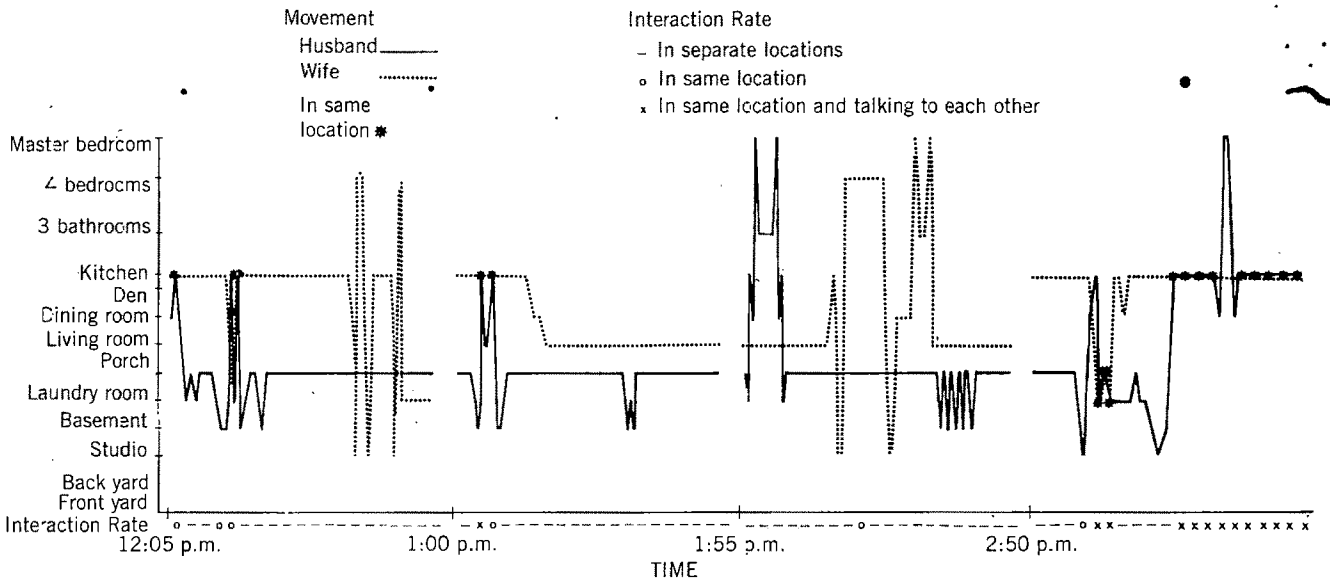
Differences Between Families in Home Behavior

The families studied, although all had one alcoholic member, varied dramatically in the behavior they exhibited in their homes. One of the aspects of behavior recorded, the subject's physical location in the home, illustrates these differences. Because the HOAM coding system records the subject's physical location sequentially as he or she moves from room to room, it is possible to reproduce the pattern of use of space during the observation session from the raw data recorded. If both husband and wife were charted on the same graph, the pattern of interaction between spouses was reflected in their relative synchrony of movement.

Figures 1-3 illustrate representative observation sessions for three very different families. The "midrange" couple (figure 1) had a mixed pattern of interaction in which blocks of time with consistent physical and verbal contact were interspersed with blocks of time in which each spouse was in a different part of the house. The "huddling" couple (figure 2), on the other hand, never seemed to leave each other's sight. The "distant" spouses (figure 3) operated like ships passing in the night. Husband and wife spent virtually all their time in separate rooms and had only minimal verbal contact during the 4 hours they were being observed.

Each of these patterns of interaction was consistent for the respective couple and would look strikingly similar for all nine observation sessions over the six-month period. It seems obvious that these families had adopted very different rules for managing their internal environments and that the concept of distance regulation is helpful in describing the family's use of time and space within the home (location shifts, the index illustrated in these figures, is one of the variables that loaded heavily on the factor of distance regulation).

FIGURE 3
Movements of Husband and Wife During an Evening In-Home Observation Session: A "Distant" Couple



Comparison of HOAM Dimensions and Traditional Measures of Behavior

Correlations comparing the five HOAM factors with SCL-90 scores reflecting overall level of symptomatology, SAAST scores indicating magnitude of alcoholism, and level of comfort or discomfort experienced by observers indicate that there were strong relationships between selected HOAM dimensions and each of the traditional measures. The correlations were performed using the Pearson product-moment correlation; significance levels were determined by two-tailed t test for 31 subjects. Two dimensions of interactional behavior, intrafamily engagement and content variability, were negatively correlated with the level of symptomatology reported by the alcoholic spouse in the family ($r=-.34$, $p=.06$, and $r=.33$, $p=.07$, respectively). All HOAM dimensions, however, were unrelated to level of symptomatology experienced by the nonalcoholic spouse. The magnitude of alcoholism, on the other hand, was negatively correlated with a different HOAM dimension, distance regulation ($r=-.68$, $p=.001$). Observer comfort was positively correlated with extrafamily engagement ($r=.41$, $p=.02$).

Further correlations comparing the five HOAM dimensions and the nine separate symptom subscales measured by the SCL-90 bring the profile of associations between home behavior and selected types of symptoms on the part of the alcoholic spouse into sharper focus (see table 1). These correlations suggest the existence of powerful associations between high depression and anxiety scores on the part of the alcoholic spouse with low intrafamily engagement and content variability. Content variability also appears to be negatively correlated with somatization, paranoid ideation, and psychoticism subscores. On the other hand, two subscales that one might expect to be related to patterns of family interaction, the interpersonal sensitivity and hostility subscales, appear unrelated to patterns of behavior as reflected in the HOAM dimensions. Also of interest is the absence of any significant associations between home behavior and level of symptomatology reported by the nonalcoholic spouse.

The SCL-90 score reflects the level of symptomatology experienced by members of the family while home observations were being made, but the SAAST score is based largely on retrospective judgments about the consequences of alcoholism on physical and social-behavioral well-being as well as the history of treatment contacts for alcoholism. Table 2 summarizes the correlations between HOAM dimensions and these three separate components of the SAAST score. One might have anticipated a connection between interactional style of the family at home and magnitude of social-behavioral consequences of alcoholism, but significant correlations were found with physical consequences and treatment contacts as well.

A series of correlations was also performed comparing demographic variables with each of the traditional measures (including separate correlations for each of the SCL-90 and SAAST subscales) to verify that the significant findings linking HOAM dimensions and these measures could not be attributed to a spurious demographic distribution in our sample. Previous analyses of HOAM data from this same sample of families (11) indicated that intrafamily engagement decreased as years of marriage increased. It was therefore particularly important to establish that these de-

TABLE 1
Correlations Between Home Observation Assessment Method (HOAM) Dimensions and Symptom Checklist (SCL-90) Dimensions[a]

SCL-90 Dimension	HOAM Dimension				
	Intrafamily Engagement	Distance Regulation	Extrafamily Engagement	Structural Variability	Content Variability
Alcoholic spouse					
Somatization	−.08	+.16	−.36[b]	−.06	−.28
Obsession-compulsion	−.24	+.23	−.26	+.01	−.25
Interpersonal sensitivity	−.24	+.21	−.22	+.05	−.21
Depression	−.47[c]	+.21	−.19	+.00	−.35[b]
Anxiety	−.37[b]	+.21	−.29	−.05	−.32
Hostility	−.27	+.17	−.27	−.04	−.19
Phobic anxiety	−.16	+.01	−.09	−.22	−.11
Paranoia	−.13	+.08	−.37[b]	−.09	−.34
Psychoticism	−.31	+.09	−.29	−.07	−.42[b]
Total	−.34	+.20	−.30	−.02	−.33
Nonalcoholic spouse					
Somatization	−.00	+.00	−.09	−.25	+.25
Obsession-compulsion	−.17	−.13	+.11	−.16	+.13
Interpersonal sensitivity	+.10	−.21	+.11	−.10	+.30
Depression	−.10	−.21	+.03	−.12	+.10
Anxiety	+.03	−.17	−.04	−.26	+.11
Hostility	+.14	−.09	−.01	−.25	+.12
Phobic anxiety	+.14	+.03	−.14	−.28	+.03
Paranoia	+.31	+.23	−.11	−.29	+.01
Psychoticism	−.02	−.06	+.08	−.15	+.05
Total	−.04	−.11	+.07	−.18	+.07

[a]All correlations were performed using Pearson product-moment correlation; significance levels were determined by two-tailed t test for 31 subjects.
[b]$p<.05$.
[c]$p<.01$.

TABLE 2
Correlations Between Home Observation Assessment Method (HOAM) Dimensions and Self-Administered Alcoholism Screening Test (SAAST) Dimensions[a]

SAAST Dimension	HOAM Dimension				
	Intrafamily Engagement	Distance Regulation	Extrafamily Engagement	Structural Variability	Content Variability
Physical consequences of alcoholism	+.13	−.50[b]	−.27	−.01	−.01
Social consequences of alcoholism	−.27	−.46[b]	−.14	−.09	+.22
History of treatment contacts for alcoholism	−.12	−.63[c]	−.14	−.08	+.14
Overall score	−.14	−.68[c]	−.15	−.01	+.13

[a]All correlations were performed using Pearson product-moment correlation; significance levels were determined by two-tailed t test for 31 subjects.
[b]$p<.01$.
[c]$p<.001$.

mographic variables (and others as well) were not also correlated with SCL-90, SAAST, or comfort/discomfort scores.

The demographic variables examined included ages of the husband and wife, their educational levels, Hollingshead-Redlich social class, years of marriage, size of the family, and size of the house (this last variable is an alternative measure of social status and is also important because this is a study using home observations). None of the correlations between these variables and the SCL-90 or comfort/discomfort scores reached even marginal levels of significance.

SAAST scores, especially for the social-behavioral subscale, were correlated with husband's age and social class. However, distance regulation, which was highly correlated with SAAST scores, was not correlated with any of the demographic variables tested.

DISCUSSION

I have presented two groups of findings in this paper. First, the families' styles of regulating their internal (home) environment could be formally measured by paying attention to concrete aspects of each family's use of time and space within the home. (The families in this sample differed widely in their regulatory style.) Second, there were striking associations between dimensions of home behavior and traditional clinical measures.

The concept of family internal environment is a familiar one in family theory. Jackson (16) used this concept in applying Cannon's idea of homeostasis to regulatory mechanisms exhibited by families. Handel (17) referred to the "psychosocial interior" of the family. Kantor and Lehr (18) proposed a model of family life

that stresses the importance of the family's internal versus external environment, proposed use of time and space as dimensions for delineating these environments, and used distance regulation as its central concept.

However, it has customarily been assumed that this aspect of family life is determined by a confluence of many factors, particularly such abstract phenomena as family themes, family boundaries (both internal and in relationship to its environment), and the family feeling-tone, and that it is consequently resistant to formal measurement. It is therefore encouraging to learn that one can observe families at home, count behaviors that relate to the family's use of space and time, and statistically identify specific regulatory dimensions. These dimensions, however, would be of only theoretical or descriptive interest if we were unable to demonstrate that the family's style of, for example, intrafamily engagement was associated with such clinical phenomena as the level of psychiatric symptomatology in one of its members. This latter connection assures us that the judgments we are making in our traditional clinical settings have predictive ramifications for family home behavior.

It is also of interest that the relationships between HOAM dimensions and traditional clinical measures were selective. For example, although distance regulation appeared exquisitely sensitive to magnitude of alcoholism in one member of the family, other aspects of behavior seemed to have been hardly affected at all (see table 2). These findings are actually more interesting than their alternative. If all aspects of behavior varied in relation to magnitude of psychopathology, it might suggest that family life at home was overwhelmed by the presence of a pathological individual in the family. Rather, the more selective findings are consistent with a hypothesis that the regulatory mechanisms for controlling each family's internal environment tended to be selectively adjusted to meet certain contingencies, such as how to cope with a chronic disease.

Significant correlations do not, of course, imply a cause and effect relationship between measures of pathology and home behavior or vice versa. For example, it seems equally feasible that a pattern of decreased intrafamily engagement would tend to "produce" symptoms of depression and anxiety in the alcoholic member of the family or that decreased family contact occurs in response to the impact of an anxious and depressed individual. (I am referring here to the significant negative correlation between intrafamily engagement and the SCL-90 depression and anxiety subscales.) Many family therapists, however, have argued that attempts to uncover such cause and effect relationships are less fruitful than a family systems approach, which concentrates on the reciprocal relationship between different aspects of behavior. The systems approach offers another explanation, namely, that as family styles of behavior develop they are both responsive to and influence the character of individual behavior. The significant correlations we found would be interpreted within this framework as evidence of a mutual and consistently defined relationship between the two dimensions being examined.

CONCLUSIONS

Examining these findings within the context of such a model I would conclude, first, that in families with a history of chronic alcoholism there is a strong relationship between the level of depression and anxiety in the identified patient and the family's pattern of intrafamily engagement and content variability. This means that there is a relationship between individual symptomatology and family patterns of home behavior. It also means that the family's style of behavior is related to individual characteristics of the identified patient; there was no correlation between HOAM factors and symptomatology in the nonalcoholic spouses. Parenthetically, the relationship between individual symptomatology and family behavior was specifically tied to depression and anxiety in the identified patient. It is important to note that separate analyses of correlations between HOAM dimensions and symptoms in the husband compared with the wife (a sex role analysis) indicated demonstrably lower correlation coefficients (in some instances the correlations were still significant because our sample had a male-female sex ratio of 3:1 for the identified alcoholic) than the alcohol role analysis. In other words, family behavior could be thought of as being organized around the chronic illness in such families (19). The direction of the correlations indicated that both intrafamily engagement and content variability decreased as symptoms increased, which seems clinically reasonable.

Second, there was a highly significant negative correlation between distance regulation and the magnitude of alcoholism experienced by the family as measured in terms of physical and social-behavioral consequences and treatment history. In other words, the greater the magnitude of alcoholism, the more the family tended to manifest a "huddling" rather than a "distant" pattern of interaction. This style of behavior might be a reaction to severe stress created by a rampant alcoholic condition, but it is also quite reasonable to hypothesize that such an interactional style tends to bring the alcoholic behavior more directly into the daily life of the family and in this sense makes it more susceptible to negative consequences. Most important, however, is the finding in this area of a relationship between a characteristic of a chronic psychopathological condition and the family style of regulating its home environment.

Third, the finding of a positive correlation between observer comfort/discomfort and extrafamily engage-

ment seems clinically reasonable. Insofar as extrafamily engagement reflects boundary permeability between the family and its external environment, observers could be expected to feel most comfortable in family situations in which they were allowed to enter the family's internal world. Surprisingly, however, such aspects of behavior as content variability and intrafamily engagement seemed largely immaterial in association with observer comfort or discomfort. Structural variability, the factor that most closely reflects a sense of order or patterning of behavior during times of observation, also did not seem to matter.

What the clinician typically hears about in the office or hospital setting are the unusual rather than the routine events in the life of a family. Crises or changes in routine, with their associated increase in uncertainty for the family, capture the family's attention and are passed on to the clinician as important data the family assumes are etiologically related to their dysphoric state. In this process, first the family and then the clinician tend to overlook aspects of behavior associated with daily routines that may play an instrumental role in the maintenance, if not the etiology, of chronic psychiatric illnesses such as alcoholism. Although our attempts to assess the family in its own home are still at an early stage, the initial findings presented in this paper suggest that the concept of a family internal environment, carefully regulated through specific behavioral mechanisms, may be a statistically discernible entity. Clinicians and behavioral observers alike can sample the flavor of this internal environment by taking a measure of the family's routines and by remaining sensitive to the quality of the boundaries established to protect its internal environment.

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The Misunderstood Spanish-Speaking Patient

BY FRANK KLINE, M.D., FRANK X. ACOSTA, PH.D., WILLIAM AUSTIN, M.D., AND RICHARD G. JOHNSON, JR.

The authors examined the reactions of Spanish-speaking patients and of therapists to initial therapy interviews conducted with or without an interpreter. Twenty-one patients who used an interpreter, 40 bilingual patients, and the 16 psychiatric residents who conducted the interviews completed questionnaires aimed at evaluating therapy and communication effectiveness. A number of patient and therapist perceptions were significantly different. Patients interviewed with interpreters felt understood and helped and wanted to return, whereas the therapists responded that patients seen with interpreters felt less understood and less helped and did not want to return. The authors recommend more bilingual/bicultural therapists and an effort to help present therapists realize how valuable their efforts with patients who need an interpreter are.

Our clinic is located in Los Angeles, in the midst of the nation's largest Mexican-American population. However, as in most mental health and public psychiatric outpatient clinics, the Spanish-surnamed are not proportionately represented among our patients and staff. In a 1975 survey of 354 consecutive new patients, only 56 (16%) were Spanish-surnamed. Nonetheless, there are more patients who want an initial interview in Spanish than our bilingual staff can interview so we use interpreters. We wondered how patients and therapists react to the presence of an interpreter so we collected, examined, and compared the therapists' and the patients' reported impressions of initial interviews conducted with and without an interpreter. We contrasted the patients in both of these Spanish-surnamed groups with patients previously surveyed in our clinic (1).

METHOD

All of the patients and therapists were volunteer subjects. Twenty-one of the patients were primarily Spanish-speaking. These Spanish-speaking patients requested and used an interpreter in their initial interview. Forty patients were fluent enough in English to not require the use of an interpreter; they neither requested nor used one. Thirty-two of the 40 further indicated that they were bilingual. Assignment of patients to evaluation with an interpreter or directly in English was thus determined by the patients' language fluency and request for an interpreter. All patients were randomly assigned to the therapists.

The therapists were 16 second-year (postgraduate year 3) psychiatric residents. Nine were Caucasian men, 2 were Caucasian women, 2 were black men, 2 were black women, and 1 was an Asian-American man. None of the 16 spoke Spanish.

Immediately after their initial intake interview, the patients were asked to voluntarily complete a 17-item questionnaire. Those who used an interpreter were given a Spanish version of the questionnaire. The questionnaire dealt with some of the following areas: patient's language use, response to clinic service, response to the therapist's service, patient's impression of what the therapist did, and whether the patient felt helped and understood.

At a later date the psychiatric residents who did the initial intake interviews were asked to complete a questionnaire. This questionnaire dealt with the therapist's feelings about interviewing patients directly in English versus through interpreters.

Some items in the patient questionnaire used in this study were identical to those in our previous study (1). This allowed comparison of some of the responses given by the patients in this study with those of 146 consecutive patients in the previous study. Comparisons were also made between the Spanish-surnamed patients in this study who used an interpreter and those who did not, and a further comparison was made between the responses given by the psychiatric residents and both groups of Spanish-surnamed patients.

The responses of the bilingual patients and the Span-

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TABLE 1
Percent of Spanish-Surnamed Patients Who Rated Clinic Services Positively After Initial Visit

Question[a]	Patients Who Did Not Use Translator (N=40)	Patients Who Used Translator (N=21)
Were the following helpful?		
Arranging appointment	93	95
Registration	78	86
Therapist interest	83	86
Therapist appearance	88	95
Therapist service generally	83	90
Medication	40	57
Understanding (self)	53	90
Specific directions (specific advice on my problem)	60	86
Did the doctor help you with your problem?	40	76
Even though your doctor couldn't speak Spanish, was the interview helpful?	40	76
Would you like to have continued to see the doctor for more visits?	73	76
Do you feel the doctor understood you?	65	76
Did you think you understood the doctor?	70	81
Would you like to have continued to see the doctor for more visits with a Spanish-speaking translator?		62
What did the doctor do?		
Advised me what to do	55	71
Gave me medication	40	52
Suggested I go to another clinic	8	10
Didn't know what to do	8	10
Mainly listened to me	50	90
Other	8	5

[a]To the first question, a positive response was indicated by the choice of "excellent" or "good" from "excellent," "good," "fair," and "poor." In the rest of the questions, a positive response was indicated by the choice of "very much" from "very much," "in general," "very little," and "not at all," except for the last question, which was answered by checking the appropriate items.

ish-speaking patients were compared by 2×2 chi-square tests. The responses of the residents and patients that were comparable were analyzed with 3×4 or 2×3 chi-square tests.

RESULTS

Patient Responses

Patients interviewed through interpreters said that they were generally better satisfied with the clinic service than were patients interviewed directly in English (see table 1). Those interviewed directly in English were generally as satisfied as our previously sampled group of 146 patients (1).

The Latino patients interviewed without interpreters were noticeably less pleased than the Spanish-speaking patients seen with interpreters with the help provided by self-understanding ($\chi^2=6.29$, df=1, $p<.025$) and, while not significantly so, were also much less pleased with the help provided by the doctor's specific advice ($\chi^2=3.20$, df=1, $p<.10$). Nearly twice as many patients interviewed through interpreters as patients interviewed without them said they were helped by the doctor in the initial interview ($\chi^2=7.12$, df=1, $p<.01$).

Based on the patients' reports (table 1), it appeared that both Spanish-surnamed groups found self-understanding and specific direction from the doctor more helpful than medication. Both groups felt that they had been understood and wanted to see the same doctor for more visits.

TABLE 2
Residents' Views on Clinic Services for Spanish-Speaking Patients

Statement	Residents (N=16) Who Agreed[a] N	%
An initial interview is satisfactory to me when the patient speaks English.	16	100
The patient appreciates the session more when we both speak English.	13	81
I think that patients see themselves as helped more when we both speak the same language.	16	100
I think that patients are eager to return for subsequent visits when interviewed via a translator.	5	31
I think that patients interviewed via a translator feel they communicate better than do English-speaking patients.	3	19
I think that patients feel they were better understood when they can talk to the therapist without a translator.	13	81
I feel comfortable seeing a patient in psychiatric treatment with the help of a translator beyond the initial interview.	1	6
I think I help non-English-speaking patients just as well with a translator as I do English-speaking patients with no translator.	0	0

[a]Residents who chose "agree strongly," "agree," or "agree slightly" from a list also including "disagree slightly," "disagree," and "disagree strongly."

Resident Responses

Table 2 shows that, in contrast, the residents thought that patients interviewed directly in English felt more appreciative, were more eager to return, and felt better understood. In addition, the residents unanimously stated that the initial interview in English was more comfortable and satisfactory to themselves and they thought it was more helpful to the patients.

Patient Versus Resident Responses

In order to make comparisons between the responses given by residents and those given by both groups of Spanish-surnamed patients, specific questionnaire items were compared. While the wording of the questionnaire items administered to the patients is not identical to that of the items administered to the residents, the commonality in meaning for the specific items compared is close enough to allow for com-

parisons. When we thus compared patient and therapist responses, several important facts emerged.

1. Eighty-one percent of the patients interviewed through interpreters thought they understood the doctor and 76% that the doctor understood them, while 70% of the patients interviewed without an interpreter thought they understood the doctor and 65% that the doctor understood them (table 1). In marked contrast, 81% of our residents thought that patients interviewed through translators felt less understood ($\chi^2=15.51$, df=6, $p<.025$) and that these patients felt they communicated less ($\chi^2=51.85$, df=6, $p<.001$).

2. Seventy-six percent of the patients interviewed through interpreters wanted to return for a second visit. In contrast, only 31% of the residents thought these patients were eager to return ($\chi^2=17.41$, df=3, $p<.005$), and only 6% were comfortable seeing them beyond the initial interview ($\chi^2=20.27$, df=3, $p<.001$).

3. None of the residents thought they helped patients interviewed through interpreters as much as they helped English-speaking patients. However, 76% of the patients interviewed through interpreters and 40% of the bilingual patients interviewed directly in English thought the interview was helpful ($\chi^2=15.32$, df=6, $p<.05$).

When the patient and resident responses were compared using the chi-square tests, the residents consistently underestimated the degree to which Spanish-surnamed patients felt understood and wished to return. They misunderstood the patients interviewed through interpreters more than those interviewed directly in English. The residents' misinterpretations seemed to be related to their discomfort with these patients, according to the second finding above.

DISCUSSION

Over 22 years ago, Saunders (2) pointed to negative effects of bias in the treatment of medical patients. The 1967 work of Yamamoto and associates (3) showed that psychiatric residents are less interested in patients from different cultures and races, and in 1974 Kline and associates (1) noted that unspoken therapist disinterest may cause patients to unilaterally leave therapy. Hart and Bassett's 1975 data (4) suggest that therapists and patients may not agree on which therapeutic modality is most helpful and that the psychiatrist may not be aware of the patient's preferences.

Patients may also stereotype therapists. Saunders (2) and Kline (5) stated that Spanish-American patients have negative stereotypes of Anglo-Americans. In contrast, Acosta and Sheehan (6) found that Mexican-Americans held more positive attitudes toward an Anglo-American professional therapist than toward an equally trained and experienced Mexican-American. Teplin's work (7) suggests Latinos may have more positive attitudes toward Anglos than toward Latinos.

Information on the treatment of Spanish-speaking patients with the help of interpreters is minimal. In a clinical report Marcos (8) pointed to potential language difficulties that may result when untrained interpreters assist therapists. MacKinnon and Michels (9) also noted potential difficulties in using an interpreter in a psychiatric interview but did recommend the help of an interpreter in working with non-English-speaking patients.

When this study was done in our outpatient clinic, our resident physicians generally underestimated the degree to which Spanish-speaking patients interviewed through interpreters felt helped. They also underestimated the patient's desire to return for a second visit. In addition, 15 out of 16 residents clearly indicated that they were not comfortable seeing these patients beyond the initial interview. Perhaps the residents' discomfort prevented them from recognizing the patient's desire to return and the patient's feelings that they were helped. Instead of being offered a return appointment, the patient was often referred to already overburdened bilingual therapists, given prescriptions, referred to another clinic, or discouraged from returning.

How can psychiatrists who take pride in accurately perceiving other people's feelings be so wrong? Is this only the result of our psychiatric residents' inexperience? Do such misjudgments occur only when therapists work through interpreters?

Undoubtedly, part of the difficulty is related to working through interpreters. A psychotherapist operates in a highly verbal field but also tries to deduce mental process by correlating speech, body movement, and facial expression. In an interview conducted through a translator, the patient speaks, the doctor watches body movement and facial expressions, and only later couples visual observations with speech. This may be like watching television without sound and then hearing the sound later. The lack of precision may make therapists unsure and uncomfortable. Also, working through an interpreter takes twice as long to gather and convey information and requires twice as many people to do the work.

Perhaps therapists' concerns about confidentiality and the desire to preserve a "pure transference" lead them to feel that patients resent the presence of a third person. Second-year residents may feel particularly unsure about the quality of their work and therefore be especially sensitive about someone observing them at work.

We think the therapist's uncertainty about his or her data base, general insecurity, and the double amount of time needed for the interview lead to feelings of doubt, helplessness, frustration, and loss of interest. The therapist then erroneously concludes that the patient does not feel helped or understood and is not interested in a second visit.

Obviously there are other possible explanations for

the therapist's discomfort and misperception. It may be, as other studies have suggested, that therapists are not comfortable with people from different cultures and different social classes and do not want to treat these people. Even though we try to help our residents become sensitive to minority patients' problems, they may be less aware of Latino culture and values. Racism or simple prejudice may be a factor. All three explanations may be partially correct. They do not preclude each other or even other explanations.

On the other hand, a few sample cases from our study did show that at least 2 patients who used a translator were seen for an average of 13 visits and showed therapeutic progress. We also have data that indicate patients interviewed through interpreters are more appreciative and feel better understood than patients interviewed in English. On the surface this seems like a violation of simple common sense. How could anyone possibly be better satisfied with an interview through an interpreter and in a foreign tongue? We suspect this occurs because most patients who do not speak English get cursory attention from English-speaking professionals. By contrast, in our study patients worked with both an interpreter and a psychiatrist and had the relatively undivided attention of both for almost an hour. In this context, it is easier to understand why these Spanish-speaking patients expressed more appreciation. A few drops of water in the desert are infinitely more valued than in a rain forest. There are other possible explanations. Latinos may, as blacks once did, prefer Anglos (7), or Latino culture may require greater compliance to authority or be more gracious and less openly critical. This list is neither complete nor exclusive.

What can we as mental health professionals do for patients who speak only Spanish? First and most obvious is that we need more bilingual and bicultural therapists. This would be helpful for direct service. It would save time and would provide positive role models for patients, staff, and community. In addition, bilingual and bicultural therapists bring to all patients and to all staff additional and sometimes unique understanding.

However, in our efforts to encourage the development of more bilingual/bicultural therapists, we must not overtly or covertly devalue, depreciate, or discourage our present therapists' efforts. We need to go even further and help beginning mental health professionals realize that they are at present not only the main, but sometimes only, resource for most Spanish-speaking patients and that their efforts may be more highly valued by patients who need an interpreter than by other clinic patients.

Overall, the most important lesson of this study is that mental health professionals may not understand what patients want or feel and may instead project their own wishes or discomfort onto patients. We need to continue to compare professionals' conclusions about what patients want and need with the patients' stated wishes.

We cannot escape the conviction that the therapists in our study did not understand that patients who requested interpreters wanted to return for a second visit, felt understood, and thought, or at least said, that their initial visit was helpful. This misjudgment seems to come not from crude anti-Hispanic prejudice but from the difficulty we all experience in bridging cultural and linguistic barriers.

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Effects of Divorce on the Visiting Father-Child Relationship

BY JUDITH S. WALLERSTEIN, PH.D., AND JOAN B. KELLY, PH.D.

Findings from a 5-year longitudinal study of 131 children from 60 divorcing families, drawn from a predominantly white, middle-class California population, revealed unexpected changes in the relationship between the visiting parent and the child after the marital separation. The changes that showed improvement and that showed deterioration in 50% of the father-child relationships were substantially related to the difficulties and psychological conflicts engendered by visitation and divorce, the father's capacity to accommodate to the constraints of the visiting relationship, and the age and sex of the child.

A 5-year longitudinal clinical study of 60 divorcing families with 131 children who were between 3 and 18 years old at the time of the marital separation has brought to light unexpected changes in the relationship between the visiting parent and the visited child that occurred in the immediate aftermath of the separation in a substantial number of families. We found no correlation 18 months after the separation between the predivorce father-child relationship and the frequency or regularity of the visiting patterns. This discontinuity reflected both improvement and deterioration in almost equal measure. Men who had been close to their children during the marriage and had spent every weekend with them in warm companionship failed to visit altogether or arrived infrequently and irregularly. Conversely, previously distant fathers, who had hardly acknowledged their children during the marriage or often appeared irritated by their presence, began to visit with a regularity that surprised the children and their mothers.

These observed changes were both age and sex related. Father-daughter relationships remained more stable over time; those which were poor during the marriage were more likely to remain so, and those which were warm and affectionate were more likely to continue unchanged. Father-son relationships were twice as likely to change as father-daughter relationships. Improved relationships were significantly more evident in children younger than 8 years old at the time of the marital rupture, whereas deterioration in the relationship was more evident in the 9- to 12-year-old age group. Sex differences were not linked to change among the adolescent youngsters. The influence of mothers' attitudes on visiting patterns appeared to be more evident when the woman encouraged or welcomed the visiting; however, the mothers' anger or opposition did not necessarily decrease the visiting frequency. In fact, the father's visiting was often fueled by the mother's opposition.

At the 18-month postseparation follow-up half of the father-child relationships were significantly changed from their dominant cast during the marriage: 25% were closer and 25% had unexpectedly and strikingly deteriorated.

These findings point to serious limitations in relying on the predivorce relationship between father and child (or mother and child) as a reliable predictor of the postdivorce relationship. Much legal testimony by psychiatrists and other mental health professionals in litigation over visitation or over custody is predicated on the assumption that the parent-child relationship forged during the marriage is likely to endure past the breakup. Moreover, clinical wisdom and psychological theory have supported the notion of general continuity in interaction between parent and child subject primarily to the developmental vicissitudes of each. Our data are at variance with these assumptions. Out of these and related findings we have come gradually to see influences that constrain and shape the visiting relationship which were not present during the marriage. We have come to recognize the visiting relationship as a singular relationship that has no counterpart in the intact marriage. The transition from a relationship within the intact family to one within an extremely different family structure—divorce—and the constraints of visitation increase the potential for change. These changes and the conditions which surround them require careful examination.

METHOD

Our population consisted of 60 families with 131 children living in a northern California suburban area.

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The families represented an ethnic and socioeconomic cross-section of the county in which they resided: 88% were white, 3% were black, and 8% were interracial marriages (Caucasian-Asian); most of the families were middle-class. According to the Hollingshead two-factor index, 23% were in Class I, 20% in Class II, 28% in Class III, 18% in Class IV, and 10% in Class V. The fathers were well educated: 27% held graduate degrees in medicine, law, or business administration. The average age at separation was 36.9 years for the men and 34.1 years for the women. They had been married an average of 11.1 years before final separation and averaged 2.2 children per family.

Of the 131 children in the study 48% were male and 52% were female. At the time they were first seen, 53% of the children were between 3 and 8 years of age and the remainder were between 9 and 18. All of the children were drawn from a nonclinical population, and none had been identified as needing psychological or educational intervention either by their parents or by school personnel.

Members of an interdisciplinary clinical team saw the children and parents individually in semistructured clinical interviews that averaged 14.2 per family, extended over a 6-week period, and took place within 6 months of the decisive parental separation. The interview data were supplemented by information obtained from the school; teachers and other school personnel were interviewed by a team member who had no access to the clinical interviews or the histories. The subjects were interviewed again individually by the same clinician within 1 year of the initial interviews and again 5 years after the separation. Of the 60 original participating families, 58 were reassessed at the 1- and 5-year follow-ups. Method and findings have been reported elsewhere (1–9).

We used clinical and statistical methods of handling our data. For purposes of clinical study, all the interviews were recorded in process note detail as a running narrative of the session and were supplemented by family drawings elicited from the children during the interview. We then summarized the interview material in accord with divorce-specific psychosocial profiles developed within the project. These profiles were sorted into small groups (according to the age and sex groupings of the children) and were inspected minutely, and items of interest that emerged were entered by hand on large charts so that similarities and differences along various dimensions of interest could be highlighted.

In addition to the clinical study, four data forms were developed to organize our same data for purposes of statistical analysis. Wherever possible, items for entry on these forms had been converted into 7-point scales; the array of items included clinical, developmental, environmental, and basic demographic variables. Parent data form I (initial assessment) comprised 290 items, and parent data form II (follow-up assessment) included 189 items. Similarly, child data form I (initial assessment) comprised 231 items, and child data form II (follow-up assessment), included 182 items. Statistical analysis included frequency distributions, intercorrelational matrices, and factor analyses. The children were grouped according to sex into two age groups: 3–8 years old and 9–18 years old. Analyses of variance indicated no significant differences in overall psychological functioning between the means of the primary age and sex groups in the correlation studies and the factor analyses.

RESULTS

Predivorce Parent-Child Relationship

In order to examine divorce-related change, we looked briefly at the parent-child relationship during the failed marriage and again at the changes that ensued. Our findings are at some variance with the commonly held view that parent-child relationships within a conflict-ridden marriage are likely to be troubled and conflicted as well. For a substantial number of the adults who were lonely and unhappy in many aspects of their lives, parenting appeared to be relatively conflict free. In fact, the most mature behavior a number of the couples seemed able to achieve revolved around their parent roles.

Of the 131 children, 36 (in 16 of the families) enjoyed the affection and close cooperative parenting of both parents, who seemed entirely able to set aside their disagreements in matters relating to the children. Approximately 50%, or 66, of the children had fathers who were adequate parents. Of these, 23 children, or approximately 20% of the total number, enjoyed exceptionally close relationships with the father. Similarly, 85 children, approximately 66% of the entire group, enjoyed adequate mothering. Of these, 43 children, or 33% of the entire group, were especially close to the mother. The fact that many of the children had a very close relationship with at least one parent was due in part to the marital unhappiness; both mothers and fathers sought out one or more children as companions, friends, or intimates during many hours of enforced loneliness. Several fathers took their children regularly on camping trips, sometimes every weekend. Such activities enabled parents to obtain relief from the presence of their spouses but also encouraged the development of close camaraderie with children who considered themselves very fortunate and were the envy of their friends.

At the other end of the spectrum 52 children, over 40% of the entire group, had relationships with their father during the marriage that were exceedingly poor, i.e., marked by gross psychopathology or chronic disinterest. Some of the fathers were continually and harshly critical in ways which were corrosive of the children's self-esteem. Others were physically abusive

or overtly seductive toward one or more children in the family. At least 33 children, 25% of the total group, had very poor relationships with their mothers, marked by emotional or physical neglect or threatened physical abuse. Socioeconomic factors were not significantly linked to psychopathological aspects of the mother-child or father-child relationship.

About 25% (N=33) of the children had witnessed chronic physical abuse between their parents. An additional 25% had seen physical violence between their parents at least once. Children also frequently heard and watched accusations and counteraccusations of promiscuity. In some families the violence and the accusations occurred only in the presence of the children. Again, neither the physical violence nor the sexual acting out was significantly related to the socioeconomic level of the family.

Response to the Father's Departure

Many children experienced the departure of the father from the household as a terrifying event. Even in homes marked by violence, the departure brought relief only to a small number. Only 10% of the children, including for the most part youngsters who were fearful for their own safety or the safety of the mother, were relieved with the divorce decision.

The younger children experienced the father's disappearance as an almost magical event and wondered where he had gone and who was feeding him or taking care of his sleeping arrangements. For these children the visit from the father and especially the visit to the father's residence provided needed assurance that he was alive and well. They took particular comfort in observing that the father had both a bed and a refrigerator. These early visits were immensely important in allaying the terror stirred by the father's sudden absence and the mostly unspoken expectation of half of the children that the remaining parent would probably disappear as well. Children well into latency also experienced these anxieties and expressed great confusion about where the departed parent had gone and who was caring for him.

Practical Problems of Visiting

From the outset the visiting relationship was beset by practical problems that seemed insurmountable to many fathers: the bewildering sense of the participants of no place to go and no idea of what to do, the combined presence of children of different ages and colliding interests, and the absence of the mother, who had often served as interpreter of the children's needs. These practical problems seem, at first, insurmountable to many fathers. What to do with young children needing nurturing care or with older children who appeared to need continual stimulation and entertainment to stem their restlessness. Some confused men changed their entire routines for the children's visits; others changed nothing; and others, equally perplexed, expected the children to take full responsibility for the agenda. "I feel like a camp director," said one exhausted father.

The sense of time as a component of the parent-child relationship also changed. For parents and children who share the repeated tasks of daily living together, time is part of the unobtrusive background of family life. For the visiting parent and child, time was a jarring presence; the constraints of time and space could impose a severe burden or, in some instances, a welcome limit on the interactions in the relationship. Because parent and child had to find time to meet and had to part on time, meeting and parting acquired new meanings and also accompanying anxieties that could have endured.

The broad and often unclear boundaries of the new visiting role heightened the anxiety of both parent and child. To what extent was the visiting father a guest, a favorite uncle, or a parent? More explicitly, to what extent did the visiting parent continue to take responsibility for setting behavioral and moral standards? To what extent was he to register approval or disapproval or enforce discipline or even homework—and how could he do so without the built-in safeguards and bedtime rituals of intact family life that mute disappointment, soften anger, and provide safe channels for alleviating the inevitable conflicts and frustrations of the relationship? Hurt feelings were more likely to continue, and misunderstandings were more likely to remain unclarified. Men who were used to having authority over their children—and many had held this role during the marriage—experienced a new sense of impotence and frustration. All of these complexities combined to keep the father's sense of who he was unsettled and unsettling.

Men were uneasy about what to do with their lovers at visiting time. They wondered how this would affect their children and the developing relationship with the woman. They did not know how to arrange for privacy in close quarters, and they often failed abysmally to understand or to be aware of the consequences of their own behavior or to anticipate the children's response. One father who customarily shared his waterbed with his young daughter was exasperated and mystified when she cried and screamed when an occasional girlfriend slept there instead of her. Some children lied because they feared the anxiety of the improvised sleeping arrangements, saying that they would prefer not to visit.

Visiting parents soon found out that the visit itself became an arena that readily evoked in both parents the conflicts of the failed marriage and the fantasies of what might have been, even without contact between former spouses or when the contact was minimal. Although the majority of parents tried to honor the children's visiting time, 20% of the women saw no use in the father's visits and actively tried to sabotage each meeting. This fighting between parents reached patho-

logical, even bizarre, intensity; one refined mother, for example, smeared dog feces on the face of her husband when he arrived to see his children. One-third of the children were consistently exposed to intense anger and stress at visiting time. Most of the visits ruined by parents involved older children; the younger preschool children were more protected. Two-thirds of the women reported that they were moderately or severely stressed by the fathers' visits during the first year of separation, as did 80% of the men.

Psychological Factors and Visitation

In addition to the external complications of visiting, subtle psychological forces had a profound influence on the nature and frequency of the visiting relationship and contributed to its fragility. The father's psychological conflicts and, more important, his feelings about the divorce were at stake. The father's role in the divorce decision and his eagerness for or opposition to ending the marriage were crucial influences on the future of the visiting relationship. Unlike what many children believe and adults conclude, the pattern of visiting after separation was not an accurate reflection of the father's love or interest. On the contrary, among the factors that contributed to making the visits difficult were the many psychological dilemmas of the father, which were not necessarily directly related to his feelings about his children.

Men who were depressed after the separation and the divorce decision found it painful to visit their children and often did so irregularly or not at all. Depression, which occurs frequently after separation, was a serious hazard to the establishment of a continuing relationship. Fathers who had been rejected by their wives often expected the same from their children. Preoccupied with their own shame, grief, lowered self-esteem, even their own expendability, such men found that visits with their children deepened their depression by bringing back painful memories of the marriage and its disruption. Some depressed men lacked the energy to mobilize themselves for the stresses and demands of the visits. Visits occasionally brought on a flare-up of somatic or other depressive symptoms, such as increased drinking, after the child returned home. Their children rarely understood this and sadly experienced the inconstancy as reflecting the father's lack of interest in seeing them. The following vignette illustrates this problem.

Mr. A had been a devoted, shy, and undemonstrative father to his young daughters. When his wife of 12 years decided on divorce, she asked him to leave the house and invited her lover to move in with her. Mr. A soon found it impossible to pick up his two daughters at the home he had built for the family he had lost. He could not bear to stand in the doorway and wait for the children; he was preoccupied with thoughts of another man in his place. His humiliation was compounded by the fact that the children seemed well and happy and not overwhelmed with longing for him. Although he loved his daughters, Mr. A did not visit them at all for 6 months after the separation. Neither his wife nor his children understood his conflict; they assumed he was not interested in seeing his children. His oldest daughter became depressed as she futilely waited for him to visit. Fortunately, counseling helped Mr. A to reinstitute regular contact with his children. His former wife, who had not wanted to keep him away from the children, readily agreed to bring them to a neutral place to meet him. Mr. A continued to visit frequently and regularly during the next 4 years.

Men who felt consciously or unconsciously guilty at having ended the marriage had great trouble in initiating and maintaining visits with their children. Some of these fathers had been devoted to their children; in fact, the father-child relationship had been especially important to them because their difficult marriages had brought father and child together to share many activities. The father's guilt was often profound at having left an affectionate parent-child relationship. Others felt guilty about leaving their children with a psychologically disturbed or otherwise incompetent mother or to pursue a different lifestyle or lover.

Guilt led either to a decline in visiting or to a guilt-ridden flurry of visiting immediately after the separation that was rarely sustained. When the father's guilt led to a decline in visits, this further reinforced the guilty feelings; it was not uncommon for fathers to project their self-blame onto the children and thereby justify discontinuing their visits. One such father referred, with irritation, to the "bourgeois values" of his 5-year-old, whom he had cared for tenderly during the marriage.

Children took on new psychological significance for the parents involved in a marital breakup. They became important sources of self-esteem whose reassurance was sought and whose absence was feared: "She is a mood elevator for me," said one father of his 7-year-old daughter. They were needed as friends and helpmates and even counselors during lonely, stressful decision-making times: "He understands everything I say before I say it," Mrs. B said of her 6-year-old son. "Yesterday he advised me to remarry." They were needed as prizes to be fought over; they became the only reliable allies in the marital battle. The complex dependence of adults on children for their self-esteem was especially apparent as fathers became vulnerable to their children in new ways that sometimes radically changed the balance of the relationship. Approximately 25% of the men feared rejection by their children. They were worried about the children's disapproval and anger because of the divorce. These fears, coupled with their own guilt and insecurity in the new visiting role, led many men to take on the Santa Claus role, which they found emotionally burdensome and economically draining. Nevertheless, they persisted in buying presents, which the children did not need, hoping to win the children's affection and allay their own anxieties. Over 33% of both parents were in active

competition with each other for the affection and loyalty of the children and resented the child's turning to the other parent. All of these factors threatened the fragile visiting relationship and generated stress.

Factors That Fostered Visiting

All of the problems with visiting were counterbalanced by the passionate, open yearning of the children, especially those younger than age 9, by the commitment of many fathers to their children, and by the interest of about half of the women in maintaining the visits. Other factors also fostered visiting. Fathers whose children expressed great pleasure in the visiting visited more. Fathers whose children were not angry at them over the marital breakup visited more. In effect, fathers who were reinforced by the children's pleasure visited more. Often the younger children provided this reinforcement in full measure and were therefore visited more than older children, who were more difficult to please or to placate. The concern of men for children who had been left in the custody of depressed or psychologically troubled women led some to visit more. Fathers who were lonely, psychologically intact, and not depressed visited more. Fathers who were economically secure and better educated visited somewhat more, and fathers in families where the relationship between the parents had calmed down and was no longer marked by intense animosity tended to visit more, although the mother's attitude toward the visiting was a less influential factor than we expected.

DISCUSSION

The visiting relationship that successfully outlived the marriage reflected not only the relationship of the predivorce family or the father's and child's motivation to maintain their relationship but the psychological capacity of fathers, mothers, and children to adapt flexibly to the new conditions of the visiting relationship. Men who could bend to the complex logistics of the visiting, who could deal with the anger of the women and the capriciousness of their children without withdrawing, who could involve the children in their planning, who could compromise between totally rearranging their schedules and not changing their schedules at all, and who could overcome their own feelings of rejection or guilt at the time of the divorce were predominant among those fathers who continued regularly and frequently to visit.

Our observations regarding the fragility of the visiting relationship at the time of the marital separation take on special meaning in light of our previous finding that the relationship between children and their fathers 5 years after separation was significantly linked to outcome in both boys and girls (9). We have reported that children who were visited infrequently because of the father's lack of interest or rejection during the years after the divorce were likely to suffer severely diminished self-esteem (9). The most stressed children were those whose relationship with their father had been close and affectionate during the marriage and who experienced a disruption in this relationship after the divorce. Children found this sudden disruption incomprehensible and remained unable to assimilate the loss and the intense hurt of the rejection during the 5 years that followed (9). There is evidence in our work that the relationship between visiting father and child is most open to intervention immediately after the father has moved out of the household (9). Because the continued relationship emerged as a significant factor in good outcome, these findings suggest the father-child relationship in the immediate postseparation period as a specific target for preventive intervention. Strengthening the father-child relationship at this crucial time and helping the parents and the children to master the new and complex demands of the visiting relationship could facilitate improved postdivorce interactions.

After a marital separation, both parents and children, without rehearsal or available role models, need to adapt their feelings and mutual needs in the relationship to the narrow confines of a visit. We have conceptualized this process as a funneling that requires complex, and sometimes exquisite, maneuvers from the several participants. The difficulties inherent in this compressive process have been insufficiently appreciated. Unfortunately, the courts and the embattled partners and their attorneys have focused on imposing restrictions and strict conditions that further encumber a relationship that, even under the best circumstances, needs encouragement. Divorcing families could benefit considerably from guidance during this difficult transition, which could help them create new or maintain close and affectionate bonds between the visiting parent and the child.

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Prompt Publication Policy

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Evaluation and Modification of Exercise Patterns in the Natural Environment

BY KELLY D. BROWNELL, PH.D.; ALBERT J. STUNKARD, M.D., AND JANET MICHELLE ALBAUM

Using a new experimental paradigm to evaluate physical activity in the natural environment, the authors made 45,694 observations of persons using stairs or an adjacent escalator at a shopping mall, train station, and bus terminal. In study 1, stair use more than doubled for both obese and nonobese persons during two 2-week periods when a colorful sign encouraging use of the stairs was positioned at the stairs/escalator choice point. In study 2, stair use remained elevated for 15 consecutive days while the sign was present, decreased during a 1-month follow-up period, and returned to baseline by 3 months. These results not only demonstrate the usefulness of this paradigm, but also suggest the strength of simple, inexpensive public health interventions to increase physical activity.

The physical activity of members of affluent societies has decreased markedly during the past century. The extent of this decrease is suggested by a two-fold increase in the prevalence of obesity in the United States since 1900, despite a 10% decrease in daily caloric consumption (1, 2). This decline in activity is noteworthy because inactivity is associated with a number of medical and psychological disorders (3).

Physical inactivity is strongly related to obesity and coronary heart disease. Obese persons are less active than their thin counterparts (4–7). Inactivity can be either a cause or consequence of obesity, but it is known that exercise decreases body fat in obese humans and animals (8–10). Coronary heart disease is more common among sedentary persons than among physically active ones (11–16). Furthermore, physical activity may protect individuals against coronary heart disease (13, 14, 17), and persons with coronary disease may benefit from physical training (18–20).

The physiological effects of exercise have been studied under experimental conditions, but little is known about patterns of physical activity in the natural environment. Such information is essential before attempts can be made to modify these patterns. We present here an experimental paradigm for the study of physical activity in the natural environment and the results of its use in two studies.

In many public places stairs and escalators are adjacent, giving people an opportunity to choose one or the other. Evaluation of the characteristics of persons making this choice provides an unobtrusive measure of physical activity in natural settings. This method, which was used in a recent study (21), showed that obese persons used stairs less than did nonobese persons.

The present report describes the measurement of the relative frequency of use of stairs and escalators to assess the impact of an intervention designed to increase activity. The first study assessed the effects of intermittent use of the intervention, and the second study evaluated long-term maintenance of changes produced by more prolonged use.

STUDY 1

Method

Subjects. A total of 21,091 observations were made of persons going up stairs or escalators in three public places where stairs and escalators were adjacent. The sex, race (black/white), age (over 30/under 30), and weight (obese/nonobese) of each subject were recorded. Subjects were considered over age 30 only if judged to be clearly over and were considered obese only if judged to be clearly so. Persons carrying baggage or items larger than an attaché case were excluded, as were children.

Setting. Observations were made during February and March in three locations: a shopping mall, train station, and bus terminal. Observations were made on the same day of the week during the same hours: 11:30

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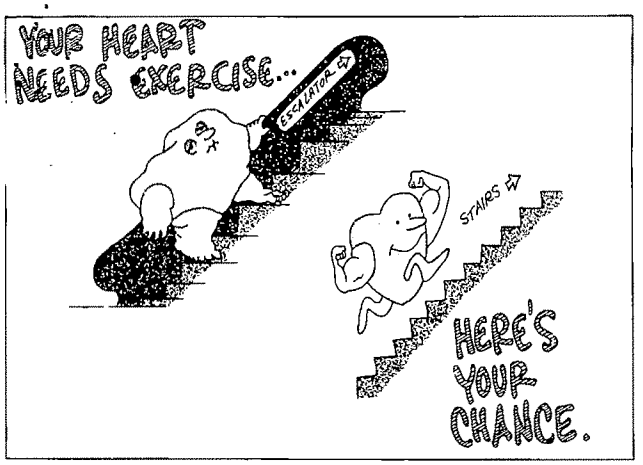
Address reprint requests to Dr. Brownell, Department of Psychiatry, University of Pennsylvania School of Medicine, 205 Piersol Building, Philadelphia, Pa. 19104.

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FIGURE 1
Sign Employed to Encourage Use of Stairs



a.m. to 1:00 p.m. for the shopping mall, 7:30 a.m. to 9:30 a.m. for the train station, and 3:30 p.m. to 5:30 p.m. for the bus terminal. There were 30 steps at the shopping mall, 18 at the train station, and 24 at the bus terminal.

Procedure. Observations were made by six undergraduate students and the authors. At each observation site, four observers were located inconspicuously at the foot of the stairs and escalator. One observer each recorded data for men on the escalator, women on the escalator, and persons on the stairs. The fourth recorded data with each of the first three in turn to estimate reliability of the measures. Reliability data were collected for each category (sex, race, age, and weight) for 14.7% of the sample (3,105 subjects). Reliability coefficients were calculated by dividing the number of agreements by the number of observations. There was agreement on 97% of the observations for age, 99% for race, 99% for weight, and 100% for sex.

Experimental conditions. Observations at each site were made once a week for 8 weeks. In an ABAB experimental design (22), the first two observations constituted a baseline phase (A), the next two were observations during the intervention phase (B), and the sequence was then repeated once. During the baseline phase, naturally occurring activity patterns were observed. The intervention consisted of placing a sign at the stairs/escalator choice point. The sign measured three feet by three-and-one-half feet and encouraged subjects to use the stairs (see figure 1).

The percentage of subjects using the stairs did not differ between the two baseline phases or between the two intervention phases, so the two like phases were combined for analysis. In order to examine the effect of several factors simultaneously, we calculated the proportion of stair users for all (2^5=32) combinations of levels of the five factors (sex, race, age, weight, and intervention). Angular transformation was then used to transform the proportions to approximately normally distributed variables, permitting a weighted analysis of variance (23). The effect of each variable was then determined after adjusting for the influence of other factors.

Results

Figure 2 shows that the sign markedly increased the percentage of subjects using the stairs. The percentage of subjects using the stairs increased from the first baseline to the first intervention phase (5.3% to 13.7%), then decreased when the sign was removed (7.1%) and increased again when the sign was reinstituted (15%). An average of 6.3% of the subjects used the stairs during the two baseline phases and 14.4% during the two phases when the sign was used ($F(1,64)=346.1$, $p<.0001$). We found that the sign had similar effects across all sites and across all categories of subjects. The analysis of variance revealed several interesting effects of the site and category of subject.

Effects of site. There were highly significant differences in stair use among the three locations ($F(2,64)=38.7$, $p<.0001$). Stair use during baseline was significantly higher at the shopping mall than at the train station or the bus terminal ($\chi^2=9.16$, $p<.002$); stair use at the latter two sites did not differ. The intervention produced far greater increases in stair use at the shopping mall (7.2% to 17.8%) than at the train station (5.6% to 12.7%) or the bus terminal (5.9% to 10.6%) ($F(1,64)=15.5$, $p<.0002$), and greater changes at the train station than at the bus terminal ($F(1,64)=4.97$, $p<.03$).

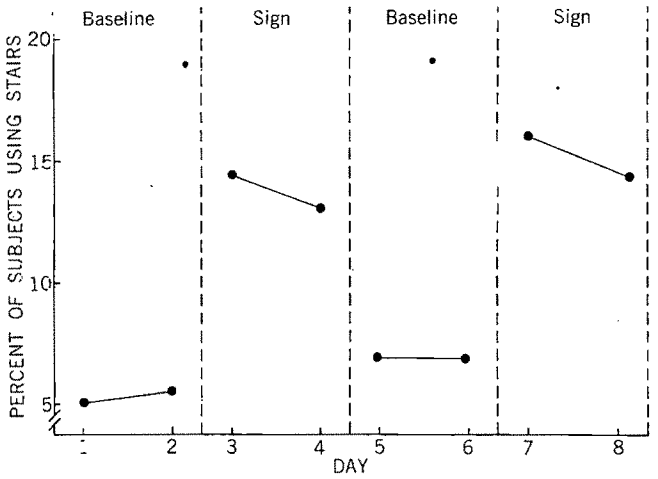
Characteristics of subjects. During baseline only 1.5% of the obese subjects used the stairs, compared with 6.7% of the nonobese subjects ($p<.0001$). The sign produced a highly significant increase in stair use for both groups (7.8% and 14.9%, respectively; $p<.0001$ in both cases), but the nonobese subjects continued to show a significantly higher level of use ($p<.0001$). The intervention was equally effective for obese and nonobese persons.

During baseline, significantly more men (7.4%) than women (4.9%) used the stairs ($p<.0001$), and the sign produced a highly significant increase for both groups (15.7% and 13%, respectively; $p<.0001$ in both cases). The differences between men and women remained significant ($p<.0001$), and the intervention was equally effective for both groups.

Although stair use by blacks (6.8%) and whites (6.1%) did not differ significantly during baseline, significantly more whites (16%) than blacks (10.4%) were influenced by the sign to use the stairs ($\chi^2=53.6$, $p<.0001$). The race × intervention interaction was highly significant ($F(1,64)=29.3$, $p<.0001$).

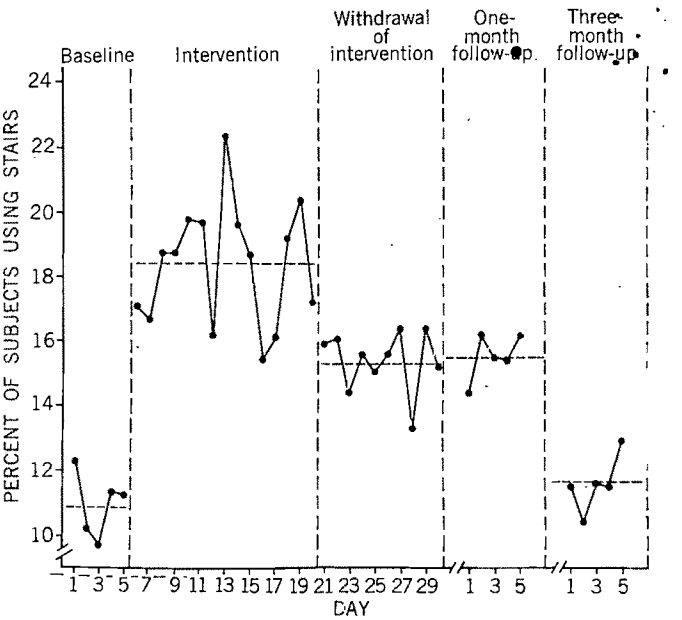
Stair use during baseline was greater for persons under 30 (8.7%) than for those over 30 (3.9%) ($\chi^2=102.4$, $p<.0001$). The percentages for subjects under 30 (18.4%) and over 30 (10.2%) also differed during the intervention phase ($\chi^2=145.8$, $p<.0001$). The intervention × age interaction was not significant, in-

FIGURE 2
Percent of Study 1 Subjects Using Stairs at All Three Sites During Baseline and Intervention Phases



dicating that both age groups responded similarly to the sign.

FIGURE 3
Percent of Study 2 Subjects Using Stairs Before, During, and After Intervention



STUDY 2

Study 2 was designed to address two issues raised by study 1. First, repeated exposure to the intervention was evaluated (in study 1 subjects were observed after a single exposure to the intervention). Second, the long-term maintenance of changes in activity was measured by observing the same group of people over time.

Method

Subjects, setting, and procedure. A total of 24,603 observations were made using the same procedures described in study 1. Observations were made at a commuter train station in the business district in downtown Philadelphia from 7:30 a.m. to 9:00 a.m. on consecutive days, excluding weekends, from May through August. Since the observations were made before commercial businesses opened, there were few tourists in this part of the city; and because the same trains entered the station during the observation hours, the likelihood that the same people were observed each day was maximized. There were 18 stairs at this site. Reliability data were collected for 12.5% of the sample (3,065 subjects). There was agreement on 95% of the observations for age, 99% for race, 99% for weight, and 99% for sex.

Experimental conditions. An ABA experimental design was used with two follow-up assessment periods. During the initial baseline phase (A), observations of stair/escalator use were made for 5 consecutive days. In the subsequent intervention phase (B), the sign used in study 1 was displayed for 15 consecutive days. A 10-day withdrawal-of-intervention phase (A) followed, during which the sign was not displayed. Two 1-week follow-up phases were then completed, during which the sign was not displayed; the first took place 1 month and the second, 3 months, after the last day of intervention. Statistical analyses were identical to those used in study 1.

Results

Figure 3 shows that the percentage of subjects using the stairs increased significantly from the baseline to the 3-week intervention phase (11.6% to 18.3%) ($F(1,11)=175.4$, $p<.0001$). The percentage of subjects using the stairs was 15.5% during the 2-week withdrawal-of-intervention phase, 15.6% at the 1-month follow-up, and 11.9% at the 3-month follow-up. An analysis of variance revealed that stair use varied significantly across phases ($F(4,11)=78.6$, $p<.0001$), but that there was no significant variation in stair use within each phase.

To evaluate stair use across phases, the Studentized range test was used (23). The .05 level was used, and the test revealed three distinct groups: the intervention phase, the withdrawal-of-intervention and 1-month follow-up phases, and the baseline and 3-month follow-up phases. No significant differences were found within each of these three groups. The intervention phase featured significantly more stair use than the withdrawal-of-intervention and 1-month follow-up phases, and these phases in turn had significantly more stair use than the baseline and 3-month follow-up

TABLE 1
Percent of Subjects Using the Stairs Before, During, and After Intervention[a]

Characteristic	Baseline			Intervention			Withdrawal of Intervention			One-Month Follow-Up			Three-Month Follow-Up			Overall		
	%	χ^2	p	%	χ^2	p	%	χ^2	p	%	χ^2	p	%	χ^2	p	%	χ^2	p
Age																		
Under 30	13.3	6.3	.01	19.0	1.7	.19	17.0	6.3	.01	17.2	3.3	.07	14.2	6.8	.009	17.0	20.5	.0001
Over 30	10.4			17.9			14.5			14.7			10.6			14.9		
Sex																		
Male	14.7	27.8	.0001	20.1	18.6	.0001	17.1	9.5	.002	18.4	17.5	.0001	14.6	15.6	.0001	17.9	77.7	.0001
Female	8.8			16.7			14.2			12.8			9.5			13.8		
Race																		
White	11.7	.8	.38	18.5	1.9	.168	15.9	7.3	.007	16.0	5.5	.02	12.1	1.3	.25	16.0	14.0	.0001
Black	9.8			16.5			10.9			10.1			9.1			12.8		
Weight																		
Nonobese	11.8	3.3	.07	18.8	39.3	.0001	16.0	20.8	.0001	16.0	8.3	.004	12.3	7.3	.005	16.1	83.0	.0001
Obese	6.6			4.8			4.2			6.2			2.8			4.9		

[a]The χ^2 and p values designate differences between groups within an experimental phase.

phases. The intervention significantly increased stair use. Much of the sign's effect remained when the sign was removed and persisted as long as 1 month later. Only after a 3-month follow-up did stair use decline to baseline levels. The sign's effect did not diminish during the 3 weeks it was displayed.

Characteristics of subjects. Chi-square tests were used to determine differences between subgroups within each phase (see table 1). Obese persons used the stairs significantly less than nonobese persons during each experimental phase. Stair use by obese persons remained fairly stable across all phases, and nonobese persons showed significantly greater response to the sign than did obese persons ($F(1,40)=7.67$, $p<.008$). Over all phases, stair use by obese persons (4.9%) was less than use by nonobese persons (16.1%) ($F(1,82)=91.2$, $p<.0001$).

Men were significantly more likely to choose the stairs than were women during each experimental phase. Over all phases, 17.9% of the men and 13.8% of the women used the stairs ($F(1,82)=98.1$, $p<.0001$). Both groups displayed significant increases in response to the sign, but after it was removed the women's stair use returned to preintervention levels more rapidly than did the men's ($F(4,82)=2.8$, $p<.03$).

In all phases combined, 16% of the white subjects and 12.8% of the black subjects chose the stairs ($F(1,82)=18.7$, $p<.0001$). Whites and blacks responded equally to the sign and increased stair use significantly. There was a trend, although not statistically significant, for blacks to return to preintervention levels of stair use more rapidly than whites.

Subjects under age 30 used the stairs significantly more than did subjects over age 30 during all phases except the intervention phase. The age × phase interaction was not significant, indicating that the two age groups did not differ in response to the intervention, withdrawal of the intervention, or the long-term absence of the intervention.

DISCUSSION

These studies demonstrate the utility of a simple paradigm for the study of physical activity in the natural environment (21). Stair versus escalator use is an instructive measure because 1) the activity is common to many people; 2) large numbers of subjects can be studied, thus allowing comparisons of subgroups according to sex, weight, age, and so forth; 3) in certain locations the long-term behavior of individuals can be evaluated; 4) the behavior appears to be sensitive to interventions designed to change it; and 5) perhaps most importantly, the behavior can be observed in the natural environment.

The study of behavior in the natural environment provides a valuable supplement to laboratory studies with their artificial and reactive quality. The results of studies of eating behavior in natural settings have often differed from those of studies conducted in the laboratory, and vigorous efforts are being made to develop experimental paradigms for studies of eating in natural settings (24, 25). Similar efforts are needed for the study of physical activity.

The most striking finding of these studies is the remarkable propensity for physical inactivity, as measured by stair use in the presence of an escalator. During baseline observations, only 6.3% of the people used the stairs in study 1, and only 11.6% used the stairs in study 2. This inactivity occurred even though the stairs were often more accessible than the escalators; people frequently waited in a crowd before gaining access to the escalator.

Although the purpose of these studies was to develop a paradigm for the study of physical activity, the finding that the sign increased stair use suggests a new means for increasing activity. Despite the apparent virtues of exercise, relatively few persons engage in long-term programs of vigorous physical activity. Vigorous programs are useful for those who are able and

willing to undertake such a regimen, but many people, the obese for example, will not enter or complete a strenuous exercise program. Of those who initiate a program, 30% to 50% terminate it prematurely and even more fail to adhere to the prescribed regimen (26, 27). For this reason, behavioral programs for weight control emphasize modest changes in routine activities such as walking and using stairs. Making these modest changes in the daily routine holds the promise of increasing *adherence* to activity programs. One study, for example, found that adherence and reported satisfaction were greater for persons prescribed a program of walking than for those prescribed a more vigorous regimen (26).

Using stairs produces a surprisingly large expenditure of energy, even larger than that for such strenuous activities as jogging, swimming, cycling, and calisthenics (28). The caloric expense of substituting walking up and down two flights of stairs each day for using an elevator or escalator amounts to 2.7 kg for an average-weight (80-kg) man over one year. Modest increases in physical activity have been viewed favorably in weight reduction programs because activity may influence appetite as well as energy expenditure. Mayer and Thomas (29) demonstrated that increased physical activity in sedentary Sprague-Dawley rats not only increased caloric expenditure but actually decreased caloric intake at low levels of activity. Similar and probably more powerful effects have been reported in the Danforth yellow mouse (30), the dietary obese rat (31), other animal species (32–34), and even young children (35).

Routine activity such as walking and stair climbing may also have cardiovascular benefits; tests of these programs are being carried out. In a study of civil service workers in Britain, Morris and associates (36) found that regular stair use was one of the few activities associated with reduced risk of coronary heart disease. Ballantyne and associates (26) found reductions in plasma lipoproteins after a walking regimen, and several studies (37, 38) have shown increased cardiac efficiency in response to walking programs.

The results from both studies show that a simple sign can increase use of the stairs and that the maintenance of these changes is a critical issue. Recent studies (39–41) have shown that there is limited maintenance of even large behavioral changes in programs for reduction of risk factors, such as those for obesity, smoking, and hyperlipidemia. In study 2, increased stair use persisted with no decline for the 15 days the sign was in place. After the sign was removed, stair use decreased promptly but remained significantly higher than baseline levels for at least 1 month. Stair use finally declined to baseline levels by the three-month follow-up. Given the minimal intervention, it is surprising that these changes in activity were maintained this long.

The percentage of persons choosing the stairs in these studies (6.3% and 11.6%) was remarkably similar to that in an earlier study of stair/escalator choice (6.1%) (21). In the present studies, nonobese persons used the stairs more than obese persons, men more than women, and persons under 30 more than persons over 30. Meyers and associates (21) also found that obese persons are relatively inactive, but found no differences in activity according to age. Stair use in response to the sign increased significantly among men, women, blacks, whites, persons over and under age 30, and nonobese persons. In study 1 there was a significant increase for obese persons, whereas in study 2 stair use for obese persons remained stable throughout. The explanation for this difference is not apparent.

There were several interesting differences among the subject subgroups regarding the response to the sign and the maintenance of changes. In study 1 whites were more responsive to the sign than blacks, but both groups were equally responsive in study 2. However, there was a trend for stair use to decline more rapidly for blacks than for whites once the sign was removed. Similarly, men and women showed equal response to the sign in both studies, but the decline in stair use after removal of the sign was more rapid for men than for women. These findings are puzzling and suggest that various subgroups of the population might respond differently to efforts to change health behaviors.

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The Role of Humor and Folklore Themes in Psychotherapy

BY JULIUS E. HEUSCHER, M.D.

In the alternation of deconstitution and constitution that is fundamental to human life, humor has a useful role, mainly as a deconstituting force, whereas folklore emphasizes the revitalization of existence. Yet humor can be harmful in situations where there is a lack of mutual respect, and it can be growth-retarding if it becomes a substitute for needed change. The effectiveness of humor and folklore themes is proportional to the genuineness of the interpersonal relationship. In folklore, humor reinforces the injunction to seek wider worlds beyond the one that has become monotonous and stifling. Humor and folklore themes can, therefore, prove helpful when used judiciously in psychotherapy.

> Remember laughter! You'll need it even in the blessed isles of Ever After.
>
> —James Thurber (1)

This paper explores the idea that humor and folklore lift us beyond the ordinary, blindly accepted limits of the everyday world and can therefore have important therapeutic implications.

The numerous and often overlapping interpretations of the meaning and function of humor include pleasurable, vicarious release of suppressed emotions or instincts; satisfaction in a one-upmanship game; and joy in mastery, in sudden discovery, or in being defeated by a good punch line (2–4). I wish to emphasize one important and apparently universal feature of humor—especially of jokes—that is implicit in most other interpretations. This is a sudden change in or widening of the recipient's experiential horizon.

This formulation might be viewed as a variant of the observation that jokes permit the release of unconscious wishes or impulses. However, it encompasses more. A change in an individual's experiential horizon may well uncover repressed or otherwise taboo data. However, it also reveals a wealth of feelings, ideas, and attitudes that are not yet, or are no longer, part of the individual's current reality. The "state of unreality" created by the humorous situation (4, 5) is then the vision of a different "world design."

The recipient's world design (i.e., the way he has structured his existence) is suddenly, albeit temporarily, modified as a result of the punch line of a joke. This world design is a crucial aspect of an individual's identity, and considerable anxiety may be aroused when it is suddenly altered in other than humorous situations. What is it that causes mirth rather than fright when a joke is told?

Although there are good reasons for equating a person's world design with his identity, genuine human relationships that are intersubjective (rather than technical, manipulative, or interobjective) are equally important to one's identity. Such relationships are nonexploitative and characterized by unconditional respect and tolerance. It is primarily when assured of his identity by such a relationship that a human being is willing to explore new and wider world designs and has less need to cling to his habitual one.

We find some of the qualities of intersubjective relationships in those social gatherings that produce various forms of enjoyable humor. Mutual respect, permissiveness, warmth, and absence of exploitation create an atmosphere in which jokes are appreciated. In turn, jokes invite intimacy (4).

Some of these observations about humor are also applicable to folklore. In fact, various forms of humor enhance the effectiveness of fairy tales (6). Folklore revolts against everyday reality inasmuch as it always turns to some extent against the established and rigid religious, political, social, or moral order, thereby opening new sources of vitality (7). Whether Beowulf defeats Grendel so that life can return to Herot, or whether the fairy-tale hero overcomes the holdfast king, the established order is always overthrown, expanded, modified, or revitalized. Like humor, folklore aims at the widening of overly rigid world designs, but it portrays the widening as a lasting achievement rather than a transitory glimpse.[1]

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[1]In addition to humor and folklore, there are other human manifestations that involve modifications of the individual's world design including play (8), dreams, art, philosophic insights, and psychotic and neurotic symptoms.

THERAPEUTIC IMPLICATIONS

Humor is an initial uncomplicated attempt at revealing new and wider horizons and can therefore further the growth of the person who is able to integrate some of the new vistas in his world design. However, humor can also be used easily to avoid change when a joking individual pretends to be open to wider options but rapidly reverts to his habitual, narrow world design, thus deceiving himself and others. At worst, joking in psychotherapy is a "playing at change" that allows therapist and patient to retain their uneasy security in their unchanging individual existences. At best, it is a helpful revelation of new options. It is the quality of the therapeutic relationship that determines whether humor becomes a holdfast or a growth-enhancing force and whether humor is a defense or involves meaningful suggestions that become integrated in the patient's lifestyle.

This is also the case with folklore. When humor or a folklore theme is introduced in psychotherapy, it is important that the therapist underline its seriousness by rephrasing it and pointing out its pertinence to the patient's current manner of being in the world. A therapist would not allow a dream to be sidestepped with the remark that it is "only a dream," and he must not allow a joke, a reference to a myth, or the mention of a fairy tale to be so dismissed.

There are no rules for determining how, when, and where humorous or folklore themes should be pointed out or inserted in the psychotherapeutic situation. In fact, the more systematically the use of such themes is planned, the less effective it is likely to be. Their proper use depends primarily on the therapist's sensitive appreciation of the current relationship's quality, the patient's receptivity to such themes, and the relevance of the tale or witticism. The therapist must also have keen insight into inauthentic or defensive factors that prompt him to focus on a particular anecdote or to make a humorous remark. Such factors range from attempts at warding off anxiety-producing situations to veiled manipulations and inordinate needs to be liked and admired. Kubie (9) cautioned against the use of humor in psychotherapy and listed 16 potentially destructive effects. Similarly, Reik (10) pointed out how jokes are used to hide problems or conflicts.

On the other hand, Levine (4) stressed the rebellious quality of humor that represents a voluntary withdrawal from "reality" in contrast to involuntary, maladaptive, pathologic withdrawals such as neuroses and psychoses. He saw humor as an intrinsic and essential component of the normal growth process and stressed its use in psychotherapy as a means to facilitate the awareness of profound insights. Hershkowitz (11) saw the use of humor in psychotherapy as a way of presenting ambiguities that are acceptable, even sought after, and that may serve to make the patient tolerate a world that is not always clear-cut. Grossman (12) mentioned that a patient's favorite joke often provides insight into otherwise obscure areas of conflict. Finally, a patient may be more willing to tell his favorite jokes than to remember dreams, and, unlike dreams, jokes and folklore themes are at the disposal of both members of the treatment dyad.

CLINICAL EXAMPLES

I will present clinical examples of the occurrence and uses of humor and folklore in therapy. For the sake of clarity I will separate what the patient brings forth from what the therapist introduces during the sessions. In practice, however, this separation is rarely clear-cut, as the effectiveness of humor or folklore emerges only in the dialogue between therapist and patient.

Humor and Folklore Mentioned by Patients

Humor expressed in waking life. Meaningful and humorous was the statement by a patient suffering from impotence that "The body is nasty; it's just something you carry around dangling below your brains!" In another instance, an exceedingly bright and anxious young patient was first able to express his ambivalent feelings toward the therapist by asking him whether he had personalized license plates. When the psychiatrist looked puzzled, the patient remarked casually, "I saw your car in front of your office and wondered whether the BBS on the license plate stood for 'Big Bullshitter'!"

Humor expressed in dreams. Not infrequently patients recognize puns in their dreams. A very obese lady who was forever deciding to start dieting the next day dreamed of receiving a phone call from a stranger who insisted he was "Tom Morrow." Exasperated, she woke up and started to laugh as she recognized her pun.

Folklore related to waking life. Patients often recall their favorite fairy tales or the epic heroes or heroines they once identified with. A very lonely patient longing for warmth and affection mentioned his frequent childhood fantasy of being the little boy loved by Hans Christian Andersen's Snow Queen. The distortion of the actual tale proved quite significant: the patient had managed to turn the icy, rationalistic queen into a warm, caring lady.

Folklore in dreams. At times patients describe dreams or daydreams whose themes are strikingly analogous to those of well-known folklore. A young man terrified of women dreamed of traveling around like Perseus, carrying a large mirror and feeling safer when looking only at the reflection of pretty girls. This led us to discuss Sartre's belief that the participants in an interpersonal relationship tend to reduce each other to objects or things (the Medusa complex).

Humor and Folklore Introduced by the Therapist

Humor in everyday situations. A therapist must be empathetic and keenly aware of his feelings toward the patient when he decides whether the introduction of a particular humorous theme is appropriate. For example, a middle-aged, eternally frustrated woman related that she was given a $35 raise when she had not expected more than $10; she immediately proceeded to enumerate reasons why this raise still represented a painful slight. I responded by telling the story of the boy who lost a quarter but was given another by a passing, compassionate gentleman. The boy started to cry again after a few seconds, and the man turned back, asking why he was still unhappy, even though he had received another quarter. The boy whimpered, "If I'd not lost my first quarter, I'd now have half a dollar!" Laughing, the patient was able to acknowledge her affinity with the youngster's attitude toward life.

Humor in dreams. The therapist may point out puns and humorous situations in a patient's dreams that he or she had not been aware of. An ex-nun longed desperately for male affection and support. She dreamed she was in a large church where she stumbled down a stairway and fell into the arms of an ex-priest whose name was Armstrong. She did not immediately recognize the pun related to her wish to be taken into the arms of a strong man.

An unhappily married woman kept assuring me that she had never hated anyone. However, she was bothered by a recurring dream in which she was preparing a breakfast tray for her tyrannical husband. When she finally placed the salt shaker on the tray, the label always read, to her great surprise, "arsenic." Only gradually was she able to acknowledge the dream's macabre humor.

I once commented to a very prudish woman that her dream of "teaching insects" might mean that she wished for "teaching in sex." As if to chide me, but again totally unaware of the pun, she reported at the next session a dream that centered on a "talking duck" ("talking 'doc' ").

Folklore in everyday life. Fairy tales were used to interpret everyday life situations by prophets, poets, and philosophers long before Freud emphasized the Oedipus myths, before Jung recognized in them archetypal images, and before transactional analysts began to view them as scripts. The choice of themes is great, and there are numerous ways of using them in psychotherapy. On several occasions I illustrated the hidden purpose of a patient's self-denial with Odysseus' famous ruse in the cave of Polyphemus, whom he told that his name was "Nobody." As you may remember, Odysseus escaped from the cave and was saved because the blinded giant could not get his giant friends to help him when he told them "Nobody" had blinded him.

The dread of success being followed by tragedy is portrayed in the tale of Polycrates, the prosperous ruler of Samos. As Polycrates' good fortune increases, his friend becomes more uncomfortable and eventually flees from the island, convinced that the gods will punish the unbounded success of a mortal. A young engineer to whom I told this saga was deeply moved and began to recall that when he was younger his parents often yelled at him and told him he was worthless whenever he began to feel happy and successful.

Folklore in dreams. Numerous occasions have been reported in which folklore promoted a deeper understanding of dreams (13). A man with many sexual problems reported to me recently a dream in which he experienced considerable confusion in regard to the correct anatomical placement of his genitals. I was reminded of an episode in the Winnebago trickster cycle where Wakdjunkaga carries his penis and scrotum in a bag on his back, learning only gradually their proper placement. The story helped the patient in recognizing that our body image is not simply given; it results from our choices or responses in the face of various experiences originating within ourselves and in our early environment.

DISCUSSION

The examples I have given do not include the numerous humorous implications of verbal and nonverbal communications in psychotherapy. These can be captured only by videotapes. It is important that the therapist be as aware of them as possible. In addition, there are many hybrids of humorous and seemingly paradoxical responses. Other treatment modalities, such as psychodrama, role playing, and gestalt therapy, are marginally related to humorous approaches. These modalities also bring into focus, and allow the questioning of, one-sided rigid presuppositions. An analogous function is performed, on a collective level, by the pranks of tricksters in folklore and by ritual clowning. Tricksters and clowns are allowed to break most social or tribal taboos, which are thereby placed into perspective and become amenable to criticism or modification. Even if these taboos are not modified appreciably in the long run, they appear less narrow and stifling when viewed against a previously ignored, variegated background.

Focusing on the background accomplishes, especially in wit, a reversal that can be likened to the well-known picture of two profiles facing each other that turns into a vase by reversal of figure and ground. An old joke illustrates this well: A schoolteacher intends to demonstrate the evil effect of alcohol on living things by placing one worm in a glass filled with water and the second in a glass filled with vodka. The second worm shrivels up and dies immediately, but the first keeps on swimming around happily. When the class is

asked about the moral of the experiment, Johnny eagerly raises his hand and declares, "If you drink enough vodka, you won't get worms!"

The state of unreality created by the humorous situation or by certain folklore themes can be viewed as a new potential reality. By the adoption of the humorous attitude or by identifying with the folklore protagonist we are free to think of doing outrageous things, to experience outrageous feelings, to contemplate outrageous actions, and to enjoy them without guilt (4). We thus gain a wider perspective of our everyday social and personal reality.

Humor, ritual clowning, role playing, and folklore make us more critically aware of habitual lifestyles by setting them off against others. By placing before us alternate, different, and wider worlds beyond our habitual world-designs, they reveal the presuppositions upon which our existence is built, or the scripts and hidden commitments by which we live. They may prove beneficial by encouraging the incorporation of some of these optional ways of being in the world into our everyday life. However, they often prove beneficial even when they do not lead to an observable change in our everyday lifestyle. Increased awareness that our individual or cultural world is always a restricted, chosen one and recognition of the fact that we could choose otherwise can promote a meaningful, new, freer recommitment to our present world. Sometimes psychotherapy focuses too much on change, growth, and exploration of potentials, overlooking that for some individuals the deliberate, free commitment to a very limited range of interests may be quite authentic.

When voicing an idea wittily, or telling a joke, or responding to a patient's humor, a therapist must be genuine and capable of savoring the comical. At the same time, he must remain alert to the opportunities that humor offers for recognizing and evaluating unacknowledged feelings, unexpressed thoughts, and neglected options. Each opportunity can be explored in depth to enable the patient to reconsider the feasibility of feelings, thoughts, and options veiled and revealed by the humor. Otherwise, humor easily becomes a means of skirting important issues. Such skirting is also seen when patients smile or laugh at their weaknesses and predicaments, and focusing on its defensive features may prove helpful.

Similar considerations hold for the effectiveness of folklore themes mentioned in the course of treatment. It is desirable that the therapist have at his disposal a wide repertoire and a broad understanding of the hidden, multifaceted meanings of these stories.

The examples presented earlier were chosen for their simplicity and their usefulness in illustrating a few basic points rather than for their wittiness, their symbolic depth, or their therapeutic effectiveness. All therapists, wittingly or unwittingly, use wit in psychotherapy, and most will occasionally introduce a folklore theme, even if it is only the Oedipus myth. In my own work with patients, the use of humor or folklore is never preplanned. With some patients I rarely find the occasion to introduce or interpret such themes. The themes usually arise spontaneously within my own or my patients' consciousness. The only task, then, is to decide whether, in what form, and at what point during the psychotherapeutic interactions they can be used propitiously.

CONCLUSIONS

"Remember laughter!" is Golux's parting remark in Thurber's delightful fairy tale (1). The smiling Buddha, the jesting Zen master, the tricked trickster, and Don Juan in Castaneda's novels all manifest their authenticity by various qualities of laughter. Without this basic potential, without humor, all other forms of transcendence become unreliable. Dreams, philosophy, art, psychotherapy, and folklore need not always include humor, inasmuch as they are more complex, enduring, and committed endeavors. Yet the dreamer, the bard, the philosopher, the artist, or the therapist who is incapable of laughter and wit is bound to be ineffective in affirming, expanding, deepening, and enriching his world or that of his fellow man.

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EDITORIAL

Charismatic Religious Experience and Large-Group Psychology

The recent upsurge of charismatic religious sects in the United States and Western Europe has attracted considerable public attention. Religious revitalization movements in the Arab world and Southeast Asia have even assumed political significance. The psychological phenomena inherent in these groups raise important issues for psychiatry. We will comment on them after characterizing these groups.

Charismatic large-groups typically have more than a score of members (usually many more) and are characterized by the following dynamics: 1) a consensual belief system, 2) highly cohesive membership, and 3) extensive influence on members' behavior and lifestyles. On these counts, they contrast in size and character with the small social group, such as that used in traditional psychotherapy. Little consideration has been given to large-group dynamics in the psychiatric literature, although some is afforded in relation to group therapy training, as developed by Rice (1).

As social structures, charismatic large-groups are relatively short-lived, but they may go through a process of institutionalization after some years, as described by Max Weber. They are frequently led by a charismatic figure to whom preternatural powers may be imputed. Some associated phenomena may be unsettling, such as the sudden induction to seemingly alien beliefs, mass experiences of hallucinatory phenomena, grossly deviant behaviors (even self-destructive, as in Jonestown), and abrupt abandonment of antecedent social ties. Many such groups have been chartered as established religions and are protected by constitutional safeguards. While these groups may employ age-old approaches, they challenge our attempts to understand them within modern psychological theory.

Who joins modern charismatic sects? Some observers have found that members are generally young adults from middle- and upper-middle-socioeconomic class families, sometimes with unhappy family relationships (2). Galanter's paper, "Psychological Induction into the Large-Group," provides information systematically collected during recruits' induction into one large sect, the "Moonies." Those who ultimately joined the groups scored considerably lower than age- and sex-matched control subjects for psychological well-being and had weaker interpersonal ties outside the sect than those workshop members who chose not to join. Such sects address the dual problems of alienation and unhappiness among their potential recruits, providing a belief system that is rapidly accepted by inductees.

After a period of membership, however, elaborate psychological defenses may be necessary to sustain members' relationships to the group and its leader. Deutsch's paper, "Tenacity of Attachment to a Cult Leader," provides a rare glimpse, through psychiatric eyes, of the mechanisms used by one cult's members to abide by a disturbed leader. They demonstrate the fragility of everyday rationality in the face of the forces inherent in a charismatic large-group.

Shaver studied a large national sample of adult women with a mean age of 35—more than a decade older than typical recruits to the charismatic sects. In "Reli-

giousness, Conversion, and Subjective Well-Being: The 'Healthy-Minded' Religion of Modern American Women" he provides systematic data on the childhood experiences of religious converts (primarily to traditional denominations) and those who had maintained the same religious affiliation all their lives. The converts reported being less happy, less religious, and more lonely in childhood than did women who had not undergone conversion. Studies such as this one, using national samples, may be necessary to appreciate the extent of religious conversion within the American population. Among Shaver's sample, 17% currently belonged to a religion to which they had converted "out of deep conviction."

Conversion experiences guide us to useful insights regarding psychiatric nosology. Transcendental experiences and visions have been reported among "normal" individuals undergoing ecstasy and meditation. William James wrote a classic description of such events, which also occur in the absence of group suggestion. Similar experiences involving modern sect members have been documented under better controlled conditions (3). It is of interest that many of these transient, self-limited experiences conform to standard definitions for the first-rank symptoms of schizophrenia. The occurrence of psychotic-like phenomena in the religious or meditative context raises questions about possible biosocial aspects of these symptoms in psychopathological conditions. Have we come to underestimate the role of cognitive and social induction in eliciting and modulating psychosis?

Therapeutic influences of large-group experiences also merit attention. Affiliation may lead to an improvement in affective state, thereby providing a basis for reorganization of behavior patterns. The influence of informal social controls (as contrasted with formal legal or police action) within the subculture is important for the diminution of behaviorally related pathology. Diminished drug and alcohol use among members of modern charismatic sects (3, 4) illustrates the potency of such behavioral influences—all the more impressive in light of the therapeutic failures in altering patterns of drug use.

Equally impressive is the role of religious conversion in altering characterological adaptation. Pattison and Pattison, in their article " 'Ex-Gays': Religiously Mediated Change in Homosexuals," report on 11 men who changed from exclusive homosexuality to exclusive heterosexuality after experiencing religious conversion. The transformations occurred without explicit religious instructions, since none had revealed his homosexuality to his religious group. This again illustrates the power of informal social controls within reference groups.

Personal change in the large-group setting occurs in a variety of therapeutic contexts not usually identified as religious. Alcoholics Anonymous, professionally directed programs, such as Soteria House for the treatment of schizophrenia (5), and certain drug-treatment therapeutic communities employ similar methods. Mass media have facilitated the rapid spread of ideologically oriented self-help groups, such as women's consciousness-raising groups and est. Since these groups are sometimes therapeutic in their effects, might they represent a cost-effective alternative to traditional psychotherapy in certain cases? Like religious groups, however, these self-help activities usually entail an ideological engagement and strong, cohesive ties—commitments that many choose not to make. Ethical issues and long-term side effects from ideological conversion are also important considerations.

Rapidly growing, highly cohesive sects that greatly affect their devotees' lives tend to appear in contexts of rapid social change, disjunction between traditional symbols and new values and attitudes, and social oppression of disenfranchised individuals without a group identity (6, 7). Hierarchical relationships and the loci of power may change dramatically in such situations, either in people's perceptions or in fact. It is to be expected, then, that political activities will frequently coincide with cult movements, varying from nonviolent crusades to violent rebellions against the established order.

How might the social and psychological effects of groups be used by the professional mental health community? Social readjustment among patients with chron-

ic or recurrent disorders is often limited by disillusionment and alienation. Rehabilitation can be hastened by peer-oriented groups, such as Recovery, Inc., Alcoholics Anonymous, and Pills Anonymous. Few office practitioners have maximally integrated these peer-led groups with traditional therapies. Bridges need to be built and specific techniques developed for effecting a complementary rather than a competitive relationship between professional treatment and self-help groups.

If tomorrow's professionals are to work with such groups, conceptual models must be integrated into a workable theoretical framework. We are seeing the development of models for small-group and family therapy that complement the psychodynamic hypotheses of individual psychology.

Certain relevant concepts for large-groups are at an early stage. Lofland and Stark (8) have proposed a model for religious conversion based on predisposing conditions and situational contingencies. Developed from observations in a small millenarian cult, their model may be useful in understanding broader aspects of affiliation to charismatic groups. One study (9) has suggested the presence in large-groups of a sociobiologic "relief effect," which consists of increasing relief from neurotic distress as affiliation with the large-group intensifies.

Reports from nonclinical settings may also contribute to our comprehension of group psychology. Lifton (10) has studied the forced "conversion" (or "thought reform") of Korean War prisoners to the belief system of the Chinese Communist state. He emphasized the role of establishing strict control over all communications during this process. His observations apply to the induction techniques that certain charismatic sects practice. They are also relevant to certain therapeutic contexts, such as induction into some therapeutic communities.

In summary, the articles in this issue's special section illustrate striking psychological effects associated with both induction into and membership in the charismatic group. They also suggest roles for self-help activities and religious conversion vis-à-vis mental health. Further, they point to the value of developing models for these group-psychological processes based on empirical observation.

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SPECIAL SECTION: Modern Religious Experience and Psychiatry

"Ex-Gays": Religiously Mediated Change in Homosexuals

BY E. MANSELL PATTISON, M.D., AND MYRNA LOY PATTISON

The authors evaluated 11 white men who claimed to have changed sexual orientation from exclusive homosexuality to exclusive heterosexuality through participation in a pentecostal church fellowship. Religious ideology and a religious community offered the subjects a "folk therapy" experience that was paramount in producing their change. On the average their self-identification as homosexual occurred at age 11, their change to heterosexual identification occurred at age 23, and their period of heterosexual identification at the time of this study was 4 years. The authors report 8 men became emotionally detached from homosexual identity in both behavior and intrapsychic process; 3 men were functionally heterosexual with some evidence of neurotic conflict. On the Kinsey 7-point sexual orientation scale all subjects manifested major before-after changes. Corollary evidence suggests that the phenomenon of substantiated change in sexual orientation without explicit treatment and/or long-term psychotherapy may be much more common than previously thought.

The possibility that homosexual orientation can be changed, or may change over time, is in vigorous debate. The gay-liberation movement asserts that any fundamental change is rarely possible (1). Most behavioral therapists and psychotherapists conclude that changes in sexual behavior may be attained or anxiety and conflict about homosexuality can be reduced, but they, too, doubt that basic change in sexual orientation is generally feasible. Some psychotherapists suggest that in highly motivated persons a basic change can be accomplished but only through long-term intensive psychotherapy. The general tenor of professional opinion is given by West (2), who stated, "Permanent eradication of all homosexual inclinations appears to be rather rare."

Occasional autobiographical reports have described a spontaneous change in sexual orientation through salutary life experience, which usually includes religious conversion (3–5), and there are individual case reports of spontaneous change without psychotherapy (2, 6, 7). However, such reports seem to be the exception.

Recently we came in contact with a religious hotline crisis program sponsored by a pentecostal church that offers lay crisis services to homosexuals. This program claimed to involve a number of persons who were "ex-gay," that is, these people had experienced a basic change in sexual orientation from exclusive homosexuality to exclusive heterosexuality. The term "ex-gay" had been deliberately selected by them to denote an ideological position in opposition to "gay liberation." This change was said to have occurred within the context of the church fellowship and without any psychotherapy or other professional intervention. The program seemed to offer an "experiment of nature" by which we could investigate this apparently spontaneous change from homosexuality.

METHOD

We obtained written permission from the church program to review all their crisis program records and

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to conduct personal interviews with their clients. The clientele for a 5-year period (N=300) yielded 30 cases claimed to have changed from homosexuality to heterosexuality. These 30 cases were reviewed in detail with the program staff, who knew all the subjects. This produced a set of corollary data that confirmed the subjects' claims that they had changed in sexual orientation. Of these 30 cases we were able to obtain the cooperation of 11 men and to collect personal interview data on them. For the 6 subjects who were married, we also obtained corollary data from their wives. We found no substantial discrepancies in the data we collected from the staff, from interviews, or from spouses.

Each subject signed a written consent form, which explained the purpose of the interview. Although the subjects knew we were investigating their changes in sexual orientation, we do not believe that this substantially biased the data; they were forthright about the personal details of their past and present life and did not attempt to distort their life experience.

Each interview, which was structured and detailed, included the following items: current mental status; developmental history; past and present sexual history; sexual fantasies, dreams, and impulses; social relations; homosexual and heterosexual experience; heterosexual marital experience; and details of alleged change in sexual orientation. All subjects were cooperative in every detail of the interview format, even though much of the subject material related to issues of considerable shame, guilt, and anguish to the subjects.

RESULTS

Individual and group data will be presented. However, to place the meaning of the data in context, we shall juxtapose our data with comparable data from two important recent large sample studies of homosexuals in the community: the 1973 study by Saghir and Robins (8) and the 1978 study by Bell and Weinberg (9).

The Saghir and Robins study sample was drawn from volunteer recruits from the Chicago and San Francisco homosexual communities; it was comprised of "healthy homosexuals" who were "essentially hard core confirmed homosexuals who have made some adjustment to their homosexuality and the world at large." The sample was composed of white men who were socially functional and of high occupational status; their age range was 19–70 years and their mean age was 35 years. The total sample size of white homosexual men was 89. The Bell and Weinberg study sample was drawn from volunteer recruits from the San Francisco homosexual community. Age stratification revealed that the ages of these men ranged from under 20 years to over 70 years; the men's mean age was 37 years. Their social status and economic status were variable. The sample size was 575, and all of the men were white. Throughout this paper when we refer to the studies conducted by these two teams of investigators, we will be referring specifically to these studies of homosexuals.

Sample Characteristics

Our sample consisted of 11 white men between the ages of 21 and 35 years (mean age=27 years). All had at least 13 years of education; 10 had a graduate education. All subjects were in white-collar technical/business or professional occupations. This comports relatively well with the higher education and occupational status of the homosexual samples in both the Saghir and Robins and the Bell and Weinberg studies but in their samples about 50% of the respondents were more than 35 years of age.

As shown in table 1, all our subjects experienced defined homosexual proclivities before age 15. This compares well with the data of Saghir and Robins, who reported that 90% of their sample had an identified homosexual orientation before age 15 and 82% had engaged in homosexual activities by that age. All of our subjects declared that they had considered themselves exclusively homosexual, although 3 (27%) at some time considered themselves bisexual. This is much lower than the 59% bisexual self-identification reported by Bell and Weinberg. During the time of exclusive homosexuality, all our subjects had engaged in active homosexual experiences.

The age at which a change from homosexuality to explicit heterosexuality occurred ranged from 19 to 29 years (mean=23 years). The majority, 7 men (63%), changed at or before they were 22 years old. Similarly, Saghir and Robins reported that in their sample the period of greatest fluidity and change of homosexual identity and orientation was between the ages of 18 and 22 years. Our subjects had been heterosexual from 1 to 7 years (mean=4 years). There were no comparable data in the other two studies.

Kinsey Scale

The most widely used scale for assessing degrees of heterosexuality/homosexuality is the original Kinsey scale (10). Although this is a gross measure dependent on rater judgment the scale represents the continuum of degree of psychic inclination and behavior toward either direction. A rating of 0 is defined as the subject having no physical contact with the same sex producing erotic arousal, no psychic erotic arousal to the same sex, and sociosexual contact exclusively with the opposite sex. A rating of 1 involves only incidental physical or psychic response to the same sex. A rating of 2 includes definite same-sex response but strong and predominant reaction to the opposite sex. A 3 rating is midway. A rating of 4 is a predominant response to the same sex, with definable heterosexual

TABLE 1
Characteristics of 11 Homosexuals Who Changed to Heterosexuality After Religious Participation

Subject	Age (years)	Age When Homosexuality Identified (years)	Age When Changed to Heterosexuality (years)	Years as Heterosexual	Marriage Data: Age at Marriage (years)	Marriage Data: Years Married	Intrapsychic Evidence of Current Homosexuality: Dreams	Intrapsychic Evidence of Current Homosexuality: Fantasies	Intrapsychic Evidence of Current Homosexuality: Impulses	Kinsey Ratings[a]: Before Change	Kinsey Ratings[a]: After Change
1[b]	24	10	19	5	17	7	No	Yes	No	6	0
2	24	12	22	2	20	4	No	No	Yes	6	0
3[b]	28	15	24	4	25	3	No	No	No	6	0
4	21	13	20	1			No	No	No	6	1
5	34	8	27	7			No	Yes	No	6	1
6	23	14	22	1			No	No	Yes	6	1
7[c]	26	10	22	4	24	2	No	No	Yes	6	2
8[b]	28	11	22	6	22	6	Yes	No	No	6	0
9	26	11	22	4			No	No	No	6	0
10[c]	35	10	29	6			No	Yes	Yes	5	2
11[c]	32	14	28	4	29	3	No	No	Yes	4	2

[a]Kinsey scale: a rating of 0 is heterosexual, 3 is bisexual, and 6 is homosexual.
[b]Subject at one time considered himself bisexual.
[c]Homosexual impulses were still source of neurotic conflict for subject.

response and relationships. A rating of 5 is predominantly homosexual, with occasional psychic response to the opposite sex. A rating of 6 is an exclusively homosexual physical and psychic response.

In our sample the before-after scores on the Kinsey scale were 6-0 (N=4); 6-1 (N=3); 4-0 (N=1); 6-2 (N=1); 5-2 (N=1); 4-2 (N=1). We cannot compare these scores with other available data. We do note that only 6% of the Bell and Weinberg sample scored in the 0–3 range, but they gave no breakdown of this small but significant group. The national sample in the Kinsey (10) data on men, using age- and education-corrected figures, indicated that most men who were the same age as our sample had a rating of 0 with a small but definable group who scored at the 1 and 2 levels (married men: 94% scored 0, 6% scored 1, 1% scored 2; single men: 68% scored 0, 7% scored 1, 2% scored 2). The importance here is that there exists in the general population married and single men who demonstrate some degree of homosexual inclination, even though they are predominantly heterosexual. This group does not include those who are loosely termed bisexuals, who are rated 3 on the Kinsey scale. Thus, all subjects in our sample demonstrated a strikingly profound shift in sexual orientation.

Despite the fact that our subjects were currently living a heterosexual lifestyle and had a commitment to heterosexuality, we were particularly interested in whether they still experienced intrapsychic homosexual proclivities. Thus, we asked them to elucidate a history of their homosexual dreams, fantasies, and impulses during the time of this claimed heterosexuality. None of our subjects reported a dramatic or immediate cessation of intrapsychic homosexual proclivity. Rather, they described the gradual diminution of their homosexual drives as they proceeded in a heterosexual life. The data in table 1 include the current intrapsychic status of the subjects. Of the 5 subjects who were rated 0, 2 had no intrapsychic evidence of homosexual ideation. One subject had occasional memories of past homosexual experiences occurring during times of emotional distance from his wife; however, he declared no impulse to return to homosexuality. One subject had occasional homosexual feelings, which he experienced as distasteful and ego-dystonic, and 1 had rare homosexual dreams of personal friendship without sexual activity. Of the 3 subjects who had a 1 rating, 1 subject had fleeting memories of past homosexual friendships and 1 had occasional homosexual impulses, which he would dismiss as a memory of the past. The 3 subjects who were rated 2 reported significant intrusion into their consciousness of homosexual impulses; although these impulses were not acted upon, they generated discomfort, guilt, and anxiety. One subject had had one homosexual experience a year in the past, for which he felt immense guilt. Only these last 3 subjects had any definable neurotic symptoms manifested in their current psychiatric histories and mental status examinations. The homosexual impulses of these 3 subjects were a source of neurotic conflict for them.

Although one might argue that any evidence of intrapsychic homosexual proclivity in these men suggests that their homosexuality had not been resolved, this argument overlooks the fact that heterosexuality or homosexuality is not an either/or, mutually exclusive phenomenon. Indeed, Kinsey convincingly showed that even among lifelong heterosexuals there were occasional homosexual dreams, fantasies, and impulses. Similarly, Saghir and Robins reported that among exclusively heterosexual men, 94% had experienced platonic feelings toward other men, and 11% had wanted physical, nongenital contact. Thus, our data do not suggest some magical change or massive denial or repression, but rather they suggest the gradual development of a rejection of the homosexual object choice as an increased cathexis of the heterosexual object is developed.

Psychological Status and Social Relations

As shown in table 2 our subjects demonstrated a remarkable state of psychic well-being, with an absence of negative affects and the presence of strong feelings of self-acceptance and happiness. The exceptions were the 3 subjects rated 2, as noted previously, who manifested neurotic symptoms. In contrast, the homosexual men from the Bell and Weinberg sample demonstrated overall much more psychological distress. In the Saghir and Robins study, there was a 33% incidence of neurotic symptoms among the homosexuals, as compared to 27% reported in our study.

At the time of this study, our subjects had an equal distribution of friends among men and women; they had no friends who were homosexual; and 82% were equally at ease with men and women. This stands in marked contrast to the Bell and Weinberg data: 62% of their sample had more men friends and 44% had only homosexual friends; only 12% were at ease with both men and women. Our verbatim case notes revealed that since pre-adolescence all of our subjects had preferred male companions and had made exclusively male friendships; they had demonstrated an aversion and avoidance of females before their change to heterosexuality and marriage was the consequence of the heterosexuality.

Another significant difference in our data from the Bell and Weinberg data is that our subjects did not attempt to hide their homosexuality; instead they sought to inform and include their spouse in the further elaboration of their heterosexuality. Consequently, rather than the demands of heterosexuality producing conflict, as is often reported in marriages involving one homosexual, our subjects reported that their heterosexual marriages further solidified and developed their heterosexual identity and sexual preference.

Another important difference exists between the previous studies and ours. According to our subjects' religious ideology, sexual intercourse is the consequence of love and an expected part of a marital relationship. Premarital and extramarital sexual experience is considered immoral; heterosexual promiscuity is as immoral as homosexual promiscuity. Therefore, there is an active prohibition against engaging in sexual intercourse to prove masculinity or to overcome homosexuality. According to the Christian norms of their church, our subjects were expected to develop loving relationships with their Christian brothers and sisters that were nonerotic. Within the church's ideology was the explicit assumption that homosexuality was a maturational deficit that resulted in an inability to engage in mature nonerotic love with both men and women. As a result of maturing in appropriate interpersonal relations, a man was expected to develop an erotic attraction to a woman as a mature love object. Sexual experience then would follow appropriately within the marriage.

TABLE 2
Psychological State of 11 Male Homosexual Subjects Who Changed to Heterosexuality After Religious Participation Compared with 575 Male Homosexuals Studied by Bell and Weinberg (9), in Percents

Item	Subjects[a]	Bell and Weinberg Sample[b]
Feelings of exuberance		
Low	0	29
Medium	27	30
High	73	41
Feelings of self-acceptance		
Low	0	39
Medium	27	40
High	73	21
Feelings of loneliness		
Never	64	26
Rarely	9	37
Sometimes	27	24
Often	0	14
Degree of worry		
Not at all	55	4
Very little	18	25
Somewhat	18	43
A great deal	9	26
Feelings of depression		
Low	73	30
Medium	27	33
High	0	37
Feelings of tension		
Low	73	28
Medium	18	34
High	9	35
Feelings of paranoia		
Low	91	25
Medium	9	33
High	0	42
Feelings of happiness		
Very unhappy	0	3
Not too happy	9	14
Pretty happy	18	55
Very happy	73	28

[a]Age range, 21–35 years; mean age, 27 years.
[b]Mean age, 37 years.

Indeed, this was the sequence our married subjects followed. They reported that they had no difficulty in achieving meaningful and satisfying heterosexual relationships. Again, these data support the sociosexual orientation of heterosexuality in our sample, which is in marked contrast to our subjects' former exclusive homosexual friendship patterns.

Heterosexual Marital Experience

In table 3 we present a synopsis of the heterosexual marital experience of our 6 subjects who were currently married (5 never married). It is widely reported that about 20% of homosexual men marry at least once. Saghir and Robins and Bell and Weinberg reported similar figures in their two studies. So it is not particularly unusual to find these marriages. Our subjects' ages at the time of marriage ranged from 17 to 29 years (mean = 24 years). Both of the other studies reported the mean age of their subjects at the time of marriage was 24 years. The number of years our subjects were married ranged from 1 to 7 years (mean = 4

TABLE 3
Self-Reports of Heterosexual Marital Experience of Homosexual Subjects Who Changed to Heterosexuality After Religious Participation Compared with Homosexuals Studied by Saghir and Robins (8) and Bell and Weinberg (9), in Percents

Self-Reported Item	Subjects[a] (N=6)	Saghir and Robins Sample[b] (N=16)	Bell and Weinberg Sample[b] (N=116)
Duration of marriage			
Less than 1 year	0	—	9
1-2 years	16	70	24
3-5 years	50	28	23
6-10 years	33	1	18
More than 11 years	0	1	25
Wife informed of homosexuality before marriage			
Yes	100		33
No	0		49
Not homosexual then	0		15
Spouse knew without being informed	0		3
Marital happiness			
Very unhappy	0		18
Moderately unhappy	0		25
Moderately happy	20		42
Very happy	80		15

[a]Mean age at time of marriage was 23 years.
[b]Mean age at time of marriage was 24 years.

years). As noted in table 3, this is not unlike the data from the other two studies, except their samples tended to have shorter durations of marriage. However, it is striking that all of our subjects had discussed their homosexuality with their spouses before marriage, whereas only 33% of the Bell and Weinberg sample did so. Further, our subjects were happily married. In the Saghir and Robins sample only 2% of the homosexual men were still married, and these men engaged in homosexual affairs by mutual consent with their marriage partner.

The major difference between our findings and the two findings previously reported, however, lies in the motivation and result of marriage. It is well known that through marriage some homosexual men attempt to prove they are not homosexual, or they attempt to overcome their homosexuality or to gain social protection. Saghir and Robins reported that 90% of their subjects married for these compensatory motivations, and only 6% married for love. Furthermore, studies of such homosexual marriages reveal that the unions are ultimately unsatisfying, unstable, and fail to resolve the issue of homosexuality (11, 12). As noted by Saghir and Robins, some of these marriages survive when there is mutual acceptance of continued homosexual activity as part of their marriage.

In contrast, only 2 of our subjects married out of motivation to "cure" their homosexuality. One married at age 17 but separated due to his homosexual activities. After his religious conversion and consequent change to heterosexuality, he reunited with his wife but first they explicitly discussed his homosexuality. The other man married at age 19, but he was divorced after 1 year because of his homosexual activities. He remarried 10 years later after his change to heterosexuality. In addition, the 3 subjects who had considered themselves bisexual before their conversion dated women and attempted to form heterosexual attachments, but they found themselves unable to maintain this pattern. After their change to heterosexuality these 3 subjects married.

As homosexuals, all of these 6 subjects found themselves unable to enter a satisfactory marital relationship. After their commitment to heterosexuality, their social relationships with women developed significantly, and their attraction to women developed, they sought marriage because of sexual attraction and love for a woman. Thus, our subjects did not use marriage as a solution to their homosexuality and each changed without difficulty to a heterosexual relationship with his wife. None of the 5 unmarried subjects engaged in heterosexual intercourse because of the religious prohibitions mentioned above. However, all of them looked forward to marriage, and 3 were actively dating.

The above attitudes and behavioral sequence appear counter to many therapeutic endeavors that attempt to change homosexuality by encouraging heterosexual liaisons (13). These endeavors seem psychodynamically inappropriate, and they force the homosexual toward a heterosexual experience without the development of a capacity for heterosexual object choice. In fact, Bell and Weinberg's data indicate that a substantial number of homosexuals can perform heterosexual intercourse without difficulty but that the problem is their gender object choice. Further, their data reveal that a significant number of homosexuals report heterosexual impulses and arousal. Thus, they found that 45% did not consider themselves exclusively homosexual, 76% behaved in a more homosexual manner than they actually felt, 33% had heterosexual dreams, 23% had heterosexual masturbatory fantasies, and 72% experienced heterosexual arousal.

Furthermore, it is not difficult to condition homosexual persons to respond to heterosexual erotic stimuli. Feldman and MacCulloch (14) reported that with sophisticated behavioral conditioning they were able to change the arousal reactions in 35 of 43 homosexual men; the subjects' response to male eroticism was eliminated and replaced by a positive response to female eroticism. These observations suggest that therapists can push homosexuals into heterosexual performance, including heterosexual erotic arousal. But the problem, as critics note, is that such heterosexual performance does nothing to change the internal cathexis of preferred sexual object. Hence, the homosexual returns to the preferred homosexual object after a satisfactory heterosexual experience.

In contrast Saghir and Robins reported that men in

their heterosexual control group first developed cognitive expectations and fantasy rehearsal of close, warm relationships with women without erotic arousal only after they had nongenital interpersonal dating contacts with them. It is noteworthy that the religious ideology and behavioral expectations of our sample followed the normal development sequence leading to heterosexuality reported by Saghir and Robins.

Religious and Social Attitudes

Beliefs and values appear to play a significant role in changing sexual orientation. Our subjects were very religious and active in religious practice. They believed that the practice of homosexuality was immoral. However, they believed that the condition of homosexuality was an emotional problem due to psychological maldevelopment. All regretted having been homosexual, and all had considered giving it up. They wanted to change to heterosexuality as a religious responsibility.

The Bell and Weinberg homosexuals were strikingly different. Only 6% were very religious; 51% were "not at all" and 28% were "not too" religious. The majority, 72%, did not believe homosexuality was immoral, and 49% did not regret being homosexual. More than one-half, 63%, of the sample disagreed or strongly disagreed that homosexuality was an emotional disorder; 71% said they had never considered giving up homosexuality. In the sample 14% said they wanted to change and then only "by magic." These differences, we believe, reflect the importance of motivation in the potential for change.

Homosexual Background

Our 11 subjects reported that the primary cause of their homosexuality was unsatisfactory relations with their parents. Eight subjects stated that their fathers were distant, aloof, and uninvolved with them. As a result, they identified more with their mothers and developed effeminate manners. They felt like "sissies" and were insecure and inadequate in their own identity as males. They saw homosexual behavior as a search for a masculine identity through identification with the male sex partner. Three subjects reported the same psychodynamics and identity conflicts but focused their dynamic conflict on their mothers, whom they experienced as harsh, controlling, and demasculinizing.

These dynamics are strikingly similar to those explicated by Bieber and associates (15) in their intensive psychoanalytic research. All of our subjects displayed some effeminate mannerisms, which no doubt reflected the maternal identification. These mannerisms persisted despite their current strong heterosexual orientation. This finding is similar to the observations by Stoller (16) on the treatment of effeminate boys. He reports that although the boys developed heterosexual orientations they still retained the early acquired body mannerism identifications with the maternal object. Thus, early body identifications are not as easily changed—if at all—as sexual object choice may be changed.

All our subjects stated they were disappointed with their homosexual experiences. They found the relationships fleeting and emotionally unfulfilling. Second, they did not find the masculine identity, security, and self-acceptance they sought. Apart from these primary motivations, they also experienced secondary guilt and shame, which generated anxiety and emotional turmoil.

In addition to the above psychological motivations, all the subjects made a decision to change to heterosexuality after their religious conversion because of their commitment to the religion's ideology, which defined heterosexuality as a necessary component of a mature member of the church.

The developmental sequences of these subjects are remarkably similar. All had been actively engaged in homosexual relationships and activities. All except 2 had "come out" and were active in the gay lifestyle. Although all were dissatisfied with their gay lifestyle, they had been unsuccessful in changing their lifestyle by any known method. All of our subjects had come from some type of religious background and most had unsuccessfully attempted to pray or use religious activities to change their homosexuality. However, they did not reveal their homosexuality to their religious groups because as homosexuals they had experienced hostility before from religious groups. Thus, their attempts to seek entrance into the church were made in private and within the context of their anticipating the church members' hostility.

When our subjects came in contact with the church's crisis service for homosexuals, they found a welcome reception as homosexuals. No attempt was made to make them change their homosexuality. Rather, they were presented with the invitation to commit their life to Christ and the church. All subjects had an explicit Christian conversion or rededication. They were then invited into small church fellowship groups where they studied the Bible and learned expected Biblical patterns of mature lifestyles. This included an expectation to engage in loving, nonerotic relationships with both men and women in the fellowship groups.

All of our subjects remarked that they soon became aware that they were psychologically immature and had poor interpersonal relationships. In essence, then, these subjects were exposed to an ideological expectational set demanding growth in personal and interpersonal maturity. In addition, there was a great deal of actual interpersonal experience, behavioral rehearsal, and behavioral practice. All the subjects said they were surprised to experience acceptance, nonjudgmental evaluation, and nonerotic love from both men and women. These were new experiences for

them, and in turn they began to learn and practice these new styles of interpersonal relationships. As a result, they began to identify with those they considered "mature Christian men," and they began to experience and practice nonerotic relationships with Christian women in the church. Thus, they felt they were growing in mature object relations with both men and women. These fellowship groups were not organized or conducted as psychotherapy or encounter groups but were goal oriented around Bible study and fellowship. Elsewhere Politser and Pattison (17, 18) have reported on similar psychotherapeutic effects of nontherapy community groups conducted for social and civic purposes.

During this time of psychological maturation there was no demand that these subjects stop being homosexuals. Homosexual behavior was simply defined as immoral, and they were expected not to engage in homosexual practices. However, their psychological condition of homosexuality was interpreted by these church members as a sign of Christian immaturity. It was expected that they would learn how to be heterosexual as they developed Christian maturity.

The subjects did not report any immediate change in their homosexual dreams, fantasies, impulses, or orientation. Rather, they reported they gradually acquired a secure and satisfying identity as men with high self-acceptance. As a corollary to this, they began to experience nonthreatening and satisfying interpersonal relations with women. Consequently, they reported a steady diminution in their homosexual feelings and a steady increase in their heterosexual feelings.

The subjects who married reported their homosexual dreams, fantasies, and impulses did not vanish. However, as they progressed in a satisfactory marital relationship, their homosexual dreams, fantasies, and impulses gradually diminished over time.

The process of change was not magical, spontaneous, or dramatic. Change was imbedded in an accepting, evaluative, and loving, nonerotic social milieu that provided expectations, ideology, and actual interpersonal experiences and thus promoted what they saw as personal growth into heterosexuality. Their intrapsychic processes demonstrated a similar pattern of gradual change in gender object choice, with a time-lag discrepancy The behavioral changes to heterosexuality preceded the intrapsychic changes.

DISCUSSION

To our knowledge, this report is the first documented study (excluding single anecdotes) of profound behavioral and intrapsychic change in sexual orientation from exclusive homosexuality to exclusive heterosexuality without long-term psychotherapy.

One of the study's limitations is that we relied primarily on retrospective data. However, the corollary data from the crisis center staff, who intimately knew the subjects during their time of change, support our data. Further, we personally knew and carefully observed 2 of the subjects over a 5-year period. Thus, we have reasonable confidence in the reported data.

Another limitation is the fact that our subjects changed early in their life and at an age when Bell and Weinberg reported that most homosexuals marry or attempt heterosexuality. Thus, we cannot make a long-term prognosis. Our data demonstrate, however, a very different pattern of change than that reported for homosexuals who either engage in occasional heterosexual intercourse or who try to escape from homosexuality without personal growth and psychological maturation.

There is also a limitation in comparing the Saghir and Robins and the Bell and Weinberg samples to our sample; our subjects' mean age was 27 years, whereas the other two samples contained somewhat older men. Thus, their samples may well represent homosexual populations beyond the optimal age for change in sexual orientation. A more comparable sample is reported by Troiden (19); 50% of his sample were in their 20s. He found that between the ages of 21 and 24 years there is a major period of critical evaluation about assuming a homosexual identity. He reported,

> A relatively large number of males conciously "test' the extent to which they may be sexually attracted to men . . . a substantial number of males may decide that homosexuality is not for them and choose to leave the scene entirely. It is therefore quite likely that only a tiny portion of the American males who practice homosexual behavior ever take on gay identities.

Troiden's data comport well with the data from our sample, the Kinsey data on spontaneous change (10), behavioral treatment data (14), psychoanalytic treatment (20), and the new Masters and Johnson sexual treatment data (21), all of which indicate that homosexuals who successfully change to heterosexuality are usually much younger than 35 years old.

This finding of change, as noted, is rarely reported in the literature. Yet clinical observers comment that this may be an artifact of our investigations. Liss and Welner (6) concluded that "this change in sexual orientation may be more frequent than is apparent." In his recent review Bancroft (22) stated,

> If in fact we had evidence that either a homo or a heterosexual identity was an immutable and fundamental aspect of an individual's nature, then any attempt to modify such an identity would be inappropriate and unjustifiable. But there is no such evidence, and we know that many individuals pass through a phase of homosexuality or bisexuality into a stable heterosexual role. (p. 1,191)

Kinsey and associates (10) concluded from their data

that change in sexual orientation in young men was common and that sexual orientation was far less fixed, more plastic and fluid, than commonly supposed. This contention is supported by their 1948 data. These data showed that of those men who were comparable to our sample in age and education, who were between the ages of 16 and 35, and who were rated 0 (exclusively heterosexual), 6% previously had a rating of 6 (exclusively homosexual) and 10.5% had a rating between 4 and 6 (predominantly homosexual). Thus, there is a significant statistical probability that a sample of homosexual men will change from exclusive homosexuality to exclusive heterosexuality. However, scientific and professional research has not been focused on detecting or studying these subjects.

Is it possible to predict subjects with a high potential for changing from homosexuality to heterosexuality? Bancroft (22) found that less feminine homosexual subjects under 35 years of age whose first overt homosexual experience occurred after age 16 and who had a history of heterosexual arousal were most responsive to treatment. Myerson and Lief (23) found that 2 of their 9 subjects who were exclusively homosexual changed orientation while undergoing psychotherapy, whereas 7 of 10 bisexuals changed. Woodward (24), on the other hand, reported none of his 6 exclusive homosexual subjects changed, while 28 of 42 bisexuals changed. Our data do not comport well with the above predictors because our subjects appear to have been more exclusively homosexual.

One possible explanation for this discrepancy is that the above investigators studied subjects who sought psychological treatment and therefore these subjects have been more neurotic. This may have skewed their potential for change, although such comparative data are not available. In our sample the 3 neurotic subjects were the only ones with significant remaining homosexual ideation. A second explanation may be that behavioral manifestations of homosexuality are less significant predictors than intrapsychic variables, such as degree of maturational fixation and ego strength. A third explanation may be that persons seeking professional treatment are older and beyond the major period of identity flux (ages 12–24 years) identified by Troiden (19).

The suppression of overt homosexual behavior or involvement in heterosexual activity does not constitute a "cure" or change in sexual object choice. Thus, Saghir and Robins state the criteria for cure as follows:

> a reversal of the psychological responsiveness from a predominantly homosexual to a primarily heterosexual one. Thus a "cured" homosexual would not only disengage from homosexual activity but he would also disengage emotionally and to a large extent from homosexual attachments, including homosexual fantasies, dreams and physical arousal by sight or touch. (p. 319)

By the above criteria, we conclude that 8 of our 11 subjects amply demonstrated a "cure." The remaining 3 subjects had a major behavioral and intrapsychic shift to heterosexual behavior, but the persistence of homosexual impulses was still significant.

None of our subjects had ever sought professional treatment for either psychiatric reasons or for their homosexuality. As we have indicated, however, their change was not spontaneous. Instead, they participated in what we consider a powerful therapeutic milieu. Frank (25) identified the generic therapeutic features of nonmedical healing activities as methods that involve

> an interplay between patient, healer, group, and the world of the supernatural, which serves to raise the patient's expectancy of cure, help him to harmonize his inner conflicts, reintegrate him with his group and the spirit world, supply a conceptual framework to aid this, and stir him emotionally. (p. 53)

Certainly each of these elements is present in the spiritual guidance our subjects participated in with their religious fellowship groups when trying to achieve religious maturity and heterosexuality. Our subjects did not participate in psychotherapy within the conceptual framework of secular medical treatment modalities. But they did participate actively in a "folk psychotherapy" within a supernaturalistic framework. As Pattison and associates (26) and Pattison (27) have documented elsewhere, such supernatural contexts for healing may be more effective for adherents to that supernatural world view than traditional psychotherapy. This seems the case here.

Other "Therapies"

It would be inaccurate to label the changes reported here as "spontaneous," inasmuch as we have identified major variables involved in producing change in the lives of our subjects. Instead we have identified a model of "folk healing." The model of change proposed here involves ideological commitment; cognitive structuring of beliefs, values, and expectations; behavioral interactions over time between subjects and their social reference groups; and a sequence of mutually expected behavioral changes. Thus, this folk treatment is embedded in the total sociocultural milieu of the subject's life. In an analysis of cross-cultural healing methods, Pattison (28) showed that these same healing elements are obtained in numerous indigenous healing methods. A comparison of "folk healing" with other therapies must be cautious because the populations that seek different treatment methods may vary substantially in their patient demography and motivation, type and degree of homosexuality, and treatment goals. In our review of the treatment literature we find such wide variation in the study populations as to render comparative treatment evaluation of little value.

Psychoanalytic psychotherapy aims at personality organization on a pre-oedipal level. But analytic treat-

ment involves only the patient and therapist as a social field, even though many of the interpersonal transactions may be of the same genre. That is, the patient matures by "working through" a relationship with an analyst or with significant others, as in our study.

A comparison with behavior therapy involves other therapeutic parameters, in that behavioral treatment focuses on the stimulus-response sets of the patient. As noted, in our study such behavioral stimulus response sets occur, although not with explicit cognitive focus. Yet, one might argue that our subjects experienced multiple social agents who provided response contingency modification. Finally, the recent report by Masters and Johnson (21) indicates the importance of ideological commitment to change and to cognitive and behavioral structuring; it can also provide a major focus for interpersonal social relations. All of these components have similarities with our "folk model." Thus we can identify common elements of method shared in the folk model with each of the three major professional treatment modalities.

Which methods might be most acceptable and effective for different homosexual populations is undetermined. Obviously the folk model mentioned here is based on a specific religious ideology, whereas the other treatment models are oriented toward a pluralistic population. Nevertheless, the data suggest the potential for development of therapy models that might encompass the larger life space of the subject rather than just discrete treatment methods practiced apart from the everyday life and social milieu of the subject.

The problem of social deviancy for the homosexual is well known. Our subjects approached this problem in a substantially different manner than that espoused by the gay liberation movement. Self-help groups approach deviancy through two different mechanisms. One approach is to change social attitudes and reduce the definition of deviancy. An example is the women's liberation movement, which seeks to change the social definitions of status and the role of women in society. The other approach is to reduce the deviant behavior of those considered deviant, e.g., the self-help groups of parents who batter their children. Some self-help groups, such as Alcoholics Anonymous, seek to reduce both the social deviancy and to change the deviant behavior of their members.

The gay liberation movement seeks only to reduce the social definition of deviancy of homosexuality. They claim "gay is good" and an acceptable alternate lifestyle; it is not to be considered deviant (29, 30). However, this perpetuates the confusion described elsewhere by Pattison (31) in that the behavior of homosexuality is transformed into the personal identity of the homosexual. Thus, the homosexual person is reinforced in finding a personal identity not as a person but as a member of a deviant class. As Sagarin (32) notes, "Such a concept is a reification, an artificially created entity that has no basis in reality." The social stereotype of deviant behavior becomes the personal stereotype of identity tied to a social label. The social role of the deviant homosexual becomes the only avenue to attain and maintain identity. Sagarin (32) again observed,

> Because they want to believe that they are not worthless (which they are not), and because they confuse the worth of a person with that of a characteristic, they go the next step and say that gay is good.

The attempt to redress the social position that "gay is bad" with the social position that "gay is good" merely replaces one stereotype with another and does not change the problem of destruction of personal identity inherent in the latter position. Sagarin (29) suggests that the ideological belief in identity via one's deviancy, in fact, reinforces the deviant role behavior.

Religious persuasions and activities per se do not solve the social problem of deviancy. Bauer (33) reported that in a homophile church, the search for identity and community support was often vitiated by its liberal theological stance, which accepted the identity of the person as homosexual. Thus, the church ideology perpetuated the confusional problem of identity. Further, the exclusive association with other homosexuals in the homophile church created continuing interpersonal homosexual tensions.

In contrast, our subjects rejected the ideology that "gay is good" within the framework of their orthodox Christian theology. They reduced the social deviancy of homosexuality by stating that homosexuality is only a behavior and not the identity of the person. Therefore, they shared a common concern to reduce the social hostility and social rejection of persons just because they were homosexual. In practice our subjects were received into church fellowship as homosexuals. They did not accept homosexuality as a fixed condition; they believed that homosexuality was a mutable condition that could be changed. Our subjects also reduced deviancy through personally changing the deviant condition. In this sense, the "ex-gay" self-help fellowship groups are similar to Alcoholics Anonymous in seeking change in both social deviancy and personal deviancy. Further, the church fellowship did not appear to provoke homosexual tension as in the homophile churches, but reduced the tension within a heterosexual fellowship (33).

The "Ex-Gay" Movement

As a result of our research, we found there are numerous informal "ex-gay" self-help groups in existence throughout the United States, Europe, and Asia. This self-help movement has not received public attention, but our correspondence indicates a lively social movement underway. Our correspondents make claims regarding change in their sexual orientation similar to those reported by the group we studied.

Thus, there appears to be a substantial number of persons who are similar to the population reported here. This phenomenon suggests that like many other self-help movements, a peer group of ex-homosexuals may be emerging that offers an effective avenue of change not currently provided through professional resources. It is noteworthy that 8 of our subjects participated in helping their peers as ex-homosexuals. They reported no homosexual arousal in these contacts, which they interpreted as evidence of their solid heterosexuality.

CONCLUSIONS

The data provide a substantial body of evidence for the plausibility of change from exclusive homosexuality to exclusive heterosexuality, which is in accordance with the Kinsey statistical probabilities for such change, the Masters and Johnson data, and the clinical or observational anecdotes of such change. Our data demonstrate that such change has occurred through significant longitudinal experiences in "folk therapy" provided within a supernatural framework and utilizing generic methods of change common to folk therapy. Our data suggest the importance of ideology, expectation, and behavioral experience in producing change. The evidence suggests that cognitive change occurs first, followed by behavioral change, and finally intrapsychic resolution. Finally, the data suggest the importance of our concepts of homosexuality. When homosexuality is defined as an immutable and fixed condition that must be accepted, the potential for change seems slim. In our study, however, when homosexuality was defined as a changeable condition, it appears that change was possible.

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Religiousness, Conversion, and Subjective Well-Being: The "Healthy-Minded" Religion of Modern American Women

BY PHILLIP SHAVER, PH.D., MICHAEL LENAUER, M.A., AND SUSAN SADD, PH.D.

Data from a survey of 2,500 American women were examined to determine 1) the nature of religiousness among women and, in particular, whether modern religiousness corresponds to William James' conception of "healthy-minded" religion; 2) how religiousness is related to self-reports of mental and physical health; and 3) differences between converts (mainly to Protestant denominations) and women who have maintained the same religious affiliation all their lives. The authors found that healthy-mindedness described the nonconverts (the majority) fairly well, and that certainty of beliefs (either strong religiousness or confident nonreligiousness) was associated with better mental and physical health. Converts were distinguished from nonconverts by the former's relatively happy and less religious childhoods, stronger adult religiousness, and greater authoritarian tendencies.

Recently published studies, and Deutsch's paper in this special section, suggest that cult members are people who were psychologically troubled and unhappy before joining a cult and who, according to their own reports, improved substantially after conversion. Because the cults are relatively new to the American scene and, in some cases, advocate unfamiliar beliefs and practices, they have captured the attention of journalists and psychiatric researchers. Few investigators have conducted parallel studies of converts to "mainstream" American religions, and the implication seems to be that cults are in some way psychologically special. However, we suggest that modern converts to Christian sects and denominations are similar, in rough outline at least, to people who convert to cults.

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THE MODERN REVIVAL OF EXPERIENTIAL RELIGION

The recent revival of religion in America has been well documented in both popular and academic sources. The cover story in the April 11, 1977, issue of *U.S. News and World Report* was "A Time of Renewal for U.S. Churches." After 15 years of steady decline in church attendance, from 49% of the total U.S. population in 1958 to 40% during the period 1972–1974, weekly church attendance turned upward, to 42%, in 1976 and subsequent years (1). More importantly, after reaching a low of 14% in 1970, the percentage of Americans who told a Gallup Poll that religion is "increasing its influence on American life" rose to 31% in 1974 and 44% in 1976. In a recent summary article, Gallup (1) stated, "Evidence is mounting that the U.S. may be in an early stage of profound religious revival, with the Evangelical movement providing a powerful thrust" (p. 50).

One of the most interesting features of the new religiousness is its apparent relationship with, and similarity to, other experiential and self-help quests. According to Gallup (1),

> These gains for religion have been accompanied by considerable interest on the part of the public in what may be termed "experiential religion"—involvement in such movements as Transcendental Meditation, Yoga, the Charismatic Movement, Mysticism, and Eastern religions. A recent survey indicates that a projected 6 million Americans are participating in, or are involved with TM, 5 million in Yoga, 3 million in the Charismatic Movement (Charismatic Renewal), 3 million in Mysticism, and 2 million in Eastern religions. (p. 50)

There are at least two kinds of explanations for Americans' intense interest in religious experience; on close inspection these explanations prove to be highly related.

Berger, a sociologist, argued in a recent book (2) that modernity (advanced technology and associated forms of social life) greatly increases the range of life choices available to the average person. Life patterns that were once taken for granted (i.e., experienced as matters of fate and faith) are now viewed as options within a vast array of possible lifestyles. Each option

is thus relative, and almost no one can be certain that his or her choices are correct. In Berger's words, "Modernity pluralizes both institutions and plausibility structures" (2, p. 17).

According to Berger, there are three ways to deal with the crisis of faith resulting from religious pluralism or relativism. One, the deductive option (neo-orthodoxy), blinds itself to rival claims and sticks doggedly to the letter of its scripture. For most informed people, this option is not viable. The second approach, the reductive option, is to secularize religion. With this approach, religion loses its experiential uniqueness and becomes a blend of ethics and self-help psychology. Berger forms the third, inductive, option:

> The inductive option is to turn to *experience* as the ground of all affirmations—one's own experience, to whatever extent this is possible, and the experience embodied in a particular range of traditions . . . religious traditions are understood as bodies of evidence concerning religious experience and the insights deriving from experience. (2, pp. 62-63)

Berger shows, in his clear and provocative analysis of modern religious trends, that the conditions of modern society force religion to either become experiential or succumb to secularization. Theology (the body of interpretive beliefs about religious experience) is debatable, but the fact of religious experience is not.

A second valuable source of ideas relevant to modern religious experience is William James' analysis (3) of what he called "healthy-minded" religion—the form of religion that emphasizes the benefits to mental and physical health which can be derived from religious experience and belief. According to James, healthy-minded religion "gives to some of us serenity, moral poise and happiness, and prevents certain forms of disease as well as science does, or even better. . . ." His explanation of the healing power of religion focused on the value of certain kinds of beliefs. Beliefs, in his analysis, are blueprints for action, and each action is, in turn, capable of verifying the beliefs that generated it. (More recently, Geertz [4] has elaborated on this argument.) The absence of firm beliefs is associated with uncertainty and inadequately guided behavior. Conflict and indecisiveness are incompatible with healthy-mindedness. (A similar, more technical analysis of cognitive dissonance has evolved from contemporary social psychology; see Abelson and associates [5] and Wicklund and Brehm [6].)

In a modern heterogeneous society such as ours, religious belief systems are prime candidates for conflict, uncertainty, and doubt, as Berger (2) has shown. Americans routinely encounter a host of alternative and competing belief systems—everything from atheism to fundamentalist Christianity, and from Orthodox Judaism to a growing number of Eastern sects. Thus, there is pressure on Americans to close their eyes to diversity (Berger's deductive option), succumb to secularization (the reductive option), or insist on the validity and primacy of religious experience and attempt to fashion a coherent belief system that acknowledges both religious experience and the pluralism of interpretations it has fostered. There are many signs that an increasing number of Americans are taking the third option.

AIMS OF THE PRESENT STUDY

In 1976, when signs of an American religious revival became evident and the possibility of electing a born-again Christian to the presidency of the United States was being widely discussed, we and the editors of *Redbook*, a popular magazine for American women, decided to poll their readers about religious beliefs and experiences. Three questions addressed by the survey will be explored here:

1. What is the nature of religiousness among modern American women? In particular, are there signs that it is healthy-minded in James' sense?
2. How is religiousness related to self-reported mental and physical well-being? Does healthy-minded religion actually contribute favorably to health?
3. How do converts compare with women who have remained in the same religious group all their lives?

For present purposes we also want to ask how our picture of Christian converts compares with what other researchers are discovering about converts to esoteric cults.

METHOD

We used a 97-item questionnaire (assessing 219 variables) that was based in part on items from earlier questionnaires reviewed by Shaver (7). (Copies of the questionnaire are available from the authors on request.) The questionnaire was published in the September 1976 issue of *Redbook*. Readers were invited to mail precoded answer sheets to the editors of the magazine before the end of the month. More than 65,000 replies were counted and bundled in order of date of receipt, and from these we drew a random sample of 2,500 for the analyses reported here.

RESULTS

Obviously, we must be cautious in drawing conclusions from a self-selected sample. However, to the extent that the sample can be evaluated by comparison with more systematically selected samples, our respondents appear to represent a reasonable cross-section of the white female population of the United States. Their ages ranged from 15 to 91 years, with a mean of 35.46 and median of 31. They lived in a variety

of environments, from rural (14%) and suburban (18%) to metropolitan (10%), and came from every part of the United States. In addition, they were normally distributed in their political views: 4% described themselves as very liberal, 19% as liberal, 52% as moderate, 22% as conservative, and 3% as very conservative. However, in certain ways they were not representative of American women. They were somewhat better educated than average; 60% had more than a high school education, and 97% had graduated from high school. Ninety-six percent were white, fewer than 2% were black, and the remaining 2% were Hispanic, Oriental, and Native American.

Respondents were asked to indicate their present religion. The resulting distribution was compared with data from a Gallup poll conducted at about the same time. In both samples roughly a quarter of respondents were Catholic (*Redbook*, 23%; Gallup, 27%), 2% were Jewish, and most of the rest were Protestant (*Redbook*, 70%; Gallup, 61%).

Twenty-seven percent of our respondents described themselves as "very religious," 56% as "moderately religious," 12% as "slightly religious," 4% as "not at all religious," and 1% as "antireligious." In the Gallup poll, 65% of the respondents said religious belief was "very important" in their lives; 25%, "fairly important"; 5%, "not too important"; and 4%, "not at all important.' While the measures used in the two surveys are not directly comparable, we can be reasonably confident that the *Redbook* sample was no more or less extreme than Gallup's national sample; 83% of the women in the *Redbook* sample described themselves as at least "moderately religious," and 91% of the respondents in the Gallup poll said that religious belief was at least "fairly important."

Meaning of Religion

The survey included many questions designed to explore the meaning of religion for American women in 1976. One series of questions dealt with religious experience; another with key religious beliefs; and a third with what children should be told about God and religion. Each of these content areas will be explored briefly here.

Beliefs. The questionnaire presented a number of statements about religious beliefs, and respondents were asked to indicate their degree of agreement or disagreement with each statement on a 6-point Likert scale. These responses were correlated with responses to the general religiousness question. The results are too numerous to present in detail, but in general religiousness correlated most strongly (r ranging between .64 and .57) with what might be considered indicators of healthy-minded religion. These included such statements as, "Without God, life just doesn't make sense," "On the whole, having religious beliefs makes for a better and happier life," "My religious faith is a major source of self-confidence," "Angels actually exist." Religiousness had a weaker relationship (r between .49 and .19) with items such as, "The devil actually exists." "There actually is a hell in which the wicked will be everlastingly punished." "God wants us to accept our lot in life even if we are poor and powerless." The emphasis on healthy-mindedness should not be overemphasized; many religious women believed in the more threatening aspects of traditional religion. Still, fewer believed in these than in the benefits of religion.

Experiences. The respondents were asked a number of questions concerning whether they had ever had (during their adult lives) a direct experience involving God or the devil. The nature of the reported experiences was consistent with an emphasis on healthy-mindedness. The following experiences had the strongest correlations ($r=.58$, $p<.001$) with religiousness: "the feeling that God was helping you through a crisis," "the feeling that God has answered your prayers," and "the feeling that God has forgiven your sins." Seventy-nine percent were *sure* they had experienced the feeling that God answered their prayers; 76%, that God had helped them through a crisis; and 70%, that God had forgiven their sins. Far fewer (53%) were sure they had experienced the "feeling of being tempted by the devil." While this feeling was correlated with religiousness, it was the weakest correlation of the series.

There was, then, a strong tendency for respondents to believe in an active God whose influence they had experienced directly. The correlations indicate that this was particularly true for the more religious women. Not only were many of them sure that their beliefs had been verified by experience, they also asserted that religion had "worked" for them (e.g., God had answered their prayers and forgiven their sins).

What to tell children. Since 72% of the respondents were parents (the median age of the oldest child was 12; of the youngest child, 9), deciding what to tell children about God and religion should have concerned them and may indicate further the nature of their conception of God and religion. Several questions related to this matter were included in the survey.

On the whole, these women wanted their children to feel that God is a beneficent figure. For example, the vast majority of the sample wanted children to believe that "God loves you" (87.84%), that "God is everywhere—in people, in nature, in church" (79.72%), but did not believe children should be told that "God rewards you if you're good and punishes you if you're bad" (17.60%).

Religious Change and Conversion

The survey also asked respondents to indicate whether they had become more or less religious over the past five years; 32.8% replied that they had become much more religious; 24.71%, somewhat more religious; 28.83%, stayed about as religious; 9.20%,

somewhat less religious; and 4.88%, much less religious. Sixty-two percent of the very religious women reported that they had become much more religious; an additional 18% reported that they had become somewhat more religious; 18% had stayed about as religious; and 2% had become somewhat less religious. Most of the moderately religious women also reported having become more religious (27%, much more; and 31%, somewhat more so). These results are in keeping with national trends (1).

Sixty-one percent of the respondents still belonged to the religion in which they were raised, 17% had converted to a religion out of deep conviction, 7% had converted because of marriage, 4% had converted out of convenience, and 11% did not belong to an organized religion. Based on the assumption that conversions resulting from internal processes (i.e., deep conviction) are more interesting and revealing than those due to extrinsic factors (primarily convenience), comparisons were made between respondents who had converted out of deep conviction (converts) and those who were still members of the religion in which they were raised (nonconverts).

The converts were significantly ($p<.001$) more religious than the nonconverts. Forty-seven percent of the converts reported being "very religious," compared with 27% of the nonconverts; 61% of the nonconverts reported being "moderately religious," compared with 47% of the converts. Furthermore, the converts attended church more often and read religious books more often than nonconverts.

A much greater proportion of the converts had become more religious in the last five years and had had a higher frequency of religious experiences. More of them were sure that they had been "in the presence of God" (69.3% versus 51.5% of the nonconverts), that God had helped them through a crisis (89.7% versus 79.1%), that God had forgiven their sins (84.8% versus 72.7%). (All of these differences were significant beyond the .001 level.)

We found that the women who converted were currently very religious, but had not been very religious as children. Women who still belonged to their childhood religious denomination had been more religious than the converts as children, but tended not to be so strongly religious as adults. Respondents were also asked to indicate (on a 5-point scale) how happy they had been as children; fewer of the converts (25%) than of the nonconverts (32%) reported being "very happy," and more converts (15%) than nonconverts (7%) reported being "fairly happy." Moreover, a greater proportion of converts (44.7% versus 29.7%) were lonely as children, but more nonconverts (28.0% versus 19.1%) were lonely as adults. (All of these differences were significant beyond the .001 level.)

Other analyses revealed that converts did not differ from nonconverts in terms of income, recent change in economic status, age, or political views.

Relationship to Religious Leaders

The nontraditional nature of modern healthy-minded religion can also be seen in the respondents' independence of thought. They were asked, "If you find yourself in conflict with what your religious leaders are saying on an important issue (birth control, sexual practices, civil rights, and so on), what do you do?" Religiousness was not strongly related to answers to this question; regardless of the degree of their religiousness, respondents overwhelmingly relied on their own consciences. Of those who had religious leaders, 82% of the very religious said they would follow their own consciences, as would 96% of the moderately religious and 98% of the slightly religious. The more religious women *were* more likely than the less religious to have consulted a religious leader about personal problems and were more likely to do so again. These data imply that while religious women are more likely to consult a religious leader when they have personal problems, they will not follow the leader's advice if it conflicts with their own beliefs.

The picture is not so simple, however, when converts are compared with nonconverts. The converts evidenced more authoritarian tendencies. (We did not measure authoritarianism directly.) A significantly greater proportion of converts than nonconverts ($p<.001$) said that they asked God to guide government leaders (73.6% versus 59.9%), preferred political candidates who claim closeness to God (58.7% versus 48.9%), proselytized for their group (23.3% versus 8.1%), doubted that members of other religious groups had as good a chance for salvation (19.2% versus 11.3%), believed it would be better if everyone belonged to the same religion (64.6% versus 50.4%), strongly agreed that people should "fear" God (62.8% versus 48.6%), and strongly agreed that it is a sin to go against a parental wish (38.4% versus 28.7%). In addition, more converts than nonconverts were likely to follow their religious leaders because "the leaders know best" (14.2% versus 6.4%). Particularly interesting in light of these authoritarian tendencies is the fact that more of the converts (9.5% versus 5.5%) reported having been discipline problems as children ($p<.003$).

Religion, Health, and Well-Being

We were particularly interested in the possibility of a relationship between religiousness and well-being. The implication in William James' writings (3), and in the writings of modern theorists on cognitive consistency (5, 6), is that confidence and consistency of belief, regardless of its specific nature, is likely to be associated with health and happiness and with the absence of tension and conflict.

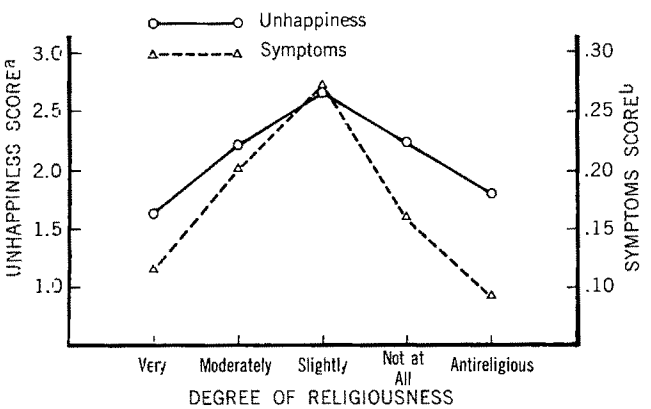
Respondents were asked, concerning 12 physical and mental health problems, "Which of the following have been true of you in the last year?" The 12 prob-

TABLE 1
Presence of Physical and Mental Health Symptoms Among 2,500 American Women, by Degree of Religiousness

Symptom	Score, by Degree of Religiousness					Overall F	Linear F	Quadratic F
	Very	Moderately	Slightly	Not at All	Anti-religious			
Headaches	.158	.222	.286	.219	.143	5.841[a]	.042	5.957[a]
Loss of sexual interest	.070	.154	.217	.190	.036	12.498[a]	.055	16.646[a]
Feelings of loneliness	.171	.298	.431	.229	.179	20.074[a]	.097	14.207[a]
Stomach upset	.104	.158	.152	.133	.143	2.761[b]	.146	.482
Crying easily	.113	.161	.255	.095	.000	10.069[a]	4.155[b]	13.118[a]
Worry and anxiety	.152	.312	.410	.219	.179	23.370[a]	.050	14.354[a]
Irrational fears	.039	.091	.131	.067	.000	7.814[a]	.895	9.370[c]
Restless sleep	.144	.230	.290	.133	.071	9.936[a]	2.282	9.312[c]
Feelings of worthlessness	.085	.163	.238	.860	.071	12.008[a]	.552	7.999[c]
Feelings of guilt	.080	.122	.176	.076	.000	6.174[a]	2.677	8.763[c]
Tense or keyed up	.195	.365	.441	.333	.143	21.757[a]	.547	22.373[a]
Just cannot go on	.094	.138	.228	.143	.107	7.862[a]	.053	5.551[b]

[a]p<.001.
[b]p<.05.
[c]p<.01.

FIGURE 1
Relationship Between Degree of Religiousness and General Unhappiness and Between Religiousness and Physical and Mental Symptoms Among 2,500 American Women



[a]Overal F=36.869 (p<.001), linear F=.355, quadratic F=29.407 (p<.001).
[b]Overal F=35.703 (p<.001), linear F=1.404, quadratic F=29.407 (p<.001).

lems are shown in table 1; the values of the variables ranged from 0 to 1. Curvilinear relationships between religiousness and problems are evident throughout the table; in fact, only the problem of stomach upset failed to follow this general pattern. Average symptom scores for each column, shown in figure 1, clearly reveal the overall inverted U pattern. A one-way analysis of variance was computed for each of the 12 symptoms and for the average of all problems, with religiousness serving as the independent variable. In these analyses both linear and quadratic (curvilinear) trends were tested for significance. For all but 1 symptom (stomach upset), the quadratic trend was highly significant. For only 1 symptom (crying easily) was the linear trend significant.

In addition to the health questions, respondents were asked, "In general how happy or unhappy have you been in the last six months?" Seven answer alternatives were provided; 34.8% of the respondents chose "very happy"; 47.4%, "moderately happy"; 5.3%, "slightly happy"; 3.9%, "neither happy nor unhappy"; only 8.7% selected any of the "unhappy" categories (slightly, moderately, very). (This is typical of results from survey studies of happiness; see references 8-10). A one-way analysis of variance was computed using religiousness as the independent variable and unhappiness (values from 1 to 7) as the dependent variable. As with mental and physical problems, the religiousness effect was highly significant (F [4,2462]= 36.87, p<.001), as was the quadratic trend (F [1,2462]=29.41, p<.001). The linear trend was not significant (F<1). The mean unhappiness score for each religiousness category is presented in figure 1. Once again, the slightly religious respondents were less happy than either the very religious or the antireligious ones.

DISCUSSION

Most of the women in this study seem to endorse what James (3) called healthy-minded religion. They characterized God as loving, benevolent, and nonpunitive. Many respondents said they had experienced supernatural forces directly, and their experiences have been overwhelmingly positive. The respondents believed they should tell their children about God's love, but the overwhelming majority did not want to tell them about being punished by God for misbehavior. This religious orientation was presented as personally beneficial; among the women who were religious at all, greater religiousness was associated with greater self-reported health and happiness.

Converts were similar to nonconverts in some ways but were also different in important respects. In general, converts were currently more religious than women who had retained their childhood beliefs, and they appeared to have gained in health and happiness as a re-

sult of conversion. Why did they convert? Two kinds of reasons are suggested by the data. First, converts were less happy and more lonely than nonconverts during childhood and were also less religious. After conversion they were reportedly happier, less lonely, and more religious than nonconverts. They may have converted specifically to solve personal problems, perhaps to reduce the kinds of conflicts and uncertainty discussed by James (3) and Berger (2). Second, converts were somewhat more likely than nonconverts to have been discipline problems as children and were currently more likely to adopt an authoritarian approach to religion. This, too, suggests that certainty was their goal, but a particular form of certainty that may not be necessary for other adherents of healthy-minded religion.

In terms of Berger's typology (2), converts (compared with nonconverts are like the neoorthodox adherents of the deductive option; they seek authority for their beliefs beyond experience and may be somewhat intolerant of alternative beliefs. Nonconverts are more like Berger's third type, who take the inductive approach to religious experience. They benefit from confidence in the existence of transcendent experience but are tolerant of alternative interpretations of that experience. Following directions from their leaders is not so important as following the dictates of their own conscience.

The curvilinear relationship between religiousness, on the one hand, and health and happiness, on the other, is especially interesting. The similarity between those who are very religious and those not at all religious supports a hypothesis, derived from James (3), that certainty of belief or lack of conflict is more important to well-being than is religion per se. The more recent literature on cognitive consistency (5, 6) suggests that a person holding two or more conflicting beliefs suffers from aversive feelings of tension or dissonance. The slightly religious respondents seemed to be suffering from such dissonance; they were religious but ambivalent, perhaps for the reasons discussed by Berger (2). The curvilinear relationship between religiousness and health suggests that religious ambivalence has measurable aversive consequences.

Our research leaves the following important questions unanswered.

1. These data were provided by self-selected women. Would the conclusions hold for a representative sample of men and women?

2. Are the self-reports of health valid? We focused on mental health, knowing that it is easily affected by optimism, conflict, and defensiveness. In future studies, it would be worthwhile to assess mental and physical health more objectively (e.g., from medical records).

3. How, exactly, does religion contribute to personal well-being? Is certainty of beliefs the important issue, or membership in a caring community, or some feature of religious experience? We have focused on certainty of beliefs, but other factors may be equally important.

4. Why do nonreligious respondents report good health and subjective well-being? Is this the result, too, of certainty of beliefs concerning a nonreligious way of life?

Our data on converts alert us to the possibility that the correlates of religious experience per se, e.g., the determinants and consequences of the experiences of modern charismatic Christians, may be distinct from the correlates of *conversion* to an experience-oriented group. Converts appear to be different from nonconverts in religious background, childhood happiness, and authoritarian tendencies. Future studies should look carefully at the issue of authoritarian traits; converts to various religions may differ in the degree to which they are looking for authority instead of, or in addition to, transcendent experiences (11). This factor may help explain why a particular person joins one cult rather than another or chooses an esoteric cult rather than one of the more familiar American religious groups. The search for certainty takes more than one form (2), and these forms may have different social, psychological, and political consequences.

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Tenacity of Attachment to a Cult Leader: A Psychiatric Perspective

BY ALEXANDER DEUTSCH, M.D.

This follow-up study describes a cult whose leader became cruel and increasingly bizarre. Six of 14 members who were investigated at the time of the cult's formation remained loyal to the leader until the dissolution of the group almost four years later. The author describes the attitudes of these and other devotees who submitted to the irrational guru. The leader's radical teachings, his purported closeness to God, and his embodiment of a countercultural ideal of freedom appeared to be factors that bolstered his overwhelming authority in his followers' lives. The devotees used denial, rationalization, and other defensive operations to maintain the fantasy that the leader was acting for their benefit.

What ties bind members of a group to a disturbed, megalomanic leader? This question, occasionally addressed in the older psychiatric literature (1, 2), is once again timely, in part because of the cult phenomenon and its bizzare and tragic offshoot in Guyana. In this paper I will approach the question by describing and analyzing developments in a cult that I followed from its inception in 1972 to its dissolution almost four years later. The leader of this group was at first the epitome of parental benevolence; later he became unpredictable, cruel, and increasingly disturbed. Nonetheless, many of the group's early members remained his faithful followers until he abdicated leadership in 1976. In another paper (3), I discussed the psychological rewards accruing to the devotees during the cult's early history. These included the feeling of acceptance, the development of purpose, and the shoring up of defenses through adoption of the leader's philosophy of renunciation. In this presentation I will deal with the more paradoxical question of what motivated devotees to remain in a group whose central figure had become cruel and clearly irrational. Two questions are addressed: 1) What was the basis for the leader's continued attraction? 2) How did his devotees deal psychologically with the evidence of their leader's sadism and irrationality? Before presenting observations on these matters, I will briefly describe the history of this Hindu-style cult and its leader, Baba.

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BABA AND THE FAMILY

In September 1972 Baba, an American in his mid-30s who had recently returned from India, began sitting daily on a park bench in mid-Manhattan. He graduallly accumulated a sizable number of followers, who called themselves "the Family." The leader addressed his devotees in a sign language of his own invention. He showed considerable warmth toward the group as he and the Family ate, sang, and meditated together. Baba preached the philosophy of "letting go"; i.e., one should detach oneself from desire, ambition, worry, guilt, and one's sense of individuality to experience peace and unity with God. In January 1973 the guru and 30 disciples left the park in a bus the group had purchased.

Later that year, after much travel, the Family settled on a mountaintop in a northeastern state and formed an agricultural commune led by Baba and devoted to the worship of Baba's Indian mentor, "Swami." The communal property, called "the Hill," was visited by people from a nearby university community, several of whom stayed. During the next two years approximately 30 followers lived on the Hill, and the total number of Family members exceeded 100. Membership was totally informal. No vows were taken; people arrived and left the Hill as they pleased; and no one was coerced to remain in the group.

Following the departure from the park bench, Baba became progressively more bizarre, inconsistent, cruel, and domineering. He used $1,000 of the group's meager resources to buy himself a Jacuzzi bath in spite of the fact that there was no electricity on the Hill. He hit his devotees repeatedly, and one was said to have suffered a broken eardrum from Baba's assault. He publicly subjected some women to forms of sexual abuse and reportedly engaged in private sexual activity with certain female devotees. Except on the rare

occasions when he designated individuals for coupling, he grew progressively more prohibitive of his followers' heterosexual activity. Toward the end he became increasingly withdrawn and for weeks at a time would see only his chief assistant for brief daily visits. The ever-rarer occasions when the leader showed his old playful benevolence were greeted with relief and joy by his followers.

Clinically, Baba showed increasing evidence of psychiatric deterioration. When I last saw him, in 1975, he was grimacing bizarrely and his sign language had become so fragmented and explosive that his devotees could no longer follow it.

In late 1975 Baba asked for a cassette player; he said he wanted to listen to a playback of his voice to see if he were still alive. He fiddled with the dials of the instrument brought to him and heard an evangelical Christian program that spoke of routing the devil from one's heart and replacing him with Jesus. Baba decided that Swami was the devil who had to be exorcized from his heart and from the commune. In the following months Baba led the Family in frenzied rituals of exorcism, the burning of all relics of Swami, and baptism into fundamentalist Christianity. He then made it clear that he no longer wanted followers; this led to the virtual dissolution of the commune and the fragmentation of the Family.

THE DEVOTEES

In my previous study of this group, I reported on 14 of the guru's early followers. Of middle socioeconomic class origin and mainly in their 20s, they had been immersed in the drug counterculture and most were dropouts from college, graduate school, or serious employment. With one exception, they complained of long-standing dysphoria and demonstrated higher than average character pathology and symptomatology. Four had schizotypal features, but psychedelic drug abuse confounded precise clinical diagnosis of 3 of the 4. Another subject appeared to be a manic-depressive in remission, while still another had been hospitalized for depression in early adolescence. Oral-dependent features were prominent in the group of subjects, as was the complaint of being oppressed by the demands, restrictions, and criticisms of parents and society. "Magical-mystical" beliefs were common, and most reported that their use of psychedelic drugs had influenced their turn to spirituality.

A brief follow-up report on these 14 subjects will give some indication of the tenacity of their affiliation with Baba. Three of the 14 left the Family before its departure from Manhattan in 1973; 11 continued to be devotees beyond that point. Three of these 11, inspired like other members to visit Swami in India, became exclusively involved with Swami and detached themselves from Baba. Two more left Baba after the establishment of the commune; in both cases, the guru's excesses had influenced their decision. Six of the 14 remained loyal to Baba until the Family upheaval that began at the end of 1975. These six who remained with Baba had not, on the average, demonstrated notably greater pathology initially than those who left.

THE PRESENT STUDY

All 6 who remained loyal to Baba were seen in follow-up; I interviewed 5 of them on repeated occasions during 1974 and 1975 with a view to ascertaining their attitudes toward their leader. I also interviewed 15 other individuals who joined the Family early in its history and remained faithful to Baba but who were not subjects in the original study. Most of the interviews were conducted on the Hill, where I spent 12 days in the course of 4 visits. While there, I observed interaction among the group and Baba's behavior. I also saw members in New York and during a visit I made to India.

It became apparent that the loyal devotees held quite similar attitudes toward Baba and responded to him in much the same ways. In the section on affirmative attitudes I will describe the shared thoughts and feelings about the guru that seemed vital in motivating the loyal devotees to remain with him. In the following section on defensive attitudes I will delineate ways in which the devotees seemed to ward off recognition of Baba's destructiveness. It should be mentioned that in addition to the all-important feelings directed toward the guru, which are the focus of this paper, members also expressed feelings of attachment to each other and to the mystic universalist spirituality preached on the Hill and viewed the commune as a cozy haven in a disturbing world.

ATTITUDES OF THE FAITHFUL TOWARD BABA

Affirmative Attitudes

1. *Baba as a benevolent leader.* Family members who remained loyal to Baba continued to assert that he was an unselfish leader motivated by a wish to aid his followers. They stated that following Baba's path was now a "hard trip," requiring much faith and fortitude, but that the rewards were correspondingly great.

2. *Baba as teacher.* The lesson of letting go, drummed into them unremittingly by the guru, was regarded as crucial wisdom by the faithful. This teaching appeared to have a powerful effect on their personalities and outlooks. It served to distance members from psychic pain and conflict arising from a variety of sources (e.g., narcissistic wounds, anger, sexual embarrassment) and ultimately to enhance their tie to Baba.

One subject met the Family after doing poorly on his examinations in law school. Baba helped him let go of his ambition and desire for wealth. He left school and became a devoted follower.

3. *Baba as God's agent.* When a decision had to be made, Baba would spin in a circle in a strange and dramatic way. He used the direction his nose pointed to or the object on which it landed at the end of the spin as a sign indicating how the Family should proceed. To the devotees these episodes meant that the guru had abolished his "ego" to obtain direction from God.

4. *Baba as a model of freedom.* In line with the notion that Baba obtained direction from God, devotees regarded their leader as totally free from socially imposed restraints, a trait they much admired. Baba seemed to do pretty much as he pleased; and indeed, his "crazy" behavior was often the source of amusement and awe. Family members particularly delighted in seeing Baba triumph over authority figures by outsmarting them or confusing them with his "craziness."

Baba's attitudes toward conventional authority proved to be quite influential. This could be observed when he encouraged Family members to obtain disability benefits for mental illness in order to increase the commune's inadequate income. Some devotees then cleverly exaggerated their symptoms during psychiatric examinations and qualified for "crazy money" (as it came to be called).

5. *The virtue of surrender.* While the guru may have represented an embodiment of freedom, the devotees were far from free in their relationship to him. A paradoxical although central development in the history of the cult was the shift in Baba's position from guide and benign "superego replacement" (3) to frightening and controlling authority figure. This change corresponded to a shift in connotation of the group's central principle of letting go and of another, related precept of "surrender to God." At first apparently meant by the guru to be guides to detachment and spiritual enhancement, these teachings became synonymous in the minds of group members with the principle of "surrendering to Baba." (Since Baba was considered a manifestation of Divinity, surrendering to him could readily be substituted for surrendering to God.) Indeed, surrendering to Baba became the central spiritual goal of Family members. Devotees reported experiencing ecstasy when hit by the guru. It was commonly felt that the guru's favorites were those who submitted most completely to him.

The shift in Baba's leadership role from permissive guide to stern authority was notable in the area of sexuality. At first he helped devotees rid themselves of sexual guilt as dictated by conventional morality (3). He then began to encourage certain pregenital group activities such as "gropes" (members huddling together and fondling each other) and public nudity, which became the norm on the Hill. (A number of members had to let go of inhibitions to participate in these practices.) Ultimately, he caused members to be terrified that any private heterosexual activity would incur his displeasure or punishment.

6. *"Baba takes on our karma."* Another prominent notion was that the guru took on the Family's suffering for misdeeds and, in this way, spared the members.

In the latter days of the commune Baba would walk into the temple backward because he suffered so deeply from the "bad vibes" produced by his disciples' holding on to anger and tension.

Defensive Attitudes

The group employed loaded language (4) and magical interpretations in conjunction with denial, rationalization, and turning against the self in their efforts to preserve their tie to Baba in the face of his irrationality and abuse.

1. *Divine madness.* Few objected to the notion that Baba was "crazy." The manifestations of the "craziness" were dressed up in Hindu metaphor, continually referred to in spiritual terms, and evidently regarded as signs of Baba's closeness to divinity. Thus, Baba's grimacing and bizarre episodes of unrestrained spinning were seen as indications of the rising of the Shakti (divine energy). When he showed kindness he was said to be in his Krishna phase (after the divine incarnation representing love), and his rage was rationalized as being a manifestation of Shiva (the Hindu god of destruction).

2. *"Baba is teaching us a lesson."* This was the ultimate defensive explanation by means of which Baba and Family members could justify any act of cruelty, sexual abuse, or bizarre inconsistency. The explanations were invariably tied in with the group's central precepts. Thus, a woman sexually abused by Baba was holding on too tightly to her chastity, and the wasteful spending on the Jacuzzi was meant to teach people to let go of money. Once the notion of surrender to Baba became a spiritual goal, any abuse, particularly if unprovoked, had its built-in justification, i.e., "he is teaching us to surrender."

3. *Denial and naiveté.* The Family often disregarded obvious facts in the service of justifying Baba's actions. Shortly after an abused woman left the Family, I was told, "Baba takes you where you want to go—he would not push anyone too far." In addition, Family members showed surprising naiveté.

Baba gave three members the privilege of signing checks for the Family. Certain devotees attributed the privilege to the intensity of spiritual surrender by the three members and ignored the fact that two of the three were the chief financial supporters of the group.

4. *Closing one's eyes.* Many devotees employed this concrete method of shutting out reality during

meetings with Baba in 1975 when the guru was fearsome, accusatory, and incoherent. One devotee said that he kept his eyes closed because he would otherwise try too hard to figure out what Baba meant, and another because he would take the scolding too personally. One might also assume here the employment of the magical notion, "If I don't look at him, then he won't see me."

5. *Magical interpretations.* These were commonly used in the service of preserving the tie to Baba.

> After much inner conflict over his allegiance to Baba, one devotee left the commune. He found himself stranded on the road in freezing weather, and in despair he prayed to Swami. Soon after, a car appeared heading in the direction of the Hill. He interpreted this as meaning that God meant for him to go back to Baba, and he waved the car down and returned to the Hill.

6. *Turning against the self.* It appeared that an important way of dealing with potential anger stirred up by Baba's abuse was to turn it against the self in the interest of preserving the tie to the guru. A consequence of this mechanism would be increased submissiveness and self-abasement (5, p. 363), as the following vignette suggests.

> One devotee said he was hit 20 times by Baba. He accepted this as a message of love and prostrated himself in the nude on the snowy ground in gratitude to his guru.

COMMENT

A cult can be defined as a relatively stable, transcendentally oriented group surrounding a powerful central figure who influences his or her followers in a direction that deviates strongly from that of the dominant culture. Elaborate strategies of persuasion, enforced isolation from outside influences, and threats to those who might leave have all been implicated as factors responsible for the power of certain modern cults over their members. This study demonstrates that profound submission of cult members can occur in the absence of these factors. The following explanatory and theoretical considerations, based on observations of the Family reported here and elsewhere (3), are advanced to account, at least partially, for this group's submission to its irrational leader.

The strength of the early bond. The needy devotees developed a child-like dependency on the guru during his original benevolent Krishna phase. He was perceived as close to God and fulfilled a multiplicity of roles for them: love object, nurturer, guide, soother of consciences, and model (1). The Krishna behavior proved to be a powerfully seductive force.

The appeal of madness. Baba's bizarreness seemed vital to his charisma. I would suggest that in his "madness" he gave full expression to tendencies admired but only partly realized by his followers. These tendencies, evidently products of both personal predilection and involvement in the psychedelic counterculture, included mystical longings, antirationalism, antagonism toward conventional authority, and the wish to feel free from its restraints (6, 7). Baba, with his mystic trances, flagrant irrationality, and forthright antinomianism, seemed to embody for them their unusual ideals. Individuals who personify a shared ego ideal tend to be nuclei for group formation (1, 8, 9).

A vacuum in guiding values. Following their attraction to Baba, the gap between these alienated young people and the values of the dominant culture was widened further by the guru's radical teachings. His abuse and accusations were doubtlessly destructive to their sense of self and hence to any residual internalized values (10). The followers would have little recourse but to turn increasingly to the guru for a guiding value system.

Regression in the group. The profound regression Baba's followers underwent was shaped by their membership in a group (2, 11, 12). It is useful to look at sexual life in the cult from the perspective of group psychology. Baba made the rules for sexual behavior in the Family. He encouraged nudity and public pregenital activity on the part of the members while exhorting them to divest themselves of shame. He tended to permit himself heterosexual liberties while restricting the members. It was as if the Family had little room for more than one male sexual adult; the situation is somewhat reminiscent of Freud's "primal horde," which he postulated as the prototypical authoritarian group (11). Baba's instructions regarding sexual pairing and privacy would not only act directly to infantilize his followers but also would strengthen their bonds to the authoritarian group and thereby potentiate their regression (9; 11, p. 140).

The need for the omnipotent object. It is curious that the devotees (like some other converts [13, 14]) felt oppressed by and antagonistic toward authority figures and then became involved in an authoritarian situation more totalistic than their earlier ones. One can conjecture, following Salzman (13), that at least for some of the devotees, this very conflict with authority influenced them unconsciously to seek out their "omnipotent," "all-good" leader, whose power offers him immunity from destruction and who can therefore provide them protection against their own hostility (15). Ambivalence toward the new authority would be a threat to this magical protection and call forth an increase in idealization (e.g., the attribution of divine qualities to his "madness"), rationalization ("he is teaching us a lesson"), or the turning of the hostility against the self.

I have focused on the devotees who remained loyal; there were also a number who became disillusioned with the leader and left the group. A systematic, comparative study of those who remained and those who

left this group (or similar ones) could lead to an improved understanding of the characterological and situational factors that might result in the submissive and self-deceptive behavior that has been discussed.

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Psychological Induction Into the Large-Group: Findings from a Modern Religious Sect

BY MARC GALANTER, M.D.

The author studied the psychological aspects of religious conversion during structured 21-day workshop sequences designed to introduce people to the Unification Church. Subjects were given a battery of tests at different times during the sequence. After the initial 2-day workshop, 71% dropped out; the 29% who chose to continue had greater affiliative feelings toward the group and greater acceptance of the church's creed than these early dropouts. The 9% who ultimately joined the church had weaker outside personal ties than the later dropouts, although their beliefs in and cohesiveness toward the church were the same as the late dropouts. These results are also compared with long-standing members of the church and matched nonmembers. The induction procedures used by the church are discussed with regard to those used by large therapeutic groups, such as self-help organizations.

In the last decade charismatic religious sects have attracted a considerable number of followers in the United States. The results of a study on the psychological issues relevant to decisions made by a sample of young adults about joining one of these sects, the Unification Church (or as its members are called, the "Moonies") are reported in this paper. We had previously studied the psychological traits of persons who joined charismatic religious sects; these were studied by means of objective tests (1-3). Similar approaches have been used for persons who left various religious cults (4). For two reasons, however, it seemed useful to consider a group of individuals making their initial contacts with a charismatic religious sect. First, the psychology of the individuals themselves is important. The characteristics defined thus far in sect members do not necessarily differentiate them from many persons who have never joined a sect. If those who consider joining a sect were evaluated, it might eventually be possible for us to differentiate between persons who finally elect to join and those who do not. Second, these sects dramatically illustrate certain aspects of group dynamics. For example, responses evinced by them bear similarity, in certain respects, to the responses evoked by many of the therapeutically oriented self-help groups (1). Therefore, by studying the induction process of these sects we may obtain valuable information on the mechanisms by which affiliation is established in large therapeutic groups. This may then enhance our understanding of psychologically oriented group treatments.

An initial study (3) was made that examined the attitudes of Unification Church members, including an examination of their experiences around the time of joining the church. When it became desirable to study the church's induction procedures, the sect's officials subsequently granted further interviews and site visits to clarify the operation of their "workshops." These officials expressed the opinion that an objective study of the induction procedure might serve to demystify the process of joining, which some critics have referred to as "brainwashing." The process examined in this study was not necessarily typical of the procedures used by the church in all parts of the country, but it reflects procedures used in late 1978 at workshops serving a number of areas, including Boston, New York City, Washington, D.C., and southern California. The actual sample was drawn from the latter location.

METHOD

A full workshop experience runs 21 days, as illustrated in figure 1. Eight workshop sequences were studied over a 2-month period. There were 104 persons who attended one of these workshop sequences. A description ensues of the workshop sequence and the disposition of these persons. Because the interactional context of the workshop may not be familiar to the reader, an attempt has been made to convey the nature of the group context in some detail so that asso-

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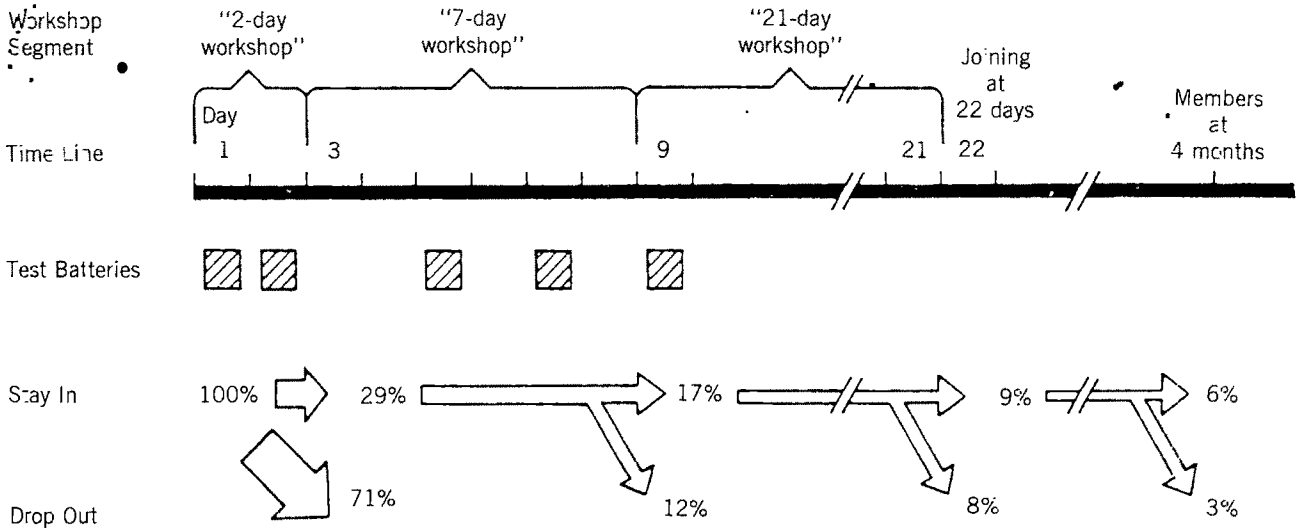
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FIGURE 1
Workshop Sequence, Testing Schedule, and Dropout Rates of 104 Subjects Attending Unification Church Workshops



ciated psychological responses will be more understandable.

People who attend the initial "2-day workshop" are generally invited by a member of the church whom they may have met in a public place while that member was "witnessing," i.e., recruiting for the church. The motivation of persons attending the "2-day workshop" may be limited, as may be their understanding of the church's religious creed. Prospective members usually leave for the Workshop Center from a large city or academic campus on a Friday evening. The Workshop Center is usually in a secluded rustic setting, a few hours from the city of debarkation, as it was in this study.

The schedule for the next 2 days is filled with planned activities from 8:00 a.m. to 11:00 p.m., including three meals a day. The principal activity is a series of six 1½-hour lectures, which convey the most salient points of the "Divine Principle,"—the religious doctrine of the Unification Church. Each lecture is followed by a ½-hour small-group discussion. The discussions and some other sharing experiences are more oriented toward intellectual exploration of religious concepts than emotional catharsis; they do, however, delve into personal attitudes and experiences. The remainder of the time is filled with group activities, typically singing, sports, and preparing and giving skits. Participants, or "guests," spend most of their discussion time in groups with 2 or 3 other guests, a leader who is a church member, and the 3 or 4 "hosts" who originally invited each of the guests to the workshop. Virtually all the guests' time, therefore, is spent either in small or large groups where more than one-half the individuals are active members in the church. In the evening of day 2, following discussions of their continuing the workshop sequence, guests are encouraged to stay at the Workshop Center for the "7-day workshop" (which actually runs 6 days). As indicated in figure 1, 74 (71%) of the guests, designated as "early dropouts," did not continue beyond the initial weekend. The status of each guest was reviewed by me with the workshop staff after each weekend. Two of the guests leaving before the second workshop had been taken away by family members who came to the Workshop Center, as were two more later on in the 21-day sequence.

During the second segment, the "7-day workshop," breakfast begins at 8:00 and is followed by a 3-hour lecture and discussion; lunch is followed by 2 hours of sports, hiking, or working at the Workshop Center. In late afternoon there is another 2-hour lecture and discussion, which is followed by dinner and further sharing and discussion. The day ends with the guests filling out their "reflection notebooks"; they may choose to discuss their entries in these notebooks with the larger group.

Persons participating in the "7-day workshop" engage in considerable discussion and introspection regarding their interest in the Unification Church. Workshop staff, however, also evaluate the guests' suitability for membership in the group primarily, as they describe, to exclude persons who lack the psychological stability and maturity for the arduous and dedicated lifestyle. Thus, by the end of day 8, the majority of the remaining guests elected to stay on beyond the initial week for the next segment. Of those who left, 3 had been asked to do so after extensive discussion among the staff. During the ensuing segment 3 more were asked to leave.

This next segment, the "21-day workshop," consists of 1 week that is dedicated to fund raising, and a second week to "witnessing." In this study the "21-day workshop" was conducted in Los Angeles and its environs. This phase has a somewhat more rigorous

daily schedule. Guests arise at about 6:00 a.m.; most of the day is directed toward intensive public fund raising or recruiting, for which the group is well known. In addition, there are prayers, lectures and some group activities around meal times. The day ends before midnight after a period of "sharing" and filling out the "reflection notebook." During the day workshop guests mix with people who are not in the Unification Church, although their compatriots are generally nearby.

At the end of the "21-day workshop" guests may elect to move into a communal Unification Church residence, as did 9 of the sample (designated "joiners"). Application forms may be filled out, but this formality can also take place before or after they move into the church residence. Church members often move at this time to remote parts of the country for fund raising, recruiting, or other church activities, and it can be difficult to track new members. The new members were followed, however, through contacts with the state membership directors, the original workshop coordinators, and the central membership office of the Unification Church in New York.

It was possible to ascertain the status 4 months later of the 9 persons who had joined after the 21-day workshop sequence. Of these, 6 were still active members. Of the 3 who had left the group after joining, 1 chose to return to his family who lived overseas; 1 was reported to have been abducted while visiting his aunt's home on the way to another church Center, and 1 was felt to have been "not serious" in his commitment.

Test Battery

In order to examine the psychological experience of the workshop guests a test battery was formulated utilizing some of the approaches previously employed in our studies on group cohesiveness and religious beliefs (1-3). In addition, by means of interviews and subsequent pilot studies with church members, additional scales were developed to address issues relevant to the psychological state of workshop guests. Demographic and background items, as well as the General Well-Being Schedule, were administered in the first 2 days of the sequence. With the exception of the general well-being scale, the scales described below were given as part of a repeating battery on days 1, 2, 5, 7, and 9, as indicated in figure 1.

General Well-Being Schedule. A 16-item scale for general psychological adjustment was developed by the National Center for Health Statistics (5). Its background and the derivation of an age- and sex-matched comparison group not affiliated with the Unification Church have been discussed previously (3). Typical items, each with a choice of scaled responses, are "How have you been feeling in general?" "Have you been bothered by nervousness or your nerves?" "Have you been feeling emotionally stable and sure of yourself?"

Cohesion Scales. These employ eight cue sentences, developed for use in previous studies (1-3, 6), that reflect different aspects of social affiliation such as, "They care for me," "They are suspicious of me" (scored in reverse), and "I like being part of their activities." Responses are made along a 5-point scale ("not at all" to "very much") in relation to two target groups. The first group is composed of "the ten or so people from the workshop you know best" from which is generated a score for affiliative feelings toward persons inside the workshop group ("affiliations inside"). Responses toward the second target group, "the ten or so people outside the Church and workshop you know best," generates a score for "affiliations outside" the group.

Creed Scale. The Divine Principle is a compilation of the sect's religious creed. Six items of creed were chosen from the Divine Principle that were specific to the Unification Church; they were also presented to the guests during the workshop sequence at the points indicated in figure 1. Guests were asked to use the same 5-point scale noted above to indicate the degree to which they "agreed with each Unification Church belief." Examples, which may sound idiosyncratic to one unfamiliar with the sect, are "Some of the problems I see around me began with an immature use of love by the first human ancestors," and "My understanding of Jesus' mission to restore God's Ideal of Creation will lead to a more meaningful life for me."

Sense of Purpose Scales. Six cue sentences each reflect some aspect of an existential sense of purpose. Samples are "I feel that my life can have a meaning," "I can experience worthwhile relationships with others," and "My life can really make a difference." Guests were asked to use a 5-point scale to indicate their own sense of purpose, yielding a score designated "own purpose." They were then asked to indicate how much the Divine Principle contributed to their feeling for each item, generating a "purpose/Divine Principle" score.

Group leaders and workshop coordinators were rehearsed in the format of administrating the tests, and questions that might be asked by the guests were reviewed. Weekly exchanges with workshop staff were also arranged to deal with problems that arose. The tests, or questionnaires, were prepackaged in envelopes for each small workshop discussion group; instructions were attached to each envelope and were read to the group by the group leaders and coordinators.

A record was kept on the face of each envelope of those who had completed the questionnaires, but the questionnaires were kept anonymous. The following system was used to allow for tracking of each subject's questionnaire. Subjects selected a 5-letter work and a number as their personal code, which they inscribed on all their questionnaires. After each workshop sequence it was therefore possible to link a series of each

TABLE 1
Psychological General Well-Being Schedule (5) Scores of Long-Standing Members of the Unification Church, a Nonmember Comparison Group, and Nonmembers Attending Church Workshops

Group	General Well-Being Scores		Comparison with Long-Standing Members	
	Mean	SD	t	Significance
Long-standing members (N=237)	74.4	17.2		
Nonmember comparison group (N=305)	83.4	16.2	6.24	p<.001
Guests attending workshop				
Dropouts after "2-day workshop" (N=74)	74.7	17.9	.28	
Guests who continued beyond "2-day workshop" (N=30)	67.2	19.2	1.95	p<.05
Guests who joined after 21-day workshop sequence (N=9)	61.9	20.1	1.89	p<.05

guest's questionnaires that reflected how long each guest remained in the workshops. Although this allowed for anonymity and fostered frank responses, it did not allow for designating questionnaires from those guests who were either asked to leave or were picked up by their families. On the other hand, in order to differentiate between those guests who joined after completing the entire workshop sequence and those who did not, the joiners were asked at the end of the 21-day sequence to reveal their respective codes. It was explained that the information would be used solely to segregate them from the latter two groups. All agreed to do so.

RESULTS

Because some test items described above were used previously in our study of long-standing members of the Unification Church (3), it was possible to compare the guests from this study with long-standing members (N=237). On the whole, the groups were quite similar in demographics and background. The workshop guests who responded (N=104) were predominantly male (77%) single (95%), and white (77%). Comparable figures for the long-standing members in our previous study were 61%, 89%, and 83%, respectively. The guests' mean age (±SD) was 21.6±3.4 years; this was .3 years younger than the long-standing members had been when they joined. Eighty percent of the workshop guests had both parents living; 79% of the long-standing active members had both parents living. The workshop guests also did not differ significantly from long-standing church members in their assessment of the degree of religious commitment their parents had. The workshop guests and the long-standing members indicated that 74% and 78%, respectively, of their fathers had no more than moderate religious commitment and 57% and 60%, respectively, of their mothers had no more than moderate religious commitment.

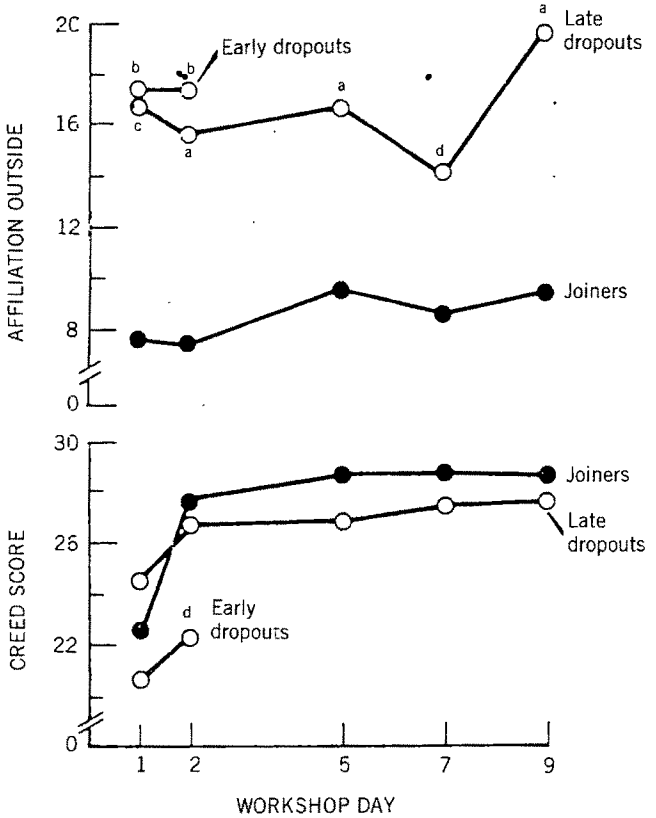
Scores for both groups on the general well-being scale are listed in table 1; they reflect the workshop guests' emotional state during the month before testing. As reported previously (3), long-standing members had significantly lower scores than the matched comparison group (N=305). In the current study, workshop guests who continued with the workshop sequence after day 2 (N=30) scored significantly lower than the long-standing members; the guests who finally joined scored lower than either group.

Results of the repeated battery were examined next using one-tailed t tests to compare guests who dropped out at the end of each workshop segment to those who did not. The early dropouts, those who left by the end of day 2 (N=74) had mean scores (±SD) lower that day on the affiliations inside scale than those who continued attending the workshops (20.2±5.7 and 24.0±5.3, respectively, t=3.01, p<.005). They also scored higher on the affiliations outside scale (17.6±6.0, 13.4±6.9, respectively, t=2.91, p<.005).

In the previous study of long-standing members, the same scale items had been used to demonstrate affiliations inside the sect, i.e., "the ten members of the Unification Church you see the most." Responses of those members can therefore provide some basis for comparison. The workshop guests who continued beyond the second day (N=30) scored as high on the cohesion scales as long-standing members (mean score, 23.7±3.9), even as early as day 2. The early dropouts, on the other hand, did not (t=5.75, p<.001). Early dropouts also scored lower on the creed scale than those who continued (mean score, 22.3±7.2 and 26.0±6.0, respectively, t=2.39, p<.01) and the purpose/Divine Principle scale (mean score, 20.9±8.6 and 25.6±6.1, respectively, t=2.61, p<.01). There was, however, no significant difference in scores on the own purpose scale between the early dropouts and those who remained.

The 30 respondents who continued beyond the second day and completed the test batteries were divided into two groups: those who ultimately joined at day 22 (N=9), designated as "joiners," and those who dropped out between days 3 and 22, the late dropouts (N=21). The cohesion scale scores of affiliations outside the church were compared, as illustrated in figure 2. On the cohesion scales there were no significant differences at any of the test points between the two

FIGURE 2
Outside Affiliations and Creed Scores for 104 Subjects During a Unification Church Workshop Sequence



[a]Significant difference between dropout groups and joiners, p<.01.
[b]p<.001.
[c]p<.005.
[d]p<.05.

groups on the inside affiliations scale, but there were significant differences at all points on the outside affiliations scale; the early and late dropouts scored significantly higher on the outside affiliations scale than the joiners. For example, mean scores on day 2 for the joiners and late dropouts on the inside affiliations scale were 25.3±4.7 and 25.0±3.8, respectively; on day 9 they were 25.5±3.1 and 26.3±2.9, respectively. The outside affiliations scores on day 2 were 15.8±6.6 and 7.4±6.2, respectively, t=2.80, p<.01; on day 9 they were 20.0±4.06 and 9.3±7.2, respectively, t=2.99, p<.01. In addition, the joiners and the late dropouts were not significantly different at any of the test points for creed, own purpose, or purpose/Divine Principle scales.

DISCUSSION

In a previous paper (1) I discussed the therapeutic large-group as a clinically useful model for the study of certain clinical phenomena, such as self-help groups and membership in charismatic sects. The large-group was defined as a cohesive association of a large number of persons who often meet in small groups and espouse a zealous philosophy with attendant rituals. The social roles and behavioral norms of group members were redefined in relation to the group's goals and functions. The model was developed from the examination of one particular sect where social cohesion was studied with questionnaire instruments similar to those employed in the present study. Comparisons were drawn to other sects and quasi-therapeutic self-help groups, such as est and the drug-free therapeutic communities. An ensuing study on the Unification Church (4) dealt with the regulatory role of the belief system as well. The present study now provides an opportunity to examine the process of affiliation to such a group. This is of interest because it reflects a psychological phenomenon of considerable potency in light of the profound influence such a group may exert and the potential adaptability of this model to psychotherapeutic ends.

How were the people who joined the Unification Church different from those who dropped out? The early dropouts were found to differ on the basis of weaker affiliative ties toward the group and less acceptance of the church's religious beliefs in contrast to those who stayed beyond the initial workshop segment. Late dropouts, however, showed a high level of affiliativeness toward fellow workshop members, which was comparable to that of the longstanding members toward their compatriots. They also achieved a commitment to the sect's beliefs comparable to the guests who ultimately joined. The later dropouts, however, were different from those who joined by their greater affiliative ties to people outside the sect. This appears to indicate that an important factor in joining the sect is the presence of relatively weak ties to family and peers. Apparently, at least at this stage in the affiliation process, considerable enthusiasm for the group may be countered by the strength of outside ties.

It is interesting to compare these findings to ones reported by Trice (7), who studied affiliation motives in members of Alcoholics Anonymous (AA), another quasi-therapeutic large group where consensual belief is important. Persons who maintained a long-term membership in AA, rather than dropping out after an initial affiliation, were found to have greater need to establish social affiliations. For a religious sect such as the Unification Church, however, a greater isolation from previous ties accompanies membership. The relative balance between affiliation to members and to old associates therefore becomes important.

In our previous studies (1–3), long-standing members gave responses to a neurotic distress scale in relation to multiple time periods. The responses indicated apparently greater neurotic distress before affiliation compared to neurotic distress after a period of membership. In the absence of prospective data, however, it was not possible to ascertain whether this reflected

an actual difference between these two periods or whether it was primarily the product of unintentional distortion due to cognitive dissonance. Although the number of joiners in this study was small, it seemed worthwhile to compare the general well-being scores of those who ultimately joined to those scores of the long-standing members. Significantly lower mean general well-being scores were found among the new joiners than the long-standing members. This supports the interpretation that members actually do experience amelioration in psychological well-being long after joining.

Members and workshop guests scored lower than a comparison group for psychological general well-being. The comparison group was drawn from a residentially and socially more stable population than that usually associated with persons entering this sect, although the comparison group was matched for age and sex. The difference in scores might therefore be partly attributed to the considerable amount of social disruption experienced by many persons who elect to come to the workshops. This latter phenomenon merits further investigation because it may provide a key to the role of social instability in valency to join a quasi-therapeutic large-group.

This explanation, however, may be contrasted with the possibility that considerable psychopathology unrelated to situational issues is common in sect members. Indeed, a global solution may appeal to the psychologically distressed. Deutsch (8) examined in detail the presence of prominent psychopathology in members of a smaller Eastern sect. In addition, we had found that 6% of the members from the Unification Church and 9% from the Divine Light Mission had been hospitalized for emotional problems (3, 2). In this study the workshop staff, apparently respecting the vulnerability of some guests, asked 6 of the 30 guests who stayed beyond the "2-day workshop" to leave because of "psychological instability." The staff accepted the fact that not all those interested in joining were suitable for membership. It should be noted, however, that an apparent therapeutic response has been observed for those who join other modern self-help-oriented large-groups, such as est (9, 10), consciousness-raising groups (11), Gestalt-oriented growth centers (12), as well as other religious sects.

In this study there was no significant difference between the joiners and the dropouts regarding an absence of existential meaning in their lives. For the potential member perhaps only a certain degree of absent meaning in life is necessary to elicit interest in the Unification Church. The church must present its belief system in such a way that the potential innate resistance is relieved and the creed can be accepted. To this end, the social context plays an important role to rapidly generate a strong sense of cohesiveness during the workshops, as observed here. This was promoted when the workshop guests continued through the initial workshop segment in close relationships with the hosts who originally invited them.

The milieu of the lectures and discussions is consequently influenced by the fact that Unification Church affiliates constitute half of the membership of the group. This presents an interesting parallel to the sponsorship system in AA. It also has antecedents in other Christian sects, such as the Moravian Protestants (13) and the Anabaptists (14), which employed group-oriented explorations of belief and feelings. Indeed, technique of mixing "successful" veterans and initiates in conjoint group experience for modeling and identification is regularly seen in contemporary self-help programs. It might also be useful in strengthening the induction process in more traditional mental health settings.

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BRIEF COMMUNICATIONS

Behavioral Desensitization of Phobic Anxiety Using Thiopental Sodium

BY THEODORE PEARLMAN, M.D., B.SC., M.B., B.CH.

The author evaluated a random sample of 25 patients suffering from a variety of phobic anxiety states who were treated with desensitization using thiopental sodium combined with progressive reduction in the use of oral anxiolytic medication. At 24-week follow-up, 11 of the patients reported mild symptoms of phobic anxiety and 14 reported none; 9 of the patients reported their use of tranquilizers had decreased and 15 reported they had used none for at least 2 weeks. These results demonstrate the usefulness of the treatment in producing sustained reduction of phobic anxiety and tranquilizer use.

The use of short-acting barbiturates administered intravenously to induce adequate relaxation is not a new practice in the treatment by desensitization of phobic anxiety. Wolpe and associates (1), in reviewing systematic desensitization of phobic patients by reciprocal inhibition, referred to the use of methohexital sodium in pairing adequate relaxation responses with a progressive hierarchy of anxiety-provoking stimuli situations. Mawson (2), in a controlled crossover trial, demonstrated 50% more effective improvement of phobic patients with the use of methohexital. Friedman and Silverstone (3) noted an economical reduction in treatment time when this technique was used.

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METHOD

Patients

Between 1971 and 1979, 10% of the more than 8,000 adult psychoneurotic patients referred to me for psychiatric treatment were diagnosed as suffering from phobic anxiety. Most of these patients came from the densely populated Witwatersrand area of the southern Transvaal, South Africa. The criterion for a diagnosis of phobic anxiety was a main complaint of fear, with or without autonomic nervous system symptoms, arising on exposure to one or more normal life situations. These situations included being in open spaces, confined spaces, traffic, or crowds; thunder or lightning; visits to dentists; college or school examinations; cancerophobia-producing situations such as visiting sick friends or relatives; and exposure to animals or insects. In many patients hyperventilation-producing cardiac or respiratory neurosis aggravated the phobic condition.

Most of the patients were taking anxiolytic drugs when first examined, mainly benzodiazepines prescribed by their family doctors for symptomatic relief. These included diazepam, oxazepam, medazepam, bromazepam, chlordiazepoxide, lorazepam, and clorazepate.

All patients except those with porphyria or a family history of the disease (a not infrequent illness encountered among neurotic patients in South Africa) were offered desensitization under the effects of intravenous thiopental. Patients who chose treatment and provided informed consent (about 70% of the more than 800 phobic patients) were instructed in the theory of classical and operant conditioning. A program of treatment spaced biweekly for 2 weeks and then weekly for 4 weeks was arranged for each. Patients were instructed not to take solid food for 3 hours before treatment, but

bland fluids were permitted. After each treatment session patients were required to remain in the waiting room until all sensation of dizziness had passed. Patients were warned not to drive until motor coordination was fully restored. No patient reported any motor vehicle accident involvement following treatment.

The Treatment Session

While the patient reclined in a comfortable position, 50 mg of thiopental sodium in a 2½% aqueous solution was injected intravenously in a single bolus. A twilight state lasting 4–6 minutes, during which the patient was mentally and physically fully relaxed but alert, was thus induced. A resuscitator and a cylinder of oxygen were available in case the patient was hypersensitive to thiopental. In no instance, however, was resuscitation necessary. With eyes closed, the patient, using visual imagery to the best of his or her ability, was encouraged to reconstruct previous attacks of phobic anxiety while aware of the thiopental-induced state of relaxation. The patient was then told to imagine an attack beginning sometime again in the future and to imagine being able to control, diminish, or eliminate the attack by exercising his or her feeling state.

Thiopental is rapidly metabolized; therefore, the state of drug-induced relaxation phased after 4–6 minutes into a more natural feeling state of relaxation. For the next 10–12 minutes the patient repeated the mental exercise of associating anxiety-provoking stimuli situations with the response of relaxation.

Patients were encouraged to reduce their oral intake of anxiolytic drugs when they felt that they could do so. They were instructed in a simple relaxation exercise based on Jacobsen's technique (4) and told to practice pairing imagined phobic situations with relaxed responses. The patients were to perform these exercises at least twice daily. If they were taking oral anxiolytic medication they were told to exercise at times coinciding with the maximal effects of the drugs when this was possible. For example, if a patient usually took a tranquilizer after coming home from work at 6:00 p.m., he or she would do the exercise at approximately 6:30 p.m.

Evaluation

I evaluated a random sample of 25 patients out of the 415 who completed 6 weeks of therapy and then complied with follow-up. At the initial interview (designated week 0) and at 6, 12, and 24 weekly intervals each patient rated his or her phobic anxiety on a scale of 0–3 (0=no symptoms, 1=mild symptoms, 2=moderate symptoms, 3=severe symptoms). In addition, ratings for tranquilizer use were recorded on a scale of 0–3 depending on quantitative comparison with the daily intake of the same drug when first prescribed by the referring physician (0=no use of drugs for at least 2 weeks, 1=decreased use, 2=no change in use, 3=increased use).

TABLE 1
Ratings of Phobic Anxiety in 25 Patients Treated with Thiopental-Assisted Desensitization[a]

Patient Number	Week 0	Week 6	Week 12	Week 24
1	3	1	1	1
2	1	0	0	0
3	3	1	1	1
4	3	2	1	0
5	2	1	1	1
6	3	2	1	0
7	2	1	1	0
8	3	1	0	1
9	1	0	0	0
10	3	0	1	0
11	2	1	0	1
12	3	1	0	0
13	3	1	0	0
14	2	1	0	0
15	3	2	1	1
16	1	1	1	1
17	2	1	1	1
18	3	2	2	1
19	2	1	1	0
20	1	1	1	1
21	3	1	0	0
22	1	0	0	0
23	2	0	0	0
24	3	1	0	0
25	2	0	1	1

[a]0=no symptoms, 1=mild symptoms, 2=moderate symptoms, 3=severe symptoms.

TABLE 2
Ratings of Tranquilizer Use in 25 Patients Treated with Thiopental-Induced Desensitization[a]

Patient Number	Week 0	Week 6	Week 12	Week 24
1	2	2	1	1
2	2	1	0	0
3	2	2	1	0
4	2	2	1	0
5	2	2	1	1
6	2	2	3	2
7	2	1	1	0
8	2	1	0	0
9	3	1	1	1
10	2	2	0	0
11	2	1	0	0
12	2	3	2	1
13	2	1	1	1
14	2	2	0	0
15	2	2	1	0
16	2	1	0	1
17	2	1	1	0
18	2	1	0	0
19	2	2	2	1
20	3	2	2	1
21	2	1	1	1
22	0	0	0	0
23	1	1	0	0
24	0	0	0	0
25	2	3	1	0

[a]0=no medication use, 1=decreased use, 2=unchanged use, 3=increased use.

RESULTS

Tables 1 and 2 show progressive reduction in symptom distress as well as in the use of tranquilizers at 6-,

TABLE 3
Distribution Frequency of Ratings of Phobic Anxiety in 25 Patients Treated with Thiopental-Assisted Desensitization

	Number of Patients			
Rating of Phobic Anxiety	Week 0	Week 6	Week 12	Week 24
Severe	12	0	0	0
Moderate	8	4	1	0
Mild	5	15	13	11
No phobic anxiety	0	6	11	14

TABLE 4
Distribution Frequency of Ratings of Tranquilizer Use in 25 Patients Treated with Thiopental-Assisted Desensitization

	Number of Patients			
Rating of Tranquilizer Use	Week 0	Week 6	Week 12	Week 24
Increased use	2	2	1	0
Unchanged use	20	10	3	1
Decreased use	1	11	11	9
No use of tranquilizers	2	2	10	15

12-, and 24-week follow-up. The frequency distributions of ratings for phobic anxiety and of tranquilizer use at follow-up intervals are shown in tables 3 and 4. The finding that 14 patients were symptom free and 15 patients were tranquilizer free at the 24-week follow-up demonstrates the usefulness of the treatment program.

CONCLUSIONS

Desensitization therapy using thiopental is not time-consuming, and progressive improvement reinforces the therapeutic process. Combined with the judicial use of and then reduction of use of anxiolytic drugs, it is a practical method of treating phobic anxiety in communities where there is a shortage of psychiatric personnel.

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Failure of Naloxone to Reduce Manic Symptoms

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The authors conducted a double-blind placebo-controlled study in which patients with a wide range of manic symptoms were administered 20 mg of naloxone subcutaneously. Naloxone failed to improve manic severity, activation-arousal, or elation-grandiosity for intervals up to 3 hours. Global nurse ratings of mania did not improve over an 8-hour period. The authors suggest that the question of endorphin involvement in mania has not been resolved and recommend clinical studies with longer acting oral narcotic antagonists such as naltrexone.

The recent discovery of endogenous morphine-like substances (endorphins) whose pharmacological effects are largely indistinguishable from those of opiates suggests that behavioral states which resemble opiate effects might be brought about by endorphins. Several investigators (1, 2) have described a resemblance between the manic state and opiate effects, and others (3, 4) have specifically suggested that an excess of endorphins may mediate euphoria and mania. In animals low doses of opiates cause hyperactivity and heightened arousal (5). In man opiates cause euphoria and are potent analgesics. Endorphins have been found to play a role in regulating pain perception in man (6). Many manic patients are more pain tolerant than age- and sex-matched controls (7). In a preliminary study elevated levels of opiate-binding substances were reported in the cerebrospinal fluid of a few manic patients (8). Thus, there are behavioral and preliminary clinical findings to suggest that mania, an illness characterized by hyperactivity, arousal, grandiosity, pressured thought and speech, euphoria, and loss of judgment might be associated with an elevation in endogenous opiate-like substances.

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If increased endorphin effects are involved in the pathogenesis of mania, then naloxone, a narcotic antagonist, would be expected to improve endorphin-dependent manic symptoms. Three groups recently reported acute clinical trials of naloxone in mania (9–12). Janowsky and associates (10) reported antimanic effects of 20 mg of naloxone on 10 manic patients, but Emrich and associates (11) were unable to demonstrate any naloxone-induced changes in 2 manic patients who took 4 and 24.8 mg of naloxone, respectively. We previously reported a dose-dependent reduction on manic speech and pressure of thought in 1 of 4 manic patients (12). We will report on 10 patients with a wide range of manic symptoms to whom we administered 20 mg of naloxone.

METHOD

Ten patients who met Research Diagnostic Criteria (13) for either bipolar affective illness, manic stage, or schizoaffective illness, manic type, participated in a double-blind, placebo-controlled, random-order study of the effects of 20 mg of naloxone on manic symptoms. All 10 patients were selected from patients on two affective disease wards at NIMH; the ward psychiatrist selected patients for study according to their willingness to participate and whether their manic symptoms were relatively stable. Patients with short mood cycles were excluded because of the likelihood that a spontaneous switch might be seen as a drug response. Clinical characteristics of the 10 patients are presented in table 1. All patients gave oral and written informed consent and participated voluntarily.

Drugs were administered between 8:30 a.m. and 10:00 a.m. A placebo of equal volume was used, and all substances were administered subcutaneously in the tissue over the deltoid. Each patient served as his or her own control; order of administration was random, and naloxone and placebo were given 1 or 2 days apart. Patients participated in routine ward activities before and after drug administration except for their interviews with the rating psychiatrist. One of us on each ward rated patients on the Beigel-Murphy Manic Rating Scale (14) and the Brief Psychiatric Rating Scale (15) at a baseline point (30 minutes before drug administration) and at 30, 60, and 120 minutes after injection.

TABLE 1
Clinical Characteristics of 10 Patients Given 20 mg of Naloxone

Patient	Diagnosis	Age (years)	Sex	Mania Rating[a]	Days Since Switch to Mania	Concurrent Medication
1	Schizoaffective, manic	30	M	10	7	Chlorpromazine
2	Bipolar I, manic	43	F	9	4	Phenelzine
3	Bipolar I, manic	50	F	4	7	Imipramine, lithium
4	Bipolar I, manic	25	F	8	9	Placebo
5	Bipolar I, manic	43	F	10	2	Placebo
6	Bipolar I, manic	45	F	9	3	Phenelzine
7	Bipolar I, manic	54	F	9	7	Placebo
8	Schizoaffective, manic	38	M			Placebo
9	Bipolar I, manic	29	M	9	10	Carbamazepine
10	Bipolar I, manic	55	F	9	21	Lithium and carbamazepine

[a]Bunney-Hamburg global mania rating; maximum score=15.

TABLE 2
Effect of 20 mg of Naloxone on 10 Manic Patients

Beigel-Murphy Mania Rating Scale	Placebo		Naloxone		Placebo		Naloxone	
	Mean Score	SE	Mean Score	SE	Mean Change	SE	Mean Change	SE
Manic severity								
Baseline	84.9	14.9	74.6	12.9				
After administration								
30 minutes	67.7	12.6	71.3	13.3	17.2	9.5	3.1	2.6
60 minutes	75.8	16.1	69.4	13.2	8.5	9.3	8.6	4.3
120 minutes	69.8	12.1	69.1	13.1	15.5	8.9	7.3	4.3
Elation-grandiosity								
Baseline	29.7	5.8	24.7	5.6				
After administration								
30 minutes	28.7	5.6	23.6	5.4	1.5	.7	1.2	.8
60 minutes	28.5	5.7	22.7	6.0	1.5	2.6	2.5	1.3
120 minutes	30.2	5.5	22.2	6.3	.2	1.5	3.0	1.7
Activation-arousal								
Baseline	51.7	7.5	45.0	7.6				
After administration								
30 minutes	41.0	6.7	44.9	7.9	10.7	4.9	.1	1.1
60 minutes	46.1	8.4	44.0	8.8	6.0	4.4	3.1	2.9
120 minutes	43.2	6.8	42.5	8.4	8.5	4.5	2.8	3.5

Patients were either stabilized on medicine or were medication free (table 1). Ratings were based on 10- to 15-minute structured interviews at each evaluation point as well as on subsequent observation of the patients during routine ward activities. Global nurse ratings of mania were performed twice daily on a modified Bunney-Hamburg scale (16) in order to assess possible long-term or delayed effects of injection.

RESULTS

Naloxone failed to bring about improvement for any of the 10 patients on the manic severity, activation-arousal, or elation-grandiosity scales of the Beigel-Murphy at 30, 60, or 120 minutes after administration (table 2). Furthermore, total psychopathology as measured by the BPRS did not improve, nor did the single item "grandiosity" or the cluster "thought disorder" (which includes unusual thought content, hallucinations, and conceptual disorganization). In fact, patients tended to improve more on placebo than on naloxone (although not significantly) at 30 minutes after administration. We found no significant differences in baseline scores between placebo and naloxone, although there was a tendency for mania to be rated as less severe on the naloxone day. Global nurse ratings of mania over an 8-hour period also failed to demonstrate improvement.

DISCUSSION

Our results do not support the findings of Janowsky and associates (9, 10). What factors could explain our negative findings in view of previous reports of a positive effect of naloxone on mania? We administered the drug subcutaneously because absorption is rapid and because this method was more convenient than using intravenous lines in hyperactive and intermittently uncooperative patients. It is possible that an intravenous route would have achieved higher blood levels. We did not isolate our patients from their usual setting and ward routine, as did Janowsky and associates, which

might possibly account for differences in drug response. Janowsky and associates (10) observed therapeutic antimanic effects in those patients who had the most severe mania. Our patients were not all severely manic, nor were they tested in the first few days of a manic episode (table 1). However, even in the most classical manic patients with pressured speech and thought, grandiosity, and elation, no ratable improvement occurred. The manic state is characterized by fluctuation in ratings (e.g., see placebo change means in table 2). As might be expected when we administered placebo to the 10 patients, their mania scores varied widely with an overall tendency toward improvement at 30 minutes (table 2). Does this reduction represent a placebo effect rather than variability in mania? If so, is naloxone blocking this placebo effect? In the Janowsky and associates studies (9, 10) and in our study the group was a mix of medicated and medication-free patients. Although we observed no effect on thought disorder, our previous findings that improvement of thought disorder in schizophrenic patients who were administered naloxone occurred predominantly in patients taking neuroleptics[1] suggests the need for continued attention to possible neuroleptic-naloxone facilitation in manic patients with thought disorder.

Although the failure of naloxone in this study to reduce manic symptoms does not support the hypothesis that mania is caused by excess activity at opiate receptors, it would be premature to dismiss this theory. The dose of 20 mg of naloxone might not be high enough to block the effects of usual doses of opiates. Naloxone's potency as an antagonist varies in various physiological preparations (17), and in fact three classes of receptors have been postulated from studies of the effects of various narcotics on the "chronic spinal dog" (18, 19). Thus, naloxone might block some endorphin effects better than others. Administration of a long-acting pure opiate antagonist such as naltrexone provides a better test of the role of endorphins in mania than that of a single dose of the short-acting antagonist naloxone

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Medicaid Records and the Psychiatric Private Sector

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The authors report a study of records maintained by private practitioners for their Medicaid patients. The quality of record keeping in general fell far short of Medicaid requirements. Board-certified or hospital-affiliated psychiatrists kept better charts than those who did not have these credentials. Educational background did not correlate with extent of record keeping.

The U.S. government, which is massively involved in the subsidy of health care, is increasingly seeking to determine if and how competently care is actually provided. In a previous study of the delivery of private psychiatric treatment to the New York City Medicaid population (1) we documented the heavy involvement of foreign medical graduates in the care of the poor and medically indigent. Other researchers (2–4) have found that the practice setting rather than the educational background of practitioners correlated with the quality of care. However, Saywell and Studnicki (4) pointed out that Board-certified physicians tended to cluster in the institutions providing the finest care. No comparable study has ever been done in the psychiatric sector, where the impact of treatment on outcome is often unclear.

A study of the credentials of individuals who had a large number of psychiatric Medicaid patients (5) revealed that significantly more graduates of U.S. medical schools (USMGs) were Board certified than were graduates of foreign medical schools (FMGs). However, the Medicaid practitioners did not differ in this or any other regard from all of the psychiatric practitioners in New York City.

For the present work, part of a continuing effort to study the psychiatric Medicaid system in New York City, we researched the completeness of record keeping. In particular we sought to address the question of whether Board certification, educational background, or hospital affiliation was related to the quality of charts.

METHOD

Data were obtained from 998 patient charts. These charts were selected from the Medicaid caseloads of a 40% (N=120) random sample of psychiatrists who billed for at least 10 Medicaid patients in 1976. We obtained data from the records of the New York City Bureau of Medical Assistance and from the 1977 edition of the biographical directory of APA fellows and members about the educational background and professional credentials of the psychiatrists in the sample.

We reviewed and scored the charts using a form that contained 35 items reflecting Medicaid requirements about information to be gathered during a patient's first three visits to a provider (see appendix 1). Charting this information is "an absolute prerequisite for the continuance of Medicaid reimbursement" (6). The checklist was grouped into six categories with a maximum possible score of 39. These categories were identifying data, data on current illness relating to chief complaint, medical history, mental status, diagnosis, and therapeutic plan. Additional descriptive data were gathered, including the patient's age, sex, ethnic group, previous inpatient or outpatient treatment, drug dependence, and the diagnosis after the first three visits. The diagnoses were based on those listed in *DSM-II* and were grouped in the following way: psychoses (including schizophrenia, major affective psychosis, paranoid states, and other psychoses), neuroses, personality disorders, alcoholism, drug dependence, and other diagnoses. All tests of significance were done using Student's t test.

RESULTS

Table 1 presents data on the quality of charts of the psychiatrists sampled according to whether the psychiatrists were Board certified, hospital affiliated, or attended medical school in the United States. Regardless of the distinctions made between practitioners,

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TABLE 1
Average Scores for 998 Patient Charts Kept by 120 Psychiatrists Using Medicaid Criteria, According to Board Certification, Hospital Affiliation, and Graduation from U.S. Medical School (USMG) or Foreign Medical School (FMG)

Medicaid Criterion	Maximum Possible Score	Certified	Noncertified	Affiliated	Nonaffiliated	USMG	FMG	Composite Score
Identifying data	4	3.3	3.2	3.3	2.8[a]	2.9	3.4[b]	3.23
Data on current illness relating to chief complaint	12	7.3	6.0[c]	6.8	5.4[a]	6.2	6.6	6.50
Medical history	4	1.3	1.0	1.2	1.0	1.4	1.0[d]	1.13
Mental status	12	7.2	4.8[c]	6.0	4.6[a]	5.8	5.6	5.65
Therapeutic plan	6	3.4	2.3[c]	2.8	2.2[a]	2.7	2.7	2.70
Diagnosis	1	0.9	0.8[c]	0.8	0.8	0.7	0.9[b]	0.86

[a]The charts of the psychiatrists who were affiliated were scored significantly higher than those of nonaffiliated psychiatrists ($p<.05$).
[b]The charts of the FMGs were scored significantly higher than those of USMGs ($p<.05$).
[c]The charts of certified psychiatrists were scored significantly higher than those of noncertified psychiatrists ($p<.05$).
[d]The charts of USMGs were scored significantly higher than those of FMGs ($p<.05$).

the quality of record keeping fell far short of Medicaid requirements. No group recorded more than 60% of the expected information on the charts. Identifying data and diagnosis were the items most often completed; medical history was most often neglected. Data are also provided on charting for the sample as a whole.

Those psychiatrists who were Board certified consistently recorded more complete information about their patients than their colleagues without certification. This difference was found in each of the checklist categories with the exception of the noting of identifying data. Those doctors having hospital affiliations kept significantly better charts than those without such affiliations (see table 1). This difference was noted for the rating of the entire chart and in each category, except for medical history and diagnosis. When USMGs were compared with FMGs no global difference in case recording was apparent. The several category differences may be noted in table 1. When psychiatrists without Board certification were compared on the basis of educational background, USMGs and FMGs were found to be comparable. There was no significant difference between USMGs and FMGs who were Board certified.

The records of the psychiatrists sampled were examined to determine whether or not the quality of charting differed for patients on the basis of sex, ethnic group, or diagnostic group. The data reveal no significant differences for these variables.

DISCUSSION

The data elaborated in this paper present a glimpse of clinical practice in a government-sponsored program as reflected through psychiatric record keeping. Whether these are representative of a wider sphere of practice is not known. An anecdote from this study may serve to illustrate the point. A physician, feeling embarrassed by the inadequacy of his Medicaid charts, remarked, "You'd see much better records if this were one of my regular, paying patients." The attitude expressed in this anecdote, often voiced in less egregious form during the audit procedure, reflects the ambivalence with which some doctors view patients seen under the auspices of Medicaid. Previous work (7) has documented physician prejudice against Medicaid rather than against individual Medicaid patients. Thus the doctors' feelings for these ostensibly private patients may be tempered to the degree that they are in fact subsidizing the care of these patients by accepting the low reimbursement offered through Medicaid as well as having to contend with the many bureaucratic obstacles inherent in the system.

Clearly, in a government-mandated and regulated health delivery system total compliance could be forced with respect to charting standards. The positive relation of record keeping to quality of care, not yet documented in psychiatry, has been demonstrated in other medical areas. Long and Rogers (8) found that the recording of a procedure was tantamount to a guarantee of its having been performed. Whether physicians' attitudes would prove as malleable is less clear. In the present study we found that psychiatrists who evidenced professional achievement through Board certification and maintenance of hospital affiliation kept demonstrably more complete charts. Conversely, to the extent a process measure such as chart review reflects quality of care, no difference between USMG and FMG psychiatrists was evident. These findings lend support to those who believe certification to be meaningful. Clearly, insufficient data are available from private charts to permit their use as retrospective vehicles for evaluative studies. These data extend to the private sector the finding of a previous paper demonstrating the inaccuracy of reporting of data to Medicaid and the resulting inability to use such information for research (9).

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APPENDIX 1
Medicaid Criteria for Information to Be Recorded on Patients' Charts

Each of the 35 items was scored 0 for absent, 1 for present, and 2 for adequate description.

Identifying Data
1. Age
2. Sex
3. Ethnic group
4. Chief complaint

Data on Current Illness Relating to Chief Complaint
5. Phenomena
6. Duration
7. When symptoms occur
8. Work situation
9. Effects on life situation, e.g., work, family
10. Present living situation, e.g., married, with children, where living
11. Vegetative signs, e.g., changes in sleep, weight, appetite, libido
12. Psychiatric history: condition, treatment, response
13. Developmental history
14. Education: highest grade achieved
15. History of psychiatric illness in family

Medical History
16. Medical, surgical, or neurological illness and how treated
17. Alcohol use
18. Use of nonmedical drugs

Mental Status
19. Orientation
20. Intellect
21. Mood, affect
22. Hallucinations
23. Delusions
24. Reality testing
25. Judgment
26. Insight
27. Speech or thinking
28. Thought content
29. Object relations
30. Motor

Diagnosis (number 31)

Therapeutic Plan
32. Therapy
33. Medication
34. Goals
35. Prognosis

Measuring Psychiatric Utilization: The Rubber Yardstick

BY JOHN KRIZAY, M.A.

The author suggests that measuring utilization of benefits for nervous and mental disorders by using the percent of total benefits paid is imprecise because even small changes in the much larger portion of benefit payments that are attributable to nonpsychiatric services can make it appear that the level of psychiatric utilization has changed. His preferred measure is a calculation of covered charges attributable to psychiatric care per each person covered under the plan. The author illustrates his points by showing that, when properly calculated, psychiatric utilization under the Civilian Health and Medical Program of the Uniformed Services is much lower than that under many other plans.

Surely, a major component of the debates over whether psychiatric services should be a feature of health insurance coverage is the question, "How much would it cost?" The known experience under various health insurance plans provides some clues, but the answers are not always as obvious as they seem. The issue here is one of finding the proper measure, some standard way of expressing utilization data that permits aggregation and interplan comparisons as well as a sound basis for projecting the experience onto a larger population.

The measure "share of benefits paid for mental and nervous disorders" has come into widespread use as the indicator of utilization of psychiatric services under health insurance plans. Insurance carriers, government agencies, and both proponents and opponents of psychiatric coverage all seem to use it. The fact is, however, that this measure is about as precise as a rubber yardstick, and it should be abandoned.

The clearest example of the dangers of using this measurement may be found in its application to the Civilian Health and Medical Program of the Uniformed Services (CHAMPUS) some half a dozen years ago, when it was revealed that the payment to beneficiaries for psychiatric services amounted, by some calculations, to as much as 23% of all benefits paid under this program (1). This payout level was thought then to be not just excessive but outrageous, and it quickly gave rise to a movement in Congress to curtail this coverage. Had the analysis focused on real rates of utilization of psychiatric services per covered person instead of the percent of total benefits paid for psychiatric treatment, it is certain that the panic response never would have occurred, for the real facts are that CHAMPUS enrollees are extremely modest utilizers of psychiatric services compared with other groups with similar health insurance coverage. Indeed, the real utilization rate of this group is similar to that of groups whose health insurance plans offer only very limited coverage for mental and nervous disorders. In terms of dollars' worth of psychiatric services used per each person covered in 1977, CHAMPUS stands well below several federal employee plans, including some offering inferior coverage (see table 1).

The measure of percent of benefits paid distorted the CHAMPUS picture because people covered by this program have access to two benefit packages: those provided by CHAMPUS (the private sector) and those available at direct care facilities of the military establishment. Persons eligible for CHAMPUS benefits use direct care facilities for most surgical and many general medical needs, but since direct care facilities do not provide long-term psychotherapy, nearly all psychiatric services are paid for with CHAMPUS funds. Thus, benefits paid for psychiatric services are a large percentage of all CHAMPUS benefit payments because CHAMPUS benefit payments for all other medical services represent only a small part of such services used by those eligible for CHAMPUS benefits.

The CHAMPUS program has unique features that readily explain why the percent of benefits measure distorts the real psychiatric utilization rate. But the measure is also inappropriate when applied to more typical plans. Supporters of psychiatric coverage in health insurance, for example, often cite the experience under Blue Cross-Blue Shield Federal Employee Program (FEP), which supposedly shows a leveling off in the use of psychiatric services at 7½% of all benefits paid. The statement is wrong both in fact and in concept. But before examining what has happened to FEP utilization over the years, let us examine why the measurement is inadequate.

Assume, for the sake of discussion, that benefits

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TABLE 1
Dollar Charges per Covered Person for Treatment of Mental and Nervous Disorders Under CHAMPUS and Various Federal Employee Health Plans Offering Different Quality of Mental Health Coverage, 1977[a]

Plan	Charges per Covered Person	Quality of Plan's Mental Health Coverage
Government Employees Benefit Association (National Security Agency)	$59.72	Excellent
Foreign Service	40.61	Excellent
Blue Cross/Blue Shield Federal Employee Program	27.00	Excellent
Aetna	18.56	Good
American Federation of Government Employees	18.41	Fair
CHAMPUS[b]	12.27	Excellent
Special Agents Mutual Benefit Association (Federal Bureau of Investigation)	12.06	Good/excellent
National Alliance	11.76	Fair
Government Employees Hospital Association	10.97	Fair

[a]Data are from Annual Reports of Insurance Carriers, Federal Employees Health Benefit Plans (2).
[b]CHAMPUS utilization rate per covered person as expressed here was computed on the basis of an eligible population of approximately 9 million persons, as estimated by the U.S. Department of Defense in its Military Health Care Study (3). In this study the Department of Defense estimated that 8,951,000 persons were eligible for CHAMPUS benefits in 1975 and projected that 9,295,000 would be eligible in 1980.

paid in a given plan and during a given year equaled 7½% of all benefits paid (as claimed in the FEP case) and that the total benefits paid during the year amounted to $100 million—$7.5 million for psychiatric services and $92.5 million for nonpsychiatric services. Assume, further, that during the next year, the second opinion requirement on surgery caused a reduction in surgical and related hospital episodes so that nonpsychiatric benefits dropped by 5% (after correcting for inflation). If the utilization of psychiatric services did not change at all, the share of benefits paid for psychiatric services would increase to 7.9% of total benefits!

In other words, the basic problem in using this measurement is that even small changes in the much larger portion of benefit payments that are attributable to nonpsychiatric services can make it appear that the level of psychiatric utilization has changed. Thus, psychiatric utilization can increase dramatically, but the increase can be obscured if utilization of all other medical services increases at the same rate. On the other hand, psychiatric utilization can appear to decline if, in fact, it does not change at all but the use of all other covered services increases.

The preferred measure is a calculation of covered charges attributable to psychiatric care per each person covered under the plan. Charges—if available—are preferable to benefits paid in that their use eliminates distortions that arise due to variations in such factors as deductibles and copayment features. Translating covered charges to a per capita (per covered person) basis yields a measure that can be compared with other plans and projected onto different populations.

For time series analysis, of course, the data must be corrected for inflation. There is something of a problem in finding an appropriate deflator when the overall utilization rate is being considered because the general medical component of the Consumer Price Index may not move in consonance with the psychiatric subcomponents taken alone. However, the general medical deflater is adequate for interplan comparisons and for judging longer-term trends.

The utility of the measure of per capita covered charges can be seen if applied to the experience with the Federal Employees Health Benefits Program (FEHBP). Here we find that the real rate of psychiatric utilization under the Blue Cross-Blue Shield portion of the total FEHBP has not stabilized, as frequently reported, but has increased from $21 per capita in 1973 to $27 per capita in 1977—a statistically significant increase of over 25%. However, if one examines the data, one finds that there was a substantial reduction in enrollment in the Blues during the period studied while, at the same time, enrollment in a variety of small plans available to federal employees increased rapidly. It is evident that as the Blues' premium rates increased, many federal employees exercised their once-a-year option to choose less expensive plans. It is a fair assumption that those confident of their health—including low utilizers of psychiatric care—were willing to risk lesser coverage to take advantage of the lower premium rates that other plans offered. Thus, if we aggregate the psychiatric utilization data of these lesser plans with Blue Cross-Blue Shield experience, the combined rates of covered charges per capita attributable to psychiatric services over the five-year period show less than a 10% increase over the entire period, with the year-to-year data showing two increases and two decreases. The rate of increase is not statistically significant, i.e., the year-to-year changes could be attributable to reporting error, inappropriateness of the deflator in a given year, or other technical factors.

COMMENT

Thus, the data properly presented not only show substantial stability in rates of use of mental health services in the FEHBP as a whole, but also provide a dollar cost per capita that may be used for projections onto larger populations to estimate the potential cost of liberal mental health coverage. (Note that all federal employees have access to liberal mental health coverage even though many do not choose to avail themselves of it.) The overall cost per capita of all persons covered under these programs was approximately $24 per year—roughly $14 for inpatient visits and $7 for outpatient costs, with the remaining $3 attributable to

other costs and to differences resulting from use of different deflators for inpatient, outpatient, and total costs.

The result is a reassuring one for advocates of liberal mental health protection in national health insurance—all the more so because it is arrived at by a measurement that is reasonably reliable, projectable, and not susceptible to the distortions likely to result when the percent of benefits measure is used.

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Psychosocial Outcome After Coronary Artery Surgery

BY MICHAEL J. GUNDLE, M.D., BOZMAN R. REEVES, JR., M.D., SUZANNE TATE, DAVID RAFT, M.D., AND L.P. MCLAURIN, M.D.

To describe psychosocial adaptation after coronary artery surgery and to identify preoperative factors associated with good outcome, the authors interviewed 30 patients before and 1–2 years after surgery. Despite good physiologic outcome (as measured by treadmill and cardiac function) this sample was found to be functioning poorly. Eighty-three percent were unemployed, and 57% were sexually impaired. A preoperative duration of symptoms of eight months or more was associated with significantly worse postoperative overall adaptation. Most patients who had suffered angina eight months or longer evidenced a damaged self-concept, which was reinforced rather than repaired by the experience of surgery.

In the continuing debate over the usefulness of coronary artery surgery, investigators have given little attention to psychological and social outcome. The one psychosocial variable that has been measured—employment—has not been shown to improve for patients after the operation (1–5). This is despite evidence that coronary bypass brings about both marked decreases in frequency and severity of angina (6, 7) and improvement in functional myocardial status (8). The number of persons being treated with coronary bypass surgery is rising; the annual costs nationally now exceed $1 billion (9). These facts should impel investigators to evaluate return to employment thoroughly and to look at factors other than relief of angina that contribute to the well-being of patients with coronary artery disease.

Heller and associates (10) studied psychosocial outcome in patients undergoing open heart surgery prior to the development of coronary bypass techniques. They found from pre- and postoperative interviews that despite little physical impairment patients became less self-reliant and more socially constricted and had decreased expression of sexual and aggressive drives. One-third were still encountering significant psychological hindrances to recovery one year after the operation. This subgroup showed considerable anxiety, depression, somatic preoccupation, social withdrawal, and impaired sexual function.

Clinical experience with coronary bypass suggests that the psychosocial outcome of this procedure is also often less than optimal. We attempted to collect data from a group of coronary bypass patients that would confirm or refute that impression. Despite the measurement difficulties inherent in such an approach, we defined outcome broadly. We measured not only employment but also adaptation to emotional and social needs. We assumed that, when successful, this adapta-

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tion would be evidenced by self-contentment, satisfying interpersonal relationships, sexual pleasure, and relative freedom from anxiety, depression, and hypochondriacal concerns. We correlated this psychosocial outcome with three preoperative factors that we hypothesized would be associated with good psychosocial outcome. These factors were a short duration of cardiac symptoms (angina), Type A behavior pattern, and a low level of psychiatric symptoms, for example, anxiety.

METHOD

Between September 1976 and November 1977, 192 unselected patients who underwent cardiac catheterization at North Carolina Memorial Hospital were examined by means of psychological tests and a semistructured psychiatric interview. Thirty-five of these patients underwent coronary bypass surgery 2–16 weeks after catheterization. Thirty of these 35 returned for psychosocial and cardiologic evaluations 12–24 months after surgery. This group consisted of 22 white men (73%), 6 white women (20%), and 2 black men (7%). They came from rural North Carolina, an economically impoverished area by national standards. They were factory and farm workers, salesmen, and managers. The mean age (±SD) at the time of operation was 51.4±7.1 years. None of the patients suffered from organic brain syndrome before coronary bypass or afterward. There were no instances of stroke or postoperative psychosis. All patients participated in an active physical rehabilitation program after surgery. The purposes of the study were explained and informed consent was obtained before the preoperative evaluation and again at the postoperative interviews.

The preoperative evaluation was based on the results of the SCL-90 (11) and a semistructured interview conducted by a psychiatrist (D.R.). The SCL-90 is a self-administered psychiatric symptom inventory. The psychiatric interviewer determined the behavior pattern (Type A or non-A) (12, 13) and asked each patient about work status and sexual functioning. Sex was recorded as unsatisfactory only when a clear problem, such as chronic impotence or lack of desire, was elicited. The number of months since the onset of angina was recorded.

Patients were again examined by means of the SCL-90 and a semistructured psychiatric interview (conducted by M.J.G.) 12–24 months after surgery. Work status and sexual functioning were evaluated using the preoperative criteria. The interviewer used open-ended questions to explore with the patient his or her emotional and social adaptations since the operation. He then scored each patient on each of the nine scales of the Adaptive Balance Profile (14), an instrument used by Heller (10) for measuring psychosocial outcome following open heart surgery. The sum of the absolute values of the nine scale scores was used as a measure of overall psychosocial adaptation. All investigators remained blind to each others' findings until the end of the data collection.

A second numerical measure of adaptation was the Psychosocial Adjustment to Illness Scale (PAIS) (15), which was administered by a trained psychiatric research assistant (S.T.). The PAIS is a structured interview designed to assess the quality of a patient's psychosocial adjustment to current illness or to residual effects of illness.

Outcome was then measured in five ways: ABP, PAIS, employment, sexual function, and SCL-90. The effect on each of the outcome variables of the following three preoperative variables was then measured: presence of psychiatric symptoms (SCL-90), behavior pattern (Type A or non-A) and duration of cardiac symptoms (predominantly angina). Eight months was chosen as the dividing point between short and long durations of symptoms on the basis of both clinical experience and the distribution of the ABP and PAIS scores.

RESULTS

Twenty-six of the 30 patients participated in tests of physiological outcome after the surgery. Fourteen patients (54%) had normal results on the treadmill test; in 6 patients (23%) S-T depression was observed on the ECG before they reached the exercise tolerance predicted by age and sex; and 6 patients stopped before S-T depression occurred and before they reached the predicted exercise tolerance. Eighteen patients (69%) reached their predicted exercise tolerance on the treadmill with or without the occurrence of S-T depression.

Despite this reasonably good physiologic outcome, a high proportion of these patients were psychologically and socially disabled at follow-up. Unemployment and sexual dysfunction (mainly impotence) were reported by 83% and 57%, respectively. A number of other manifestations of maladjustment that are not easily quantified were also reported, including constricted social life, low self-esteem, and lack of pleasure from close relationships. A small but significant number of patients revealed persistent dependency needs of varying severity, serious distortions of body image, and symptomatic depression.

As a first step in understanding the poor psychosocial outcome of this sample we looked for a statistical association between each of the three preoperative variables and the five postoperative outcome measures (see table 1). We found employment at follow-up to be significantly associated with preoperative duration of symptoms and behavior pattern as well as with preoperative employment status. Sexual function at fol-

TABLE 1
Preoperative Variables Affecting Psychosocial Outcome in 30 Patients Who Had Coronary Artery Surgery

Preoperative Variable	Postoperative Status: Employed N	%	Significance	Sexually Functional N	%	Significance	ABP Score[a] Mean	SE	Significance	PAIS Score[a] Mean	SE	Significance
Duration of symptoms			p<.05[b]			p<.0001[c]			p<.01[d]			p<.01[d]
8 months or less (N=11)	4	36		10	91		12.5	0.9		26.8	3.8	
Over 8 months (N=19)	1	5		3	16		18.3	1.2		43.3	3.1	
Behavior pattern			p<.05[e]			n.s.			n.s.			n.s.
Type A (N=17)	5	29		8	47		15.5	1.3		32.6	3.8	
Non-A (N=13)	0			5	38		17.0	1.6		43.3	3.6	
Preoperative status			p<.01[f]			p<.05[f]						
Employed (N=23)	5	22										
Unemployed (N=7)	0											
Sexually functional (N=22)				11	50							
Sexually dysfunctional (N=8)				2	25							

[a]Lower scores indicate better adaptation for both the Adaptive Balance Profile and the Psychosocial Adjustment to Illness Scale.
[b]χ^2=4.8, df=1.
[c]χ^2=16.0, df=1.
[d]Student's t test.
[e]χ^2=4.6, df=1.
[f]McNemar's test.

low-up was associated only with duration of symptoms and preoperative sexual status.

The preoperative and postoperative SCL-90 scores were not found to be associated with any other pre- or postoperative variable measured; therefore these results were dropped from the analysis. We observed symptoms in a number of patients who had denied them on the SCL-90, suggesting that the self-administration format of this test seriously limits its usefulness in this population. The interaction terms between each pair of preoperative variables were found to be insignificant, so they were also dropped from the analysis.

The Psychosocial Adjustment to Illness Scale and Adaptive Balance Profile were the best overall measures of psychosocial outcome. They were highly correlated with one another (rho=0.66, p<.001). Each was significantly associated with preoperative duration of symptoms but to no other preoperative variable (see table 1).

Age is known to influence employment and is therefore a possible confounding variable. Levels of cardiac function could similarly distort the results if unevenly distributed among the groups. There was no significant difference, however, in the mean ages (±SEM) among any of the subgroups analyzed: 51.8 years (±2.8) and 51.1 years (±1.3) for the patients with short and long duration of symptoms, respectively; and 51.8 years (±1.9) and 50.6 years (±1.7) for patients with Type A and non-A behavior, respectively. There was also no difference in cardiac function at follow-up between patients with a short duration of symptoms preoperatively and those who had had symptoms for a long period. Nor did Type A patients exhibit a mean cardiac function value that was significantly different from that of non-A patients.

DISCUSSION

Our data show that considerable psychosocial morbidity may persist despite correction of coronary insufficiency. This is demonstrated in the patients' poor adaptation to life, work status, and sexual functioning. Previous studies have assessed the quality of life following coronary bypass surgery solely on the basis of return to work and relief of angina. In our opinion sexual and adaptive ego functioning are also important aspects of the quality of life. It is well known that restoration to normal functioning in such areas of a person's life, once they have deteriorated, is difficult to bring about. Our findings suggest that the clinical assessment of patients with ischemic heart disease should be expanded to include evaluation of psychosocial adjustment. Changes in this adjustment since the onset of cardiac symptoms should be paid particular attention.

One reason for the higher employment rate found in other samples (62%–79% versus 17% in ours) may have been the lower socioeconomic status of our population. Croog and Levine (16) have shown that heavy lifting at work is inversely related to employment after myocardial infarction. Over one half of our sample was derived from blue-collar workers. However, even among those who could engage in less strenuous work, we found an inhibition to work. This was frequently related to the patient's persistent image of him- or herself as being "damaged." We found that those with prolonged symptoms prior to surgery (particularly those who also had limited education and income) commonly evidenced a damaged self-concept. This damaged self-image was highly correlated with poor sexual functioning, particularly in males.

Our initial clinical impression had been that those with a Type A behavior pattern, characterized by ag-

gressiveness, a sense of urgency, and competitiveness, would have less psychological morbidity. This did not prove to be the case. Type A patients in our population were more often employed but were not better adapted overall or more sexually functional than non-A patients. Since Type A behavior is defined in part in terms of attitude toward work, higher employment postoperatively among Type A patients compared with non-A patients has little meaning.

Our findings indicate that at least for certain groups of patients (those in the lower socioeconomic categories) the decision to perform or not perform coronary artery surgery should be reached expeditiously. To assure a good functional outcome, the physician needs to be aware of the changes in self-image that have occurred during the period the patient has been experiencing anginal pain. Functional outcome is likely to be enhanced by considering psychosocial adjustment, in addition to physiologic factors, in the evaluation and selection of patients for coronary artery surgery.

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Family Therapy Training for Psychiatric Residents

BY HENRY T. HARBIN, M.D.

The author describes a comprehensive four-year educational program for training psychiatric residents in marital and family therapy. Residents receive an average of 70 hours of didactic instruction and 175 hours of one-on-one supervision in family therapy; they conduct approximately 500 hours of family and marital therapy with 75–85 outpatient and inpatient families. The author identifies faculty and resident resistance to an additional theoretical model and the major involvement of nonphysician faculty but stresses that residents now have the opportunity to truly understand and implement a biopsychosocial model and that psychiatrists must increase their experience in specialized therapeutic modalities to remain clinical leaders.

In this paper I will describe an educational program designed to train psychiatric residents in the practice of marital and family therapy. The program provides intensive and comprehensive clinical and didactic training and covers all four years of basic residency training at the Institute of Psychiatry and Human Behavior, University of Maryland Medical School.

The University of Maryland's graduate training program has approximately 50 residents with the first postgraduate year consisting of 6 months of inpatient psychiatric work and 6 months of medicine. Second-year residents usually spend 12 months on a university admitting ward. The third-year rotation is in the outpatient service and emphasizes liaison psychiatry, emergency services, and long-term psychotherapy. The fourth year is primarily elective and residents are encouraged to concentrate on a specific area of interest. The general educational philosophy of the University of Maryland is eclectic with an emphasis on producing clinicians who have had the opportunity to work with small caseloads in depth and use a variety of treatment strategies.

Family therapy was offered to the residents at the University of Maryland as a clinical skill in addition to the practice of traditional psychotherapy, i.e., individual (supportive and psychodynamic) and group; the objective was not to minimize or reduce training in other areas. Attempts were made to integrate the treatment of families into the present service delivery system, and this was possible in the inpatient service. However, a separate family outpatient clinic was established by the director of the Division of Psychiatric Social Work within the Department of Psychiatry primarily to provide experience with outpatient couples and families to trainees of all disciplines. In addition, the director of the social work division was a key figure in the development of the family therapy training program for residents.

An additional goal of the family therapy training program was to teach residents a modality of psychotherapeutic treatment that was not systematically included in the rest of the clinical curriculum. As do many residency programs, the University of Maryland tended to emphasize the teaching of psychodynamically oriented group and individual psychotherapies that relied primarily on 1) nondirective techniques, 2) a long-term orientation, 3) the use of interpretation and clarification as techniques with the goal of greater self-awareness, 4) a focus on transference and countertransference, and 5) a cognitive/affective rather than behavioral orientation. The inclusion of a family treatment approach that was goal-directed, short-term, and oriented toward symptomatic change by means of directives and in which insight was a secondary rather than a primary goal would provide residents with skills that they were not learning in any of the other psychotherapeutic training programs. The fact that these techniques of psychotherapy seem to be more appropriate for the severely disabled patient than what is usually taught has made the training more relevant to psychiatrists, who are increasingly working with such patients.

A process of development by trial and error preceded the present program, which is described below. A great deal of the outcome depended on idiosyncratic circumstances, luck, the personalities of certain faculty and residents, and the professional resources in the local area. This educational program is not being presented as an ideal model suitable for adoption in toto

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by other psychiatry departments. Certain elements are acceptable and others can stand improvement or would need to be changed to fit the needs of a particular department of psychiatry. Nevertheless, the program is successful in accomplishing the objectives of training residents to autonomously practice marital and family therapy and of teaching them a psychotherapeutic orientation appropriate for the more disturbed patient.

THE TRAINING FORMAT

First and Second Years

The first postgraduate year rotation, the psychiatric internship, has only recently been created, and family therapy training and supervision requirements are still evolving. Presently, first-year residents receive once a week supervision in family therapy during their six-month inpatient rotation, but the training during this year is less intensive. Second-year residents are assigned a supervisor in family therapy in addition to three supervisors for individual psychotherapy (which includes individual assessment and ward management) and one for child and adolescent therapy. Each resident and supervisor meet a minimum of one hour per week. Most of the residents seek out either more time from the same family supervisor or extra supervisors who are available. As more of the supervisors for individual patients have become proficient in family therapy, they have become an extra resource for training.

At present, four years after the implementation of this program, over 85% of inpatients are treated by conjoint family therapy on a weekly basis. In 1977–1978, 73% of all inpatients were treated with family therapy; in 1978–1979, this figure was 92%. Residents treat between 35 and 50 families during their second year; the number of sessions per patient ranges from 2 to 50. As the average length of stay for inpatients is approximately 30 days, the majority of them are seen with their families three to four times for brief intensive intervention. The age, sex, and socioeconomic status of the patients varies widely as does the family composition. Couples are treated, as well as families with two generations, single-parent families, and other configurations.

These statistics highlight the fact that the inpatient service has become a major training site for family therapy. This work with inpatients and their families provides the resident with enormous experience in the treatment of various types of families and ensures a great sense of confidence in the handling of almost any situation. Most of the family supervisors use one-way mirrors or videotapes for supervision. The curriculum emphasizes direct clinical involvement with families at the beginning of the training program and close, one-on-one supervision.

Two seminars in family therapy and theory are given for the second-year residents. An eight-week introductory course is taught at the beginning of the training year. The course covers such issues as how to conduct initial interviews with a disturbed inpatient's family, special techniques for handling such families, how to engage resistant families, and how to give directives (1–3). This seminar is designed to familiarize the residents with some of the basics of family therapy techniques and to give a brief outline of some of the therapeutic principles underlying this approach to psychiatric symptomatology.

Later in the year, as part of a basic psychiatric concepts seminar, an eight-week course in family theory is given. Here the focus is on the historical development of family theoretical concepts, including an overview of the various models and therapists, presentations of general systems theory, communications theory, Bowen's family theory, strategic/structural family theory, and recent research articles focusing on outcome as well as family interactions of schizophrenic patients. Some classical papers are assigned for reading, particularly earlier papers concerned with the family dynamics of schizophrenia and articles that first presented new theoretical linkages (4–14). As many medical schools now include courses in normal family and marital development, an attempt is made to avoid repeating the teaching of this information.

Third Year

The third year is the outpatient year for most residents; traditionally it emphasizes learning psychodynamic psychotherapy. The residents are required to treat a minimum of one family or couple and receive a minimum of one hour of supervision a week by a family therapy supervisor. Some attempts have been made to cluster the family supervisors who have an experiential and psychodynamic orientation during this year, partly because this is more congruent with what the residents are learning in other areas.

Fourth Year

The fourth year is an elective year for all residents at the University of Maryland and has become a major part of the family therapy training program due to the option of obtaining supervision from Jay Haley and Cloe Madanes, who direct the Family Therapy Institute of Washington. All fourth-year residents can elect this supervision one day a week for nine months. The training consists of one hour of supervision by means of a one-way mirror and videotape, a one and one-half hour seminar oriented toward strategic/problem-solving family therapy, and several hours of observation of other therapists being supervised live. A number of training videotapes made and narrated by Jay Haley are shown during the seminar. This part of the overall program offers the residents an intensive experience in two methods of family therapy: strategic and structur-

al. This has been a popular elective in each of the three years that it has been offered, and each year an increasing number of residents have signed up. Almost all child fellows have taken this elective. Residents who choose not to take this course are required to have one hour of family supervision and treat a minimum of one outpatient family or couple.

After completing these four years (three and one-half years in psychiatry), the average resident will have evaluated and treated approximately 75 to 85 outpatient and inpatient families. Residents also will have received a minimum of 175 hours of one-on-one supervision in family therapy. They will have received approximately 70 hours of didactic seminars and another 70 hours of observing other residents working with families. Residents who have completed the four years in the program will have received individual supervision in approximately 500 to 600 hours of marital and family therapy. These hours are in addition to the attention paid to the assessment and treatment of families by a resident's clinical supervisors in areas other than family therapy. In our program the major emphasis is on the teaching of clinical skills by means of close supervision, with less emphasis on acquiring theoretical knowledge through didactic instruction. Unfortunately, many graduate education divisions attempt to teach sociocultural perspectives (including a family systems model) about mental illness by offering seminars only. This reliance on didactic instruction rarely requires the young psychiatrist to change his or her clinical identity and behavior, which are based on treating a unit of one. Residents must learn new clinical skills and adopt new conceptual models when treating dyads or triads within families.

FACULTY RESOURCES

At the University of Maryland, the situation before the implementation of a comprehensive training program for residents was such that only a few faculty members, mostly social workers, offered elective family therapy supervision. Some of these social workers had been blocked by the psychiatrists on the faculty when they attempted to move toward a more rigorous nonelective training program for residents. There were problems with the training and experience of some of these nonphysician faculty. Many had not received advanced clinical training themselves in family therapy or had not worked extensively with the families of severely disabled patients. Nevertheless, these individuals provided the only teaching available to residents for many years, often in the face of considerable hostility and prejudice by their physician colleagues. Unfortunately, most residents graduated in the past without learning much beyond the most superficial knowledge. Even more disturbing is the fact that many of these residents who had learned so little viewed themselves as competent in the delivery of this type of therapy when they graduated.

A more comprehensive and high-quality educational program began to develop when some of the faculty pursued intensive postgraduate, formal clinical training in family and marital therapy and then began to teach. Some of the physicians on the faculty were included in this group, giving the teaching of family therapy more credibility in the eyes of residents and physician/faculty members. The director of graduate education began to push more aggressively for the hiring of more qualified faculty. The National Institute of Mental Health (Psychiatric Education Division) played a part in this by acknowledging deficiencies in the family therapy training program, which led to the hiring of Jay Haley and Cloe Madanes on a part-time basis to train both faculty and residents. The faculty who received this training were primarily social workers who had been teaching family therapy to residents for years but who benefited from an upgrading of their own skills. Naturally this led to an increasingly sophisticated core faculty who could teach residents in all four years of the program. The benefits of this enriched training program are being realized as psychiatrists who graduate from the program are now providing supervision to their younger colleagues in the residency as well as to medical students. In 1980, 11 psychiatrists were serving as family therapy supervisors.

ADVANTAGES AND DISADVANTAGES

The ideological struggles between faculty members who adhere to the biological/genetic, psychodynamic, or behavioral approaches are confusing enough for most residents. A family systems perspective, still another disparate and complex way to explain human behavior, must survive and evolve within this confusing and competitive intellectual milieu. Residents and faculty need to be flexible and tolerant to accept the integration of an additional model. One of the most important positive consequences of adding a comprehensive family therapy training track to a residency program is that it provides the resident with the opportunity to truly understand and implement clinically a biopsychosocial model. The psychiatric resident can incorporate in clinical work with a single family the prescription of medication, the giving of directives to alter the family organization, and the use of interpretations to alter self concepts; all of these interventions are based on diverse but potentially interlocking theoretical principles.

In my opinion, part of the resistance to teaching family therapy in residency programs has been due to the fact that most of the trained faculty/staff are not physicians. When a non-M.D. attempts to supervise a young physician in training, problems can arise because the resident does not have a clear and congruent

professional role model. Sometimes if a family case is not going well and the supervisor is a non-M.D., the resident will begin to experience some doubt and mistrust of the supervisor based on this lack of professional identification. The resident may then resist supervision and try to assert his area of expertise, diagnosis and somatic interventions, in order to compete with the supervisor. It is a difficult situation for the supervisor as he has to try to support the resident but must also not allow his authority to be undermined. These circumstances often provoke the latent ambivalence of other faculty members about the teaching of family therapy. A frequent question is, What are the indications and contraindications for family therapy? Unfortunately this question is often answered by a professional who has never treated a couple or family. These conflicts heighten the resistance of residents to learn family therapy and are particularly evident in the inpatient setting with younger residents, who are more insecure about their identity as physicians. In this context it is desirable that the resident have for a role model a physician who practices family therapy. At a later stage of the resident's training, the issue of a non-M.D. supervisor becomes less critical.

In recent years psychiatrists have assertively articulated the necessity of their being the clinically responsible leader and authority of multidisciplinary teams of professionals. However, to fulfill this role psychiatrists must have sufficient experience in the various modalities of treatment, including family and marital therapies, in order to know if a particular treatment is indicated and to be able to monitor the quality of the therapy. Clearly, psychiatrists must learn something about specialized therapeutic modalities if they are to be adequately prepared for their role as clinical leader.

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Impact of the Outpatient Clerkship on Medical Students

BY PAULA F. EAGLE, M.D., AND LUIS R. MARCOS, M.D.

Using an anonymous questionnaire, the authors assessed the professional relevance of outpatient and inpatient curricula and the didactic value of different parts of the clerkship for 80 junior medical students randomly assigned to a pilot clerkship. Significantly more medical students preferred outpatient to inpatient psychiatry; they supported the view that working with outpatients is more useful, rewarding, and educational for the nonpsychiatrist physician. The implications of this study include the need to increase the outpatient psychiatric experience in medical school, the importance of surveying the impact of learning settings on students, and the reasonableness of adapting the curriculum to the didactic needs of students.

The goals of psychiatric education for medical students have received considerable attention in the literature (1–4). However, no consensus has been reached as to how best to achieve these goals. In fact, physicians have reported dissatisfaction with undergraduate psychiatric teaching, especially in regard to the amount of knowledge acquired and the ability to apply what is taught (5, 6).

Although there are few evaluative reports of clinical clerkship programs, there is evidence that both faculty and students find direct patient contact and patient responsibility the most promising methods for teaching psychiatry to medical students (7–13). The type of patient and the format for providing patient contact have not been agreed on. In this regard, the existing literature may be generally divided into those studies which focus on inpatient clerkship settings and those which emphasize outpatient experience.

Two studies of inpatient clerkships in which medical students functioned as primary therapists (7, 8, 13) found that learning was enhanced not only by the students taking direct responsibility but also by the clinical supervision of the students. However, because the majority of the patients had chronic schizophrenia, the students felt that exposure to a narrow diagnostic range of patients gave them a limited view of psychiatry, was not very relevant for the nonpsychiatrist physician, and made them question the therapeutic value of their intervention. Kasuboski and Marshall (11) tried to improve the clerkship by using an acute short-term inpatient setting with a therapeutic milieu orientation. Because of the acute nature of the patients' disorders, the students saw a substantial number of their patients improve. This experience reinforced the students' inclination to treat similar patients in their later practice (11). In our earlier work we found that medical students who treated patients with a better prognosis in an acute treatment inpatient unit, compared with students who treated chronic patients, changed their attitudes toward the mentally ill to those which are known to facilitate treatment (14) and became more interested in psychiatry as a specialty (15). We suggested that a critical issue affecting the students' attitudinal change was their perception of the treatability of the patients.

Hunt and associates (16) developed a clerkship in which evaluation of outpatients was the central experience for the students, who also functioned as the primary physician. Both faculty and students indicated that this constituted a highly successful learning experience. Medical students have also rated as most useful the learning experiences in outpatient psychiatric clinics and emergency rooms, where they diagnose and treat patients as primary physicians within the medical model (10). Further, Kress and associates (17) and Maxmen (18) found that students' frequent therapeutic successes in short-term outpatient crisis interventions and consultation-liaison helped them develop positive attitudes toward psychological approaches to patient care, psychiatry, and psychiatrists.

In summary, although the majority of psychiatric clerkships use inpatient settings, an increasing body of literature points to the educational value of outpatient psychiatric experiences. In this paper we will present the effects of a pilot outpatient psychiatric curriculum on medical students undertaking their psychiatric clerkship. We will focus on the students' own re-

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sponses and evaluation of this outpatient component as well as their comparison of it with the more standard inpatient curriculum. We believe that in psychiatric education it is particularly important to monitor and take into account the students' feedback because it often reflects not only the didactic value of the curriculum but the students' image of psychiatry and their attitudes toward mentally ill patients. Furthermore, Johnson and associates (19) found students' ratings of the goals of psychiatric education to be similar to those of psychiatrists and nonpsychiatrist physicians.

PROCEDURE

Description of the Clerkship

At New York University School of Medicine the psychiatric clerkship is part of the junior-year curriculum and lasts for five weeks. For the purpose of this study, 80 junior medical students were randomly assigned to a pilot clerkship with both an outpatient and an inpatient component. The average age of the students was 24 years, there were 49 men and 31 women in the group, and there was an average of 5 students per rotation. Neither the students nor the faculty had any knowledge of the aim of this evaluative project.

Outpatient Component

The students' outpatient psychiatric experiences took place at Gouverneur Hospital. The curriculum primarily included direct clinical work with outpatients and individual supervision with emphasis on the different modalities of psychotherapy and chemotherapy used with outpatients. Students also participated in crisis intervention in the community and in clinical seminars oriented to issues of outpatient psychiatry. Each student functioned as the outpatient physician and worked with patients individually. The patients' diagnoses included neuroses, personality disorders, and nonpsychotic affective disorders. They participated in the treatment voluntarily.

Inpatient Component

The inpatient part of the clerkship took place in the inpatient teaching unit of Bellevue Psychiatric Hospital, which consists of 24-hour adult receiving wards. The students were responsible for evaluation and short-term treatment, primarily chemotherapy under faculty supervision. After a short stay, the patients were either discharged to the community or transferred to a state facility for long-term treatment. Students also participated in theoretical seminars on major clinical areas of psychiatry. The most common diagnoses included chronic undifferentiated schizophrenia, alcohol addiction, and acute schizophrenic episodes. Approximately 50% of the patients were admitted on an involuntary basis. Although this figure may appear unusually high, it is representative of many inpatient units in urban areas like New York City, where municipal and state psychiatric hospitals constitute the training ground for most medical students.

Evaluation

To evaluate the impact of the outpatient component of the clerkship on the medical students, at the end of the clerkship the students were required to complete anonymously an extensive evaluation questionnaire in which they were asked to address two issues: 1) to choose between and compare the outpatient and inpatient curricula as to their value to them as prospective physicians and 2) to rate the different parts of the clerkship and describe in detail what they had learned.

RESULTS

All of the students participating in the study completed the questionnaire and made extensive comments about their experience. Of the 80 junior medical students, 51 (64%) selected the outpatient setting as more valuable to them as prospective physicians, 15 (19%) chose the inpatient clerkship; and 14 (17%) would not make a choice, stating they felt that both outpatient and inpatient experiences were essential ($\chi^2=33.33$, df=2, $p<.001$).

The students' comments illustrate the reasons for their preferences. In their choice of outpatient over inpatient settings the majority of the students emphasized the patients themselves as the decisive factor. The students saw the outpatient as someone to whom they could relate, understand, and help as compared with the inpatient. For example, one student wrote, "The outpatients could function and were much easier to empathize with, and that made me feel as though my efforts were worth something to the patient." Another commented, "We only see the most critical or chronic inpatients. As a consequence there is a feeling of hopelessness because you cannot improve the patient's condition except for the effect of medication." In addition, the students felt that working with outpatients was more useful to the future nonpsychiatrist physician. One student indicated, "Most physicians will have to deal with patients on an outpatient basis and therefore should learn how to detect underlying emotional or psychiatric problems and then how to help the patient deal with them." Another student: "Seeing outpatients is more relevant and valuable because in our practice we are more likely to see patients who are not psychotic but who have problems coping with day-to-day life."

Although a majority of the students felt that working with outpatients was more instructive, a number of students indicated in their comments that exposure to both types of patients was important to gain a comprehensive view of psychiatry. For instance, one student

remarked; "Both experiences are very valuable because one without the other would give you a skewed view of psychiatry," and another wrote, "Although I felt that outpatient experience was more valuable, I believe that inpatient experience does have merit even for those not going into psychiatry. The ward provides clear examples of different mental disorders and allows students to play a major part in their management."

In their ratings of the different activities of the clerkship, the students found the direct evaluation and treatment of patients the most valuable component of the clerkship. They considered the individual supervision second and theoretical seminars of the least value to them. When the students were asked to state what they had learned, they emphasized two major areas: the exposure to different treatment modalities and the exposure to different types of patients during their outpatient clerkship experience. For instance, one student wrote, "I learned that all patients are not grossly psychotic, and I learned how to deal with patients and their illnesses while they are still coping with reality." Another representative comment: "The most valuable learning experience was the wider range of psychiatric treatment that I saw with outpatients."

DISCUSSION

The results of this study indicate that during their psychiatric clerkship a majority of medical students selected the outpatient psychiatric experience as the most valuable to them as prospective physicians. Although the outpatient and inpatient settings were different in other ways than the type of patients (e.g., faculty and seminars), the students emphasized the learning value of direct patient responsibility and being able to understand and help the outpatients. Furthermore, they felt that outpatients were more likely to be the type of patients they would see in their future nonpsychiatric practice. This finding supports the literature suggesting that students should be exposed to the kind of psychopathology that future general practitioners, internists, or surgeons face in their practice and that the clerkship should focus on developing the skills nonpsychiatrists will need in the future (3, 6, 10, 15, 16, 19–21). Our findings also indicate that a critical issue affecting students' learning of and interest in psychiatry is the assignment of patients with whom they can work, relate, and achieve some change. In recent years there has been a substantial increase in outpatient as compared with inpatient service demands (22); a realistic view of psychiatric education should therefore include an outpatient experience.

Programmatically, there are some limitations to the outpatient curriculum. The process of patient selection necessitates more active participation and coordination by the instructors. The relative unpredictability of patients' attendance may also be a problem at times. It has been our experience, however, that after their first interview patients rarely miss their appointments with the students. Finally, the brief time available for patient contact with students during the five-week rotation requires the formulation of short-term goals, such as crisis intervention and the stimulation of the patient's motivation for further treatment.

This study brings into question the value and use of medical students' evaluation of their own educational programs. In this regard, we agree with the views supporting the desirability of the students' feedback (19, 23). In our experience, students generally have a realistic idea of the areas of most use to them and how the material may best be learned. A fundamental question is whether the curriculum should be adapted to the different needs and career goals of the students. Recent reports (23, 24) have demonstrated that letting students select among different clerkship settings increases their motivation, learning, and interest in psychiatry. As our understanding of both medical students' personalities and the factors involved in their career goals improve (25, 26), the tailoring of the learning experiences to the students' needs becomes increasingly reasonable.

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Explaining the Increased Arrest Rate Among Mental Patients: A Cautionary Note

BY LARRY SOSOWSKY, M.P.P.

The author compared the annual arrest rates through 1978 of 301 state mental hospital patients released in 1972–1975 with arrest rates of the local county (San Mateo County) population in 1978. The data do not easily allow acceptance of the explanation for the increased arrest rate among ex-patients suggesting that a definable group with previous arrests accounts for the increase. In San Mateo County, ex-patients with no previous arrests were arrested roughly three times more often than the local county population and five times more often for serious violent crimes.

Several papers have appeared recently reconsidering the relationship between mental illness and crime in view of the increased arrest rate among the mentally ill (1–6). Two other studies limited this inquiry to the disposition of arrests for homicide (7, 8). Although these papers have identified several contributing factors, two principal explanations seem to be in contention. They are not mutually exclusive; rather, each reflects a different emphasis.

Grunberg and associates (7, 8) and I (3) suggested that more liberty for the traditional hospital patient is likely a crucial factor in explaining the observed increase. Moreover, mental illness as such seems to be an important factor in accounting for the increase, and, mutatis mutandis, the proportion of patients admitted to state hospitals with previous criminal records has increased. In contrast, Steadman and associates (4, 5) suggested that there are not more mentally ill people at risk for criminal activity in the community, there are simply more criminals in mental hospitals in the first place.

Judith Rabkin (6) recently appraised the literature on criminal behavior among ex-patients and concluded, in agreement with Steadman and associates (4, 5),

> At the present time there is no evidence that . . . mental status as such raises . . . arrest risk; rather, antisocial behavior and mentally ill behavior apparently co-exist. The major factor associated with increases in arrest rates of discharged mental patients . . . is the increased proportion of mental patients who have arrest histories before their hospitalization. (p. 25)

Rabkin noted that my data (3) did not contribute to an

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TABLE 1
Annual Arrest Rates per 1,000 for the San Mateo County General Population, the San Mateo County Ex-Patient Cohort, and Patients with No, One, and Two or More Previous Arrests

Reason for Arrest	General County Population (N=473,471)	Patients with No Previous Arrests (N=141)	Patients with One Previous Arrest (N=39)	Patients with Two or More Previous Arrests (N=121)	Total Patient Cohort (N=301)
Violent crimes	3.5	18.5	43.4	49.6	34.2
Potentially violent crimes	.7	6.5	0	8.9	6.6
Sex crimes	.2	0	7.9	5.1	3.1
Drug crimes	2.3	9.8	15.8	29.2	18.4
Crimes against property	9.7	41.5	63.1	96.6	66.4
Other crimes	22.1	37.1	78.9	156.4	90.5
Total arrests	38.5	113.5	209.1	345.8	219.3

understanding of the causal relationship between mental illness and crime because I did not classify arrests in terms of whether they preceded or followed psychiatric hospitalization. I also did not analyze the recidivism rates of ex-patients with previous arrests. Others (9, 10) have also noted these omissions. Rabkin did not comment on the findings of Grunberg and associates (7, 8). In this report I fill in these gaps.

METHOD

I obtained the criminal histories through 1978 for 301 patients admitted to and released from Napa State Hospital in 1972–1975. I also obtained data to compute general population 1978 arrest rates for San Mateo County, Calif., one of the counties served by the hospital. I analyzed arrests before hospitalization and over a maximum 6½-year follow-up period—through December 1978. I also collected data identifying patients' legal status at the time of admission to supplement social-demographic data and analyzed specific differences between patients.

RESULTS

Forty-one percent (N=124) of the 301 patients were arrested within the follow-up period. Fifty-three percent of the 301 patients (N=160) had been arrested before hospitalization. Of these, 56% (N=90) were subsequently arrested. The correlation between these two factors is .272 (χ^2=57.6, $p<.001$), which strongly confirms the correlations reported by Steadman and associates (4). However, a large proportion of the 141 patients without previous arrest were also subsequently arrested, 24% (N=34) within the follow-up period; of these, 53% (N=18) were arrested within 19 months after hospital discharge. This percentage is three times higher than the largest comparable percentage reported for former mental patients (6).

The principal factor sustaining the contention that the increased arrest rate is simply explained by there being more criminals in mental hospitals is the finding in all earlier studies that patients without previous arrests have postrelease arrest rates less than or equal to those of the general public. Table 1 shows the postrelease arrest rates for my sample of patients without previous arrest, patients with one previous arrest, and patients with two or more previous arrests, along with base rates for the population of San Mateo County in 1978. The striking feature of the data is that not only were patients with previous arrests subsequently arrested at an increasing rate, as expected (4), but patients without previous arrest also had a markedly higher arrest rate in every crime category except sex crimes than did the county population generally. The overall arrest rate for all of the patients was five times that of the county. Patients who entered the hospital without a criminal record were subsequently arrested about three times as often as the average citizen and fives times as often for violent crimes.

I examined several variables to determine if any of them distinguished the patients without previous arrest who were subsequently arrested (N=34) from those patients arrested both before and after hospitalization (N=90) and those who were not arrested at all (N=107). (Seventy of the 301 patients were arrested only before hospitalization.) The variables that appeared most salient were age and legal status at the time of admission (see table 2).

DISCUSSION

To conclude that mental illness and antisocial behavior are independent because of the strong relationship between arrests before hospitalization and arrests after hospitalization is, it seems to me, premature if not mistaken. Although this relationship was confirmed in San Mateo County and Napa State Hospital, it does not sufficiently account for the high arrest rate among mental patients. Patients without previous arrest had a postrelease arrest rate markedly higher than that of the general public. This would seem to suggest that mental status, as such, is causally related to the increased arrest rate. It is true, however, that patients who had no previous arrests were underrepresented

TABLE 2
Demographic Characteristics of 231 Patients Discharged from a Mental Hospital[a]

Characteristic	Patients Arrested Before and After Hospitalization (N=90)	Patients Arrested Only After Hospitalization (N=34)	Patients Never-Arrested (N=107)
Mean age (years)	32	25	34
Percent hospitalized voluntarily	6	26	26
Percent hospitalized involuntarily	94	74	74
Percent men	80	74	56
Percent women	20	26	44
Percent white	77	85	84
Percent nonwhite	23	15	16
Percent schizophrenic	73	73	75
Percent nonpsychotic	27	27	25

[a]Demographic characteristics of 70 patients arrested before hospitalization but not after are not given.

among the patients who were subsequently arrested: patients with no previous arrests constituted 47% of the entire sample but only 27% of those subsequently arrested. Notwithstanding this observation, the inference of causality is supported by the findings of Grunberg and associates (7, 8).

There are now more patients in mental hospitals who have been arrested before: 53% of my sample had been arrested before, and Steadman and associates' 1968 sample contained 31.9% and their 1975 sample contained 40% (4). This finding appears to be due to the fact that those who would have been institutionalized according to traditional legal standards are in the community and thus subject to arrest (11). Moreover, according to modern legal standards dangerous individuals are released sooner than they would have been in the past. The contention that there are *simply* more criminals in the state hospitals in the first place is not supported.

Why did I find a greater percentage of patients without previous arrest who were subsequently arrested as well as more provocative overall arrest rates than Steadman and associates found in New York? It appears to be due to the fact that in California we set about attaining the goals of deinstitutionalization and civil commitment reform earlier than other states and perhaps with greater zeal. Consequently, we have a more concentrated situation, which is particularly manifest in San Mateo County (12). The principal difference between my sample of inpatients and others (1, 2, 4) is the greater concentration of young schizophrenic patients. That many of the patients without previous arrest who were subsequently arrested entered the hospital voluntarily is probably a reflection of the fact that the pressures operating to limit hospital use in the period under study (13) also affected the voluntarily admitted patient.

These data suggest that knowledge of diagnostic factors alone does not enable a more reliable prediction of recidivism. This finding in the current milieu, however, should not be too surprising, nor should we allow it to mislead us. Gordon (14) explained the implications for prediction of dangerousness and recidivism given current legal standards: a more concentrated group of individuals now entering institutions; perhaps this imposes insurmountable difficulties for isolating more meaningful salient factors.

Perhaps the critical issue involved here is the greatly contested capacity of the psychiatrist to predict dangerousness and recidivism. Doubtless, the new legal relationship between the state and the mentally ill is in part a response to the belief that too much discretion was granted the psychiatrist in the past. Apparently this new relationship imposes an unavoidable social cost. However, we may have passed the degree to which we should constrain the exercise of psychiatric authority. This is not to imply that we should eliminate the current social cost entirely by returning to days gone by. However, we do grave damage, it seems to me, by averting our gaze from this persistent dilemma or taking comfort from the fact that the social cost is not greater.

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Review of 13 Cases of Capgras Syndrome

BY RAMZI KIRIAKOS, M.D., AND JAMBUR ANANTH, M.D.

The authors describe 13 psychotic patients who had Capgras syndrome. Only 4 patients had evidence of organicity. The authors conclude that Capgras syndrome occurs equally in functional psychosis and in the presence of organicity and recommend that the presence or absence of organicity be determined in every patient with Capgras syndrome.

In 1923 Capgras and Reboul-Lachaux described a woman who was unable to identify others well known to her (1). They called this condition Illusion des Sosies, but it has since become known as Capgras syndrome. The hallmarks of this syndrome are a systematized delusion of misidentification of a person or persons, objects, or animals. Physiognomy, attributes, mannerisms, and clothing are well recognized, even though the integrated whole is not recognized and is considered to be a double. Most cases have been described in women. There is considerable debate as to whether Capgras syndrome is a specific clinical syndrome (supported by the French school) or a symptom common to many conditions (supported by the German school).

In 1924 Capgras and Carette (2) described a schizophrenic girl who alleged that her parents sent doubles of themselves to visit her. In 1931 Larrivé and Jasienski (3) described the case of a paranoid psychotic woman who claimed her lover, who was not financially well off, had a rich and aristocratic double. In 1933 Coleman (4) described a case that extended the doubles to inanimate objects; the patient claimed that the letters from her daughter had been written by somebody else. In 1936 Murray (5) for the first time described a male schizophrenic patient who claimed that doubles impersonated his parents. Later, Davidson (6) described two cases—one a male patient who claimed that his wife had a double and the other a 73-year-old paranoid schizophrenic man who believed that the devil had displaced his wife in the form of a double who advised him to commit suicide. In 1967 Enoch and associates (7) supported the prevailing opinion that the Capgras syndrome occurred only in patients with affective disorders or schizophrenia: "Since it occurs in a clear sensorium, it is obvious that there is no organic basis to the condition, but it is rather a functional illness and as such, is explained on a psychodynamic basis" (p. 7).

In 1930 Vié (8) advanced the hypothesis that the Capgras syndrome was an organic delusional psychosis. Gluckman (9) and Weston and Whitlock (10) each reported a case that occurred in the presence of apparent organic brain disease, and MacCallum (11) described 5 more such cases. Hay and associates (12) discussed a middle-aged mentally retarded woman with pseudohypoparathyroidism who developed Capgras syndrome after a course of ECT. Her EEG revealed a focal abnormality in the left temporal region. Interestingly, the EEG dysrhythmia coincided with the florid Capgras symptoms and improved as the other symptoms resolved. Faber and Abrams (13) presented the case of an XYY male with symptomatic schizophrenia who, in addition to mild mental retardation, displayed Capgras syndrome; his EEG revealed predominantly left-sided slow activity. Hayman and Abrams (14) reported two cases of Capgras syndrome in association with severe brain disease. Christ-

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odoulou (15) described 11 patients with Capgras syndrome, 8 of whom had both clinical and laboratory findings suggestive of organicity.

METHOD

All of the 13 patients with Capgras syndrome whom we evaluated were psychotic, and the nosological entity in which the syndrome developed was paranoid schizophrenia for 10 patients, undifferentiated schizophrenia for 1 patient, borderline personality disorder for 1 patient, and hysterical psychosis for 1 patient.

There were 7 men and 6 women, whose ages ranged from 18 to 50 years (average=33 years). For 5 the Capgras syndrome appeared simultaneously with the major psychotic illness, and for the remaining 8 the onset was between 2 and 8 years after the basic psychotic illness began.

We investigated organicity by taking a detailed history and doing a neurological examination, which included X rays of the skull and an EEG; psychometric tests were conducted in those patients with an abnormal EEG. Organicity was not detected on formal mental status examination in any of our patients. All the patients were alert and had a clear sensorium. We found definite EEG abnormalities in 4 patients: 2 had focal temporal paroxysmal abnormalities and the other 2 demonstrated diffuse slowing; 2 were episodically disoriented on few occasions, which was not related to the severity of the Capgras symptoms. Another patient who had Capgras syndrome persistently for 17 years was reported to be disoriented, agitated, and delirious on the second of his six hospital admissions; however, he had a normal EEG and skull X rays on two different admissions.

Our findings indicate that the basic double for married, divorced, or separated patients was the spouse (N=3), a sister (N=1), a psychiatrist (N=1), and a father (N=1). For 3 of the single patients, the basic double was often their father and less often their mother; for the remaining 4 single patients, the basic double was a sister (N=3) or an exceptionally close cousin (N=1). Nine of the 13 had more than one double and extended it in most cases beyond the immediate family, commonly to the hospital staff or the treating psychiatrist. In addition, for 3 patients, the double included inanimate objects—the hospital buildings (N=1), the entire city of Los Angeles (N=1), and a car (N=1). Unlike what has been described in the literature (16, 17), less than 50% of the patients (N=6) had negative feelings toward the double; approximately 25% had positive feelings (N=3), and 33% were ambivalent (N=4).

All 13 patients had been treated with neuroleptics, and 3 had had ECT. The Capgras syndrome had disappeared completely in 1 patient after 1 week and lasted between 3 and 12 months in 8 patients. Three of the 4 patients who had had protracted delusion had also had ECT. We have followed 3 patients whose Capgras syndrome persisted for 4–17 years; 1 had had Capgras syndrome for 1 year when we lost track of him.

DISCUSSION

The rarity of Capgras syndrome is indicated by the fact that it took us over 8 years to collect this series of 13 cases, which we obtained from four different university hospitals. The age of our patients tended to be lower than the average age of patients reported by other authors who stressed the etiologic importance of organicity. The average age of our patients was 33 years; the patients studied by MacCallum (11) averaged 52 years of age, and those studied by Hayman and Abrams (14) averaged 44 years of age. We detected organic changes in only 4 of our 13 patients. In fact, 1 of our male patients who had the syndrome for over 17 years did not manifest organicity on clinical examination during his illness and his EEG was normal on two occasions. We think it is possible that the relationship of organicity and Capgras syndrome alluded to by these authors may be associated with age.

Although initially the syndrome was reported in women only, the first male patient was reported by Murray in 1936 (5). The higher number of men in our sample (7 men, 6 women) may have been the result of our having obtained 2 patients from an all male Veterans Administration hospital population. Since the first male patient, described by Murray (5), was a latent homosexual, it has to be postulated that only homosexual males develop the syndrome (18–20). However, we noted no homosexuality in our 7 male patients.

Our data suggest that Capgras symptom can occur in functional as well as organic states. We observed it in schizophrenic patients and in patients with borderline states and hysterical psychosis. Furthermore, for many patients this symptom improved when the primary disorder was treated. All these factors suggest that the delusion of doubles is a symptom and not a syndrome. In our opinion, the Capgras syndrome occurs equally in functional psychosis and in the presence of organicity, but we feel that the presence or absence of organicity should be determined in every patient with Capgras syndrome.

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Screening for Adult Phenylketonuria in State Psychiatric Hospitals

BY ALLAN BROCK WILLETT, M.D., JEFFERSON E. NELSON, M.D., AND DANIEL K. FOSS, M.D.

The authors report the results of screening patients admitted to a state psychiatric hospital for unrecognized adult phenylketonuria. The results suggest that unrecognized adult phenylketonuria is very uncommon in these patients.

The elucidation of the metabolic pathways and the development of easy screening techniques for the detection of phenylketonuria (PKU) provided the basis for one of the major accomplishments in preventive medicine of the last 24 years, namely, the effective screening of newborn children for PKU. Identification of infants with PKU made it possible to prevent mental retardation by controlling their diet. A massive literature concerning the incidence of abnormalities in phenylalanine metabolism and the success of neonatal screening programs in various countries has occurred since easy screening techniques were developed. Behavioral disorders encountered in the mentally retarded child with PKU have been reported occasionally in the literature (1-3). In 1973 Perry and associates (4) reported unrecognized adult PKU in psychiatric inpatients and recommended that ferric chloride urine screening tests be performed on all psychotic patients on admission to mental hospitals "toward correcting the tendency of such hospitals to give stereotyped diagnoses and treatments to all admitted patients." Fisch and associates (5) subsequently reported treating schizophrenic symptoms in a man with known PKU with a low phenylalanine diet; their treatment was based in part on the 1973 article (4). Although the dietary treatment lowered serum phenylalanine without decreasing symptoms, Fisch and associates recommended seriously considering PKU in the differential diagnosis of schizophrenic symptoms.

Recommendations for laboratory screening procedures on psychiatric inpatients are not uncommon; they are usually prompted by a case report of one or two missed diagnoses on a psychiatric unit (4, 6). Even though concern about overuse of laboratory tests is widespread, the yield of laboratory screening procedures with psychiatric patients is seldom analyzed (7). Nevertheless, the persistence of the idea that unrecognized adult PKU causes psychotic symptoms prompted us to investigate psychiatric inpatients.

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METHOD

We screened 635 consecutive patients admitted to the General Adult Psychiatry Unit of the Colorado State Hospital over 8 months for urinary phenylpyruvic acid by testing the first voided morning urine after admission. The method used was the dry reagent ferric ammonium sulfate technology described by earlier authors (8, 9). The sensitivity of this method is 99.5% when urinary phenylpyruvic acid levels are 15 mg/100 ml or more and 82% for levels of 8 mg/100 ml or more (9). Neither false positives nor false negatives occurred in a sample of 25 patients known to have PKU (9). No patients were excluded, even if they had received phenothiazines or other medications before testing, because any specificity error would be caught by follow-up study of serum phenylalanine levels. Those patients who had a positive ferric ammonium sulfate test subsequently gave informed consent for us to obtain a serum sample to test for phenylalanine levels. Patients who had a weakly positive test had repeat morning urines checked. Serum phenylalanine levels were obtained if mental retardation were present or if repeat urine screens were positive.

The patients came from all over the state, and most of them had been sent for admission by a local mental health center. Demographic data were not available, but we presume that the patients represented an unselected population of state hospital admissions.

The primary discharge diagnoses were as follows: 44.5% were psychotic (37.2% had schizophrenia, 4.2% had other functional psychoses, and 2.1% had organic psychoses), 11% were alcohol or drug dependent without psychosis, and 44.5% had other nonpsychotic diagnoses. The diagnosis of mental retardation was made in 3.3% of the psychotic and nonpsychotic patients.

RESULTS

The dry reagent urine screens on 635 consecutive admissions revealed only 6 patients with weakly reactive ferric ammonium sulfate tests. Two of these patients, both of whom were taking phenothiazines at the time of screening, showed negative urines on repeat testing. One patient who was not taking phenothiazines showed a negative urine on repeat testing. Two other patients, the only ones with positive tests who had a diagnosis of mental retardation, had low normal serum phenylalanine levels (both were less than 2 mg/dl). This represents a false positive rate of less than 1% (5 of 634 tests). The last patient, who had a diagnosis of hysterical personality disorder, refused to participate in further study. Of the 635 patients, 629, many of whom were taking phenothiazines, had completely negative urines.

DISCUSSION

This study failed to demonstrate a substantial prevalence of undetected adult PKU in a population of patients admitted to a state hospital by use of the dry reagent ferric ammonium sulfate method of urine testing. These results would suggest that PKU screening of adult psychiatric populations in the United States is of very low yield. This may be an indication of how thoroughly this population has been screened at birth or by the failure of patients with PKU to survive to adulthood.

Although low urinary phenylpyruvic acid (less than 15 mg/100 ml) may be missed by the dry reagent (false negatives), the 2 patients most likely to have had PKU had unequivocally low serum phenylalanine, suggesting that the screen is not insensitive. The findings suggest that subclinical PKU does not occur in this population at an appreciable frequency.

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CLINICAL AND RESEARCH REPORTS

This section contains 1) new research findings, including preliminary data from pilot studies, either clinical or laboratory; 2) worthwhile replication studies; 3) case reports that describe a truly new syndrome or cast new light on established ones; and 4) case reports that indicate a new therapeutic procedure of potential value or call attention to adverse effects of drugs or previously unreported complications of therapeutic interventions. Program descriptions and literature reviews cannot be printed in this section. Criteria for format are listed in "Information for Contributors" in each issue; papers that do not adhere to these criteria will be returned to the author.

Atypical Major Depression in a Patient with Severe Primary Degenerative Dementia

BY GEORGE W. DEMUTH, M.D., AND BARRY S. RAND, M.D.

Conflicting reports appear in the geriatric literature concerning the existence, presentation, and treatment of depression in the elderly patient with severe dementia (1–4). Major depression, however unusual, does occur in the severely demented patient and may be successfully treated (5). Accurate diagnosis of depression during acute deterioration may be difficult because the depressive symptoms may be confused with organic states (6), the depressive syndrome may be atypical (7), and the criteria for diagnosis of depression under these conditions have not been clearly elucidated (8).

We present a case of an elderly man with documented severe primary degenerative dementia who developed an intercurrent major depression that responded to ECT. This is done in an effort to depict a clinical picture that warrants consideration of depression, to describe and explain the atypical features of the depressive syndrome under these circumstances, and to illustrate our conceptualization of the interaction between depression and dementia.

Case Report

Mr. A, an agitated 80-year-old married man, was admitted to the hospital after he threw statuettes at home while yelling, "I'm a crazy dead man. I'm going to my funeral." A 10-year history of primary degenerative dementia with mild depression characterized by progressive memory loss, apathy and drowsiness had been documented 2 years earlier. At that time Mr. A had an abnormal mental status examination and a CT scan that showed cerebral atrophy and mild ventricular enlargement. Low doses of thiothixene and desipramine relieved Mr. A's agitation and depressive symptoms. He remained stable until 6 weeks before his admission. During this time he experienced increasing confusion, bizarre behavior, and agitation while he was being treated with a combination of levodopa and carbidopa for parkinsonism; these symptoms led to his admission.

On admission Mr. A, who was dressed in pajamas, was unkempt, agitated, drowsy, frowning, and unable to relate to the interviewer. His speech was slow and sparse. His mood was depressed, and his affect was constricted, intense, and inappropriate. He had no hallucinations, delusions, or derailment, but there was loss of reality testing. He was disoriented to time and place. He was amnesic for recent events and unable to perform simple calculations. No overt suicidal or homicidal ideation was elicited. His judgment was severely impaired. Abnormalities found on physical examination were a marked shuffling gait, moderate cogwheel rigidity, and partial mask-like facies. His EEG was abnormal (diffuse 4–6 cycles per second background slowing and semi-arrhythmic, sharply contoured activity in the frontal regions, more on the right hemisphere); a CT scan showed mild to moderate atrophic hydrocephalus. All his other tests were normal.

All medications were stopped for an evaluation. Three weeks after admission Mr. A was started on 3 mg of haloperidol b.i.d., resulting in decreased combativeness. However, he was withdrawn, sulky, and mute, and he continued to refuse to wash, eat, or rise from his bed. At times he wandered naked from his room, mistook staff for his family, and became agitated at night. He yelled, "Dead people don't have to eat" and "I'm at my funeral." He began crawling, urinating in corners and spreading feces on the walls. When he was stressed, he yelled, "The priest is coming, I'm dead" and slid to the floor. With increasing parkinsonism and con-

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fusion, Mr. A's haloperidol was stopped. Brief trials of 2 mg of trihexyphenidyl b.i.d. and 25 mg of diphenhydramine t.i.d. were given, which resulted in florid atropine-like psychosis, confusion, and drowsiness.

Despite concerns that ECT might cause further deterioration by increasing the organic symptoms, unilateral modified ECT was begun following reevaluation and the obtaining of family consent. Antidepressants were not tried because of Mr. A's anticholinergic sensitivity. After several treatments, he dressed himself, walked unaided on the ward, and developed normal hygiene. Mr. A's agitation, uncooperativeness, and cries of being dead ceased. A total of eight ECT treatments, 3 times a week, were given. His mood lifted to the normal range; he smiled, played cards, and enjoyed family visits and personal items. He spoke clearly in simple phrases. Although his confusion had not increased, he continued to wander aimlessly. His family noted he was "back to himself."

Discussion

This case illustrates the atypical symptomatic presentation that results from the complex interaction between severe organic mental disorder and depression and the difficulties encountered in establishing the correct diagnosis. Although some depressive features were apparent, symptoms more typical of organic mental disorder, such as cognitive disturbances, poor judgment, regressed and inappropriate behavior, agitation, and poor social functioning, were predominant. Therefore, primary degenerative dementia with delirium and secondary depression and a parkinsonian syndrome were initially diagnosed, but they were later excluded when treatment of these syndromes only maintained or worsened the patient's condition. Reevaluation, diagnosis, and subsequent treatment for major depression with ECT led to improvement.

The absence of the typical depressive syndrome, including a report of depressive affect, may be accounted for by the effects of the underlying dementia. We conceptualize a threefold interaction between major depression and severe dementia that we have found useful in clarifying the atypical symptoms of depression with dementia. These components are as follows: 1) the depressive symptoms are partially obscured by the dramatic, severe symptoms typical of dementia that predominate; 2) the elaboration and expression of typical depressive behavior, thought, and affect are severely hampered by the patient's decreased cognitive capacity and may be replaced by simpler equivalents (i.e., slumping to the floor replaces statements of loss of interest, or sulking and frowning replaces statements of feeling depressed, or simple statements like "I'm dead" replace complex suicidal ideas); and 3) the decreased functioning due to depression is synergized by the functional deficits of dementia leading to unusually disturbed, regressed behavior (i.e., urinating in corners, smearing feces, crawling).

Using this conceptualization to reevaluate the patient, we note that the diagnostic criteria for major depression (*DSM-III*) are met. Depressed affect is exhibited through frowning and sulking, loss of interest through slumping to the floor, and suicidal preoccupation by yelling "I'm dead." Eating and sleeping disturbance, agitation, and inability to function and concentrate are manifestations of depression as well as dementia. Severe regression is evidence of depression acting synergistically with dementia. Successful treatment with ECT helps confirm our hypothesis of major depression because it is unlikely that ECT would successfully reverse an organic mental disorder.

Because depression in the elderly patient with dementia may be successfully treated (5, 9, 10), it becomes necessary to scrutinize the symptoms in any case of deteriorating dementia to identify an atypical major depression. Unlike more typical depressions, major depression in the elderly patient with degenerative dementia may be characterized by depressive affect and symptoms expressed through simple statements and behaviors that are partially obscured by the predominance of severely regressed behaviors and symptoms more typical of organic disorders. Major depression may be distinguished from organic mental disorders by the presence of these simple affect-laden statements and behaviors and by the absence of contributing organic factors in addition to nonresponsiveness to symptomatic treatment of organic mental disorders.

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D-Phenylalanine in Endogenous Depression

BY JOHN MANN, M.D., ERIC D. PESELOW, M.D., SELMA SNYDERMAN, M.D., AND SAMUEL GERSHON, M.D.

The catecholamine hypothesis of affective disorders was first described by Everett and Tolman in 1959 (1). It suggested that a deficiency in catecholamines might produce depression and lassitude. A theoretical approach to correct this postulated deficiency of catecholamines is the strategy of "precursor loading." Administration of the metabolic precursors of norepinephrine synthesis such as L-dopa, tyrosine, and phenylalanine might be given to patients in order to alleviate their depressive symptoms.

Four controlled studies evaluating L-dopa in the treatment of depression (2) have yielded mixed results. Gelenberg and associates (3) recently reported improvement in one depressed patient given tyrosine, and Kishimoto and Hama (4) noted decreased plasma tyrosine levels in depressed patients with a corresponding return to a normal tyrosine level upon recovery from the depressive episode.

D-Phenylalanine has been evaluated in several open studies with promising results and in one double-blind study (5) with a reported efficacy comparable to imipramine when an average dose of 200 mg/day of D-phenylalanine is used. A proposed antidepressant effect would be consistent with the reported findings of low urinary excretion rates of phenylethylamine (the decarboxylated minor breakdown product of phenylalanine) in patients suffering from depression with a return to normal values after antidepressant treatment (6). Administration of D-phenylalanine has been reported to increase brain phenylethylamine concentrations in animal experiments, suggesting that this might be the mechanism of action of antidepressants (7).

In the work reported here we evaluated the clinical efficacy of D-phenylalanine at the reported effective dose ranges in endogenously depressed patients who had previously responded to other treatments. We also determined if the dose administered had any appreciable effect on plasma levels of phenylalanine and tyrosine (the hydroxylated product of phenylalanine and the next step in norepinephrine synthesis).

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Method

We studied 11 patients who met Research Diagnostic Criteria (8) for major depressive disorder with endogenous features and obtained signed informed consent from each patient. Patients were drug-free for 1 week and then given a placebo capsule identical to the active medication for a second week (a single-blind experiment). All patients who at that point had Hamilton depression scores of 18 or greater on the first 17 items and who were rated as at least moderately depressed on the Clinical Global Impression (CGI) scale were started on active drug treatment for 4 weeks. The maximum daily dose range was 200–600 mg/day of D-phenylalanine with a mean of 350 mg/day reached in the third week. The patients were seen weekly and rated with the Hamilton Depression and CGI scales. Blood levels for phenylalanine and tyrosine and urinary levels for phenylpyruvic acid were obtained from 8 of the 11 patients 45–90 minutes after administration of a dose at baseline and after each week of active treatment. Phenylalanine and tyrosine levels were assayed by ion-exchange column chromatography (9). The variance in measurement of replicates was less than 4%. Tests for vital signs, CBC, electrolytes, and liver function, as well as ECGs, were carried out on all patients.

Results

The results are shown in table 1. Over the course of 4 weeks, the patients' average Hamilton depression and CGI scores did not significantly change ($p>.05$). We had to drop 2 of the 11 patients from the study at the end of 3 weeks because their clinical symptoms had worsened and they had become suicidal. Only 2 of the 11 patients had any therapeutic effect from the drug, as measured by a 50% reduction in Hamilton depression scores. There was no relationship between clinical improvement and dosage. The only side effects observed were slight heartburn in 2 patients. There were no changes in blood pressure, CBC, electrolytes, or ECG for any of the patients.

The plasma phenylalanine and tyrosine levels for 8 patients (2 of these had responded to D-phenylalanine and 6 had not) were all within normal limits at baseline. As noted in the table, the dose of phenylalanine given did not result in any change in plasma phenylalanine or tyrosine levels for these 8 patients over the 4 weeks of treatment. The patients' urine was negative for phenylpyruvic acid at baseline and throughout the 4 weeks.

TABLE 1
Effect of D-Phenylalanine on Clinical Conditions and Plasma Phenylalanine and Tyrosine Levels in 11 Patients with Endogenous Depression

Treatment Day	Mean Daily Dose (mg)	Hamilton Depression Score[a]		Clinical Global Impression Scale Score[a]		Plasma Levels (mg/dl)[b]			
						Phenylalanine		Tyrosine	
		Mean	SE	Mean	SE	Mean	SE	Mean	SE
−7		22.27	1.44	4.18	.30				
0		22.36	1.12	4.09	.27	1.10	.20	1.27	.19
7	200	18.27	1.87	3.45	.39	1.27	.12	1.39	.13
14	275	18.73	1.91	3.45	.41	1.29	.08	1.43	.09
21	350	18.00	2.44	3.27	.58	1.32	.06	1.41	.10
28[c]	350	18.89	2.44	3.33	.62	1.29	.10	1.45	.16

[a]Scores did not differ significantly from baseline (day 0) at weeks 1, 2, 3, and 4 (two-tailed t test, $p>.05$).
[b]Normal ranges: tyrosine, 0.75–2.50 mg/dl; phenylalanine, 0.50–2.00 mg/dl. Plasma phenylalanine and tyrosine levels for 8 patients did not differ significantly from baseline (day 0) at weeks 1, 2, 3, and 4 (two-tailed t test, $p>.05$).
[c]Two patients were dropped because they showed marked deterioration and suicidal ideation.

Discussion

The results of this study do not support the previously published reports (5) of the efficacy of D-phenylalanine at this dose range in the treatment of depression. A highly significant point to be made is that 8 of these 11 patients previously or subsequently responded to other types of antidepressant therapy (tricyclics and MAO inhibitors). The dosages of D-phenylalanine given here had absolutely no effect on plasma phenylalanine and tyrosine blood levels in the 2 patients who responded and 6 who did not respond. This is not surprising because the D-amino acids are poorly absorbed by the gastrointestinal tract and excreted in excess in the urine. This would suggest that either phenylalanine and tyrosine blood levels have nothing to do with clinical response or that phenylalanine's effect in the 2 patients who responded was nothing more than a placebo effect.

The results of the earlier studies utilizing D-phenylalanine (5, 6) were surprising in view of the fact that the studies involving other catecholamine precursors used much higher doses. The investigators (10) reporting efficacy with L-dopa in depression required doses of 7–8 g, whereas other investigators (11) reporting no response gave the patients doses of 80–120 mg/day of L-dopa. Gelenberg and associates (3) gave tyrosine to one patient in a dose of 100 mg/kg per day and reported a clinical response plus a significant rise in blood tyrosine levels. Thus before D-phenylalanine can be ruled out as an effective antidepressant, it must first be reevaluated at higher doses; then it must be reevaluated in placebo-controlled studies if promising results are obtained. It would also be useful to obtain plasma phenylalanine and tyrosine levels at these higher dosages and observe the correlation between clinical efficacy and plasma levels. This study found no evidence for the antidepressant effect of D-phenylalanine in the dosages reported in the literature.

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Lupus-Like Illness Associated with Chlorpromazine

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To our knowledge, we report here the second documented case of a chlorpromazine-associated, lupus-like illness that manifested itself with dyspnea and bilateral pleural effusions, and we point out that chlorpromazine and other psychotropic medications may induce immunological abnormalities and a lupus-like syndrome. It is important to be familiar with this possibly life-threatening iatrogenic syndrome because the process is reversible when the drug is discontinued. We describe below a patient receiving chronic chlorpromazine therapy who developed dyspnea, low-grade fever, bilateral pleural effusions, immunological abnormalities, elevated antinuclear antibody titer, and polyserositis suggestive of a lupus-like syndrome.

Case Report

Mr. A, a 31-year-old single man, came to our medical emergency room complaining of pleuritic chest pain and a dry nonproductive cough; his chest X ray demonstrated bilateral pleural effusions, the right one greater than the left. He was admitted for diagnosis and treatment.

When Mr. A was 21 years old he was diagnosed as having paranoid schizophrenia after he developed auditory hallucinations and paranoid delusions. At that time he was a college student and active in radical politics. During the next 6 years he was hospitalized many times, including a 3-year stay at a hospital for the criminally insane. For the past 4 years, he had been treated in aftercare clinics. Mr. A never returned to college and had never been employed. He had not married and had no close friends; he lived at home with his mother and his aunt. Since his diagnosis 10 years ago, he had received various neuroleptics, primarily chlorpromazine and haloperidol. During the past 2 years he had received 800 mg/day of chlorpromazine.

A physical examination revealed that Mr. A's temperature was 38.3° C; pulse, 120 beats/minute; and respirations, 30/minute; there was dullness at the base of his right lung. He had no abnormal joint or skin findings, and his spleen size was normal. His erythrocyte sedimentation rate was 90 mm/hour (normal range, 0-15 mm/hour). SGOT, SGPT, and alkaline phosphatase levels were moderately elevated. A chest X ray showed a large right pleural effusion and a small left one; an ECG showed diffusely increased ST segments and T waves consistent with pericarditis.

Mr. A's skin test for tuberculosis was negative. His pleural fluid showed an exudate with 9200 white cells (75% polymorphonuclear). Cultures for bacteria, fungi, and acid-fast bacilli were negative. A pleural biopsy showed acute fibrinous pleuritis. Other laboratory studies showed a serum globulin of 3.6 g/dl with an elevated IgM of 961 mg/dl (normal range, 60-250 mg/dl) in a polyclonal pattern by electrophoresis. A prolonged partial thromboplastin time of 63.3 seconds (normal time, 36.4 seconds) was found and was demonstrated to be secondary to a circulating anticoagulant. Lupus erythematosus preparations were not performed. His serum complement level was normal, and his antinuclear antibody titer was 1:60 in a homogeneous pattern. Anti-DNA antibodies were not detected, and a VDRL was nonreactive. Titers for cytomegalovirus and toxoplasmosis as well as assays for hepatitis and heterophile antibodies were negative.

On admission a mental status examination revealed a heavyset young man who was oriented to person, place, and time and whose memory was intact. He responded to questions in a loud staccato voice and would often giggle. His speech was tangential with occasional derailments. He admitted to auditory hallucinations, and there was a marked suspiciousness to his thoughts. His range of affect was blunted and his mood was predominantly one of barely suppressed anger.

Mr. A's hospital course was characterized by spontaneous defervescence, decreasing dyspnea following the drainage of a pleural effusion, and stabilization of the erythrocyte sedimentation rate to 43 mm/hour over 14 days. Because of the bilateral pleural effusions, the pericarditis without an infectious explanation, and the elevated antinuclear antibody titer, we considered the diagnosis of chlorpromazine-induced polyserositis. Mr. A's chlorpromazine was stopped on the 14th hospital day, and he was discharged on the 16th hospital day with a prescription for 20 mg/day of haloperidol; a chest X ray showed residual pleural effusions. We decided that a clinical rechallenge of chlorpromazine, so that we could more clearly establish the existence of a drug-induced lupus syndrome, would be clinically contraindicated because of the patient's suspiciousness. Mr. A's behavior and mental status were unchanged throughout his hospitalization until the evening before his planned discharge; at that time he struck another patient who he thought was secretly talking about him. Mr. A was discharged later that evening and returned to his mother's home without further incident.

After Mr. A's discharge, a psychiatrist at the aftercare clinic changed Mr. A's neuroleptic from haloperidol to 25 mg of depot fluphenazine decanoate every 2 weeks; this was increased over 4 months to 75 mg every 2 weeks. After his hospitalization Mr. A's functioning was unchanged from that prior to his hospitalization. Follow-up after 5 months showed the patient's pleural effusions had resolved, his erythrocyte sedimentation rate was 5 mm/hour, and the antinuclear antibodies and anti-DNA antibodies were not detectable. His transaminase and alkaline phosphatase levels were normal, and his IgM had decreased to 456 mg/dl. The circulating anticoagulant was still present with a partial thromboplastin time of 54.4 seconds (normal time, 37.5 seconds).

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Discussion

The association between the induction of the lupus-like syndrome and certain medications, including phenytoin, hydralazine, procainamide, and isoniazid, has been described (1, 2). The phenothiazines, thioxanthenes, butyrophenones, and lithium carbonate are reported to induce antinuclear antibodies in some patients receiving these medications chronically (3–5). Although few individuals receiving psychotropic medications develop symptoms and signs suggestive of lupus erythematosus (3), many patients, particularly those taking chlorpromazine, develop immunologic and coagulation disorders. Berglund and associates (6) reported that 26% of a series of 177 women taking chlorpromazine had elevated titers of antinuclear antibodies compared to 2.5% of the 42 controls. None had any symptoms of lupus. Fabius and Gaulhofer (3) reported 8 psychotic patients who developed mild clinical manifestations of lupus while taking chlorpromazine, perphenazine, promazine, perazine, thioridazine, or methotrimeprazine. Gold and Sweeney (7) reported one case of perphenazine-induced systemic lupus-like syndrome.

Bergland and associates (6), as well as Quismorio and associates (8), found that treatment with chlorpromazine often is associated with the induction of antinuclear antibodies; Zarrabi and associates (9) demonstrated that most patients on long-term chlorpromazine treatment develop immunologic abnormalities of a significant elevation of serum IgM and prolongation of the partial thromboplastin time. They found a good correlation between the dose and duration of chlorpromazine treatment and the results of the IgM activated partial thromboplastin time. There were 2 findings that were particularly striking in their study: the presence of antibodies to native DNA in some patients treated with chlorpromazine and the presence of splenomegaly in almost 50% of the patients treated with chlorpromazine.

Drug-induced lupus is a syndrome characterized by clinical and biochemical abnormalities similar to those found in systematic lupus erythematosus; regression of the disorder occurs after discontinuation of the offending drug. The drug-induced syndrome does not exhibit renal or CNS involvement, and there is no consumption of complement; with the exception of hydralazine antibodies to native DNA (the hallmark of spontaneous lupus) are rare (1, 2). The mechanism of induction is not known.

In this case report a schizophrenic patient who took chlorpromazine for several years manifested a low-grade fever, dyspnea, bilateral pleural effusions, elevated erythrocyte sedimentation rate, elevated antinuclear antibodies titer, and ECG changes. These symptoms are compatible with a chlorpromazine-induced lupus syndrome; to our knowledge this is the second reported case in which pulmonary symptoms and signs predominate (10). The patient also had abnormal IgM and partial thromboplastin time findings known to be associated with chronic chlorpromazine ingestion (9). It is important to note that the patient's abnormal X ray and laboratory findings reversed after the chlorpromazine was discontinued.

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A Case Report of Cognitive Impairment and Movement Disorder Associated with ECT

BY ROBERT L. RUFF, M.D., PH.D.

There are few neurological complications associated with ECT. The most common side effects are transient confusion and short-term memory impairment and amnesia for the period of treatment (1, 2). ECT has rarely been implicated in causing parkinsonian rigidity (1) or a movement disorder (3–5). In 4 of 8 reported cases of buccolingual dyskinesia associated with ECT, the patients had received neuroleptics (3). Another patient with transient asterixis following ECT had primary hyperparathyroidism (5). The clinical data are insufficient to implicate ECT definitively in the other cases (1, 4). For some patients ECT has been reported to improve rigidity and dyskinesia. The rigidity and tremor in depressed patients with Parkinson's disease was ameliorated by ECT (6), and one patient's tardive dyskinesia improved following ECT (7). I describe below a middle-aged woman without a history of neuroleptic use who developed a movement disorder, rigidity, and cognitive impairment during a course of ECT. Her symptoms partially abated over 2 years.

Case Report

Ms. A, a 53-year-old woman with bipolar disorder, was admitted to the Payne-Whitney division of the New York Hospital with a 6-day history of manic behavior characterized by pressured speech, flight of ideas, and hyperactivity. She had six prior manic episodes in the past 20 years, each of which was treated successfully with ECT. Ms. A had never received neuroleptics. She developed severe diarrhea with subtherapeutic doses of lithium carbonate, and consequently was not maintained on lithium carbonate. The only complication she had had with ECT was transient short-term memory impairment. A WAIS was performed 2 months before her admission to determine whether she had any residual cognitive deficits from her previous courses of ECT; the results were normal (table 1). She had had no trauma or illness in the 2 months before her manic episode.

The findings from Ms. A's physical examination were normal except for the presence of a snouting reflex and bilateral involuntary grasping. A screening EEG showed bilateral anterior frontal slowing; therefore, she underwent further neurological testing. A CT scan demonstrated isolated bilateral mesial frontal lobe atrophy. A lumbar puncture revealed a normal opening pressure and CSF analysis, including a normal γ-globulin level. Her levels of hematocrit, white blood cell count, serum electrolytes (including calcium, magnesium, and phosphorus), serum vitamin B_{12}, and urine heavy metals were normal.

TABLE 1
Neuropsychological Characteristics Before and After a Patient's Last Course of ECT

Test	Normal Value	Before ECT	Months After ECT 3	6	12	24
WAIS						
Verbal	100	110	80	94	92	94
Performance	100	102	72	86	86	86
Raven's Progressive Matrices, percentile	>50		62	83	86	88
Wisconsin Card Sorting, categories completed	≥ 4		2	4	5	5
Kinesthetic apraxia			Present	Absent	Absent	Absent

The frontal lobe atrophy was not viewed as a contraindication to ECT. Ms. A was started on a course of unilateral right frontotemporal ECT (modified with sodium pentothal and succinylcholine) three times a week. Immediately after her second treatment, she developed a bilateral fine resting pill-rolling tremor (4 tremors/second) and cogwheel rigidity. She had short-term memory impairment, but she was oriented and no longer manic.

After Ms. A's third ECT treatment, her cognitive and movement deficits worsened, and the ECT was discontinued. Her tremor became coarse and involved both upper extremities, but it was still ablated by intentional movement. In addition to cogwheel rigidity, Ms. A had motor and speech perseveration and kinesthetic apraxia (8). She was bradykinetic with a festinating gait. Ms. A was disoriented to time and place. Her short-term memory was markedly impaired. She could not recall a person who had been in the room only a few minutes before, and she remembered 0/3 words after 1 minute. She could recall past events well. Vigorous encouragement was necessary in order for her to eat or bathe. When left alone, she would lie in bed. She was unaware that she was incontinent of urine and feces. A repeat CT scan and lumbar puncture were unchanged; her EEG demonstrated background disorganization with anterior frontal slowing and sharp wave activity.

Ms. A's tremor did not respond to therapeutic trials of physostigmine, L-dopa, propranolol, diphenhydramine hydrochoride, phenytoin, or trihexyphenidyl. The tremor persisted when her attention was diverted from the affected regions, and it worsened during an amytal interview. The cogwheeling rigidity responded to L-dopa and trihexyphenidyl, but she was maintained without medication.

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During the next month Ms. A's memory, tremor, and rigidity improved sufficiently to allow her to leave the hospital. She was not motivated to carry out the activities of daily living, and she remained incontinent of urine and feces. By the third month after ECT her recent memory had improved (3/3 words at 3 minutes), and she was oriented. She still showed motor and speech perseveration and kinesthetic apraxia, and she had urinary frequency and incontinence. There was a mild bilateral pill-rolling tremor (4 tremors/second) and no rigidity. Neuropsychological testing (table 1) indicated that she was significantly impaired compared to her performance before ECT. A CT scan was unchanged from that made before the ECT. Her EEG no longer showed sharp wave activity, but the frontal slowing persisted and was more prominent than it had been in the pre-ECT study.

Six months after ECT she was no longer incontinent, did not display perseveration or kinesthetic apraxia, and required minimal encouragement to perform the activities of daily living. The tremor and an EEG were unchanged compared to 3 months after ECT. Her snouting and grasping reflexes were the same as her pre-ECT responses. Ms. A's performance on neuropsychological tests improved (table 1). During the subsequent 18 months she had little change in her cognitive (table 1) or motor function.

Discussion

This patient had impaired cognitive and motor function that developed during a course of ECT. Although it is likely that the patient had diffuse cerebral dysfunction, the pattern of her motor and cognitive deficits suggested that she had a specific injury to the frontal lobes and nigrostriatal pathways. Lack of motivation, kinesthetic apraxia, perseveration, incontinence, and poor performance on the Wisconsin Card Sorting Test are frequent findings in patients with frontal lobe damage (8). Her resting tremor and cogwheel rigidity suggested parkinsonism, which is usually attributed to nigrostriatal impairment (9).

The progressive development of her deficits during ECT suggested a possible causal relationship. However, the manner in which ECT was associated with her deficits was not clear. ECT has been associated with superficial cerebral cortical hemorrhages (2), but there was no evidence for this on either Ms. A's CT scan or lumbar puncture. She had CT scan and EEG evidence of frontal lobe damage before her last course of ECT. Although brain damage is generally not considered a contraindication to ECT, it is possible that the ECT aggravated a preexisting injury. There is a precedent for a subclinical deficit becoming manifest after ECT. Dysken and Halaris (5) reported that a patient with hyperparathyroidism developed asterixis only after ECT. Experimentally, ECT reduces the activity in central catecholaminergic pathways (10), so it is possible that ECT could produce or aggravate parkinsonism. However, this does not explain why the signs of parkinsonism partially resolve in some patients after ECT (6).

The occurrence of a persistent neurological deficit following ECT is rare. We have found no reports associating ECT with a movement disorder and cognitive impairment. This case suggests that if a neurological deficit develops during ECT, the patient should be thoroughly evaluated for treatable causes of underlying cerebral injury, and alternate modes of treatment should be considered.

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Tardive Dyskinesia Following Low Doses of Piperacetazine

BY ROBERTO A. DOMINGUEZ, M.D.

Early in treatment antipsychotic drugs cause acute extrapyramidal syndromes that decrease with a reduction in dosage. Tardive dyskinesia, unlike early extrapyramidal syndromes, usually occurs only after at least 1 year of treatment and the dyskinesia is potentially irreversible. Although patients with dyskinesia most often display buccolinguomasticatory movements, involuntary movements of the extremities and trunk can also occur.

Tardive dyskinesia has been associated with all classes of neuroleptics. Piperazine phenothiazines, probably because they are stronger dopaminergic blockers, may more frequently produce the dyskinetic syndrome. However, piperidine phenothiazines are less often implicated in tardive dyskinesia. Some clinicians (1) recommend using the piperidine class of drugs if continued neuroleptic treatment is necessary after the onset of dyskinetic symptoms.

Piperacetazine is an infrequently used antipsychotic of the piperidine group. In a recent Veterans Administration survey (2) it was prescribed to less than 0.1% of schizophrenic patients. A review of the literature found few reports of tardive dyskinesia with thioridazine and none with piperacetazine. The following report, therefore, appears to be an exceptional case.

Case Report

Mr. A, a 65-year-old man, had a history of chronic schizophrenia, with several hospitalizations before 1971. During this period he had received haloperidol, thioridazine, and thiothixene in that order, all in relatively low dosage. A review of his hospitalizations showed no signs of tardive dyskinesia. In late 1971 Mr. A was started on 25 mg of piperacetazine h.s. He was followed as an outpatient every 3 months and received no other psychotropic drugs. His clinical course from 1971 to 1974 was uneventful. For undocumented reasons his dosage was increased to 50 mg of piperacetazine h.s. in 1974, and he was maintained at this dosage until mid-1976. During a scheduled outpatient visit in June 1976, Mr. A complained of "funny movements in my mouth." Further examination revealed involuntary orofacial and lingual movements and dyskinesia of the fingers, primarily on the left side. At that time tardive dyskinesia was diagnosed, and piperacetazine was discontinued. Within 1 month he had begun to regress. Mr. A's principal complaints were restlessness, anxiety, and insomnia, and 50 mg of piperacetazine h.s. was restarted. Four months later severe dysarthria, chorea of the hands, and increased abnormal orofacial and lingual movements were present. Piperacetazine was discontinued, and Mr. A began taking hydroxyzine. No other psychotropic drugs were prescribed. Within 6 months his involuntary movements had decreased. He was examined by me 3 years later, and he had no signs of tardive dyskinesia except for increased blinking.

Discussion

There are more prominent issues to be reviewed than simply the association of tardive dyskinesia and piperacetazine. It is important to recognize that the syndrome developed in a patient taking low doses of a piperidine phenothiazine. The rapid progression of symptoms while the patient received that drug and their gradual disappearance once the drug was discontinued are also aspects that merit further discussion.

The recommended dose of piperacetazine on the package insert is 10 to 160 mg/day. Most reports regarding the clinical use of this drug confirm this recommended therapeutic range (3, 4). However, Gallant and Bishop (5) found improvement to be superior in doses of 400 mg/day. Based on the recommended dose, the daily amount of medication that this patient received (25–50 mg/day) was relatively low. Nevertheless, tardive dyskinesia appeared. Thus, clinicians should know that tardive dyskinesia can evidently develop with low dosages of piperidine phenothiazine.

The rapid progression of dyskinetic symptoms led to discontinuing the drug, even at the risk of the patient's clinical regression. As noted in this report, the orofacial movements had become so severe that dysarthria occurred. Quitkin and associates (6) reported the need to diagnose the syndrome early because it may be reversible if detected in time. In this case prompt discontinuation of the drug led to almost total symptomatic improvement of the patient's involuntary movements.

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Gilles de la Tourette's Disorder Associated with Pemoline

BY ELIZABETH MITCHELL, M.D., AND KENNETH LEE MATTHEWS, M.D.

According to *DSM-III,* Tourette's disorder is characterized by "recurrent, involuntary, repetitive rapid movements (tics) including multiple vocal tics. . . . The tics typically involve the head and, frequently, other parts of the body such as the torso and upper and lower limbs. The vocal tics include . . . clicks, yelps, barks, sniffs, and coughs, or words." Age of onset ranges from 2 to 15 years. The tics are exacerbated by stress and can be voluntarily suppressed.

Recent reports in the literature have begun to support an organic etiology for this syndrome. A neurophysiologic impairment of the CNS has been postulated, with varying support from data showing anatomic pathology implied by abnormalities found on psychological testing, EEGs, and neurological examinations (1). The neurotransmitters dopamine and norepinephrine have been suggested as mediators for this disorder. This hypothesis is supported by the observation that haloperidol, which blocks postsynaptic dopamine and norepinephrine receptors, will relieve symptoms in most affected individuals. There have also been case reports of precipitation or worsening of symptoms by methylphenidate therapy. Methylphenidate is a pharmacological antagonist of haloperidol that increases the availability of neurotransmitter at postsynaptic catecholaminergic receptor sites (2–4). The following case report introduces another pharmacologic agent that may be implicated in movement disorders.

Case Report

David, a 10-year-old boy whom we studied at a child guidance clinic, had had learning difficulties and behavior problems at school. He was rude to adults, did not finish his work, could not sit still, and interacted poorly with his peers. In groups David was disruptive, distractable, and impulsive.

David was an only child. His mother was of Jewish ancestry, had insulin-dependent, juvenile-onset diabetes and severe peripheral vascular disease. His father, who had been raised in an orphanage, had a mild to moderately severe speech disorder that involved increased rate of speech, decreased intelligibility, and loud volume.

David's mother was 38 years old when she gave birth to David. She had miscarried once before. In addition to insulin-dependent diabetes, she had elevated blood pressure during the pregnancy and had an IUD in place. At 35 weeks, when an abruptia placenta was diagnosed, the attending physician artificially ruptured membranes to induce labor. David weighed 2.5 kg at birth. He was placed in 30% oxygen and received dextrose intravenously. After 2 days the hospital staff noted that David had physiologic jaundice, which was treated with phototherapy. He also had episodes of diarrhea on his 10th and 14th hospital days, but no pathogen was isolated.

David's developmental milestones were delayed. He walked without support at 18 months and his language development was inconsistent. When David was 4½ years old, an evaluation showed a mild articulation problem and telegraphic, unintelligible speech with neologisms but very clear intelligible sentences when David was encouraged by the examiner. It was noted at testing that the unintelligible speech occurred when his father was present and speaking to the boy in a loud and punitive manner.

When David was 6 years old, he was treated with methylphenidate for extreme hyperactivity. This was discontinued after one year because the side effect of vomiting occurred. He was then treated with up to 30 mg/day of thioridazine. The boy had no history of movement disorder before the age of 6 or on either medication. When David was 10 years old, thioridazine was stopped in June for a summer drug holiday, and this therapy was not resumed. In November David was noted to have difficulty sitting still. He frequently talked out in class and did not finish his work. He fidgeted and ground his teeth when watching television. Neurological examination revealed poor fine motor movements, synkinesis, and poor balance. We began pemoline therapy, with a daily morning dose of 37.5 mg.

David's initial response to this medication included marked reduction in impulsivity, less intrusiveness, and a lowered activity level. However, in January he developed a tic-like motor movement involving the head and shoulder. This tic later included eye rolling, blinking, grunting, and a distinct jerking of the head and shoulders. We noted that the tic worsened when David was emotionally stressed, and that David could suppress the tic with extreme effort. When we

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discontinued the pemoline in February, all tic movement ceased within 2 weeks. When we then restarted pemoline therapy, the movements reappeared, somewhat worsened in intensity. We again discontinued the medication, which caused a marked reduction of movements.

Discussion

Pemoline, a relatively new medication used for the treatment of hyperactivity, was first studied clinically in 1956. Pemoline is structurally "dissimilar from the amphetamines and methylphenidate but has similar pharmacologic effects without significant sympathomimetic activity" (5). Reported side effects include insomnia, anorexia, stomachache, mild depression, nausea, dizziness, headache, and drowsiness. The 1979 *Physicians' Desk Reference* states, "There have been reports of dyskinetic movements of the lips, face, and extremities occurring with the use of Cylert [pemoline]. . . . A definite causal relationship between Cylert and these reactions has not been established." Page and associates' double-blind study (5) of the efficacy of pemoline in managing hyperkinetic behavior caused by minimal brain dysfunction showed improvement in gross behaviors of all subjects as well as cognitive and perceptual functioning. The authors concluded that pemoline was a "highly useful clinical alternative to the amphetamines and methylphenidate as an adjunct in the management of hyperactive children" (5).

It is unclear why David developed tic-like movements during pemoline therapy. It may be that the administration of pemoline precipitated the development of Tourette's disorder or that David's tic-like movements were a side effect of pemoline. What part, if any, the previous treatment with thioridazine and its minimal extrapyramidal stimulation played in the development of this movement disorder also remains unclear. Dopamine has been implicated in the mechanism of tardive dyskinesia production. Thioridazine and other major tranquilizers have been postulated as sensitizing striatal neurons to dopamine. If pemoline and methylphenidate both act through dopaminergic mechanisms, a model is suggested in which neurons sensitized by thioridazine, are further stimulated by dopamine through the mediation of pemoline. Genetic factors may also play a part in increasing vulnerability to movement disorders. Pollock and associates (2) have reported an increased incidence of Tourette's disorder among children of Jewish parentage. The tic described in our case report more closely resembles Tourette's disorder than it does tardive dyskinesia, but an association among Tourette's disorder, tardive dyskinesia, and Huntington's chorea, has been suggested previously (4). Further study is indicated to elucidate the relationship between dopamine and movement disorders.

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Common Mistakes Made in Group Psychotherapy by Beginning Therapists

BY HENRY L. SPITZ, M.D., FREDERIC KASS, M.D., AND EDWARD CHARLES, M.A.

The treatment of psychiatric patients in group psychotherapy is a practice that has expanded dramatically in the past two decades. Along with this trend the training of group therapists has taken on added significance. Despite the increased use of groups as a form of treatment and the increased interest in the effective teaching of group methods, there is a virtual absence of systematic study of many elements of group therapy in several essential areas. One noteworthy example is our lack of knowledge about the common mistakes made by beginning group psychotherapists and the negative outcomes these mistakes can cause.

A review of the group therapy literature reveals that therapist error has been discussed only in the context of broader issues, such as group formation and management, or as part of general discussions of group therapy education. Grotjahn (1) anecdotally discussed

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TABLE 1
Rating by 42 Group Therapy Supervisors of Mistakes Made in Group Psychotherapy by Beginning Therapists

Questionnaire Item	Mean Rating of Each Item[a]
Most common mistake or problem area	
Handling problem patients who, for instance, reject help, monopolize the group, or complain	3.22
Managing resistances in the group	3.11
Discussing transference issues	3.06
Wanting to be liked by the group	3.03
Dealing with acting out among members	2.93
Properly orienting prospective members	2.86
Handling problems arising in the coleadership relationship	2.84
Defining the most useful focus for the group	2.79
Lacking awareness of intertransference feelings	2.74
Making premature interpretations	2.71
Handling the termination phase of the group	2.69
Least common mistake or problem area	
Violating confidentiality	1.36
Consciously disliking the group	1.47
Handling length of sessions	1.52
Handling hospitalizations for group members	1.61
Inappropriate openness about therapists' personal life	1.69
Dispensing medications to the group	1.74
Encouraging group members to meet in therapist's absence	1.77
Unawareness of therapists' bias against group therapy	1.81
Handling group size	1.84
Clarifying common misconceptions about group therapy	1.86
Avoiding fee setting	1.99

[a] 1=not at all; 4=very often; group means were 2.91 for the most common mistakes and 1.69 for the least common mistakes. Differences between the lowest rated and the highest rated items were statistically significant ($p<.001$, two-tailed t test).

mistakes in analytic group psychotherapy; he defined a mistake as anything that interferes with group formation and process. Williams (2) described "everyday problems" of group therapy trainees based on his personal experience as a supervisor of 50 trainees. Dies (3), in a recent survey of supervisors, referred to problems encountered by beginning group therapists. Utilizing a content analysis of responses, he found that most difficulties involved the transition from an individual therapy model to a group model.

In most modern residency training programs teaching and supervision of group treatment methods now occupies a prominent place. Mistakes made by beginning therapists are of central concern for teaching and supervision. The major goals of the present study were to identify and quantify the mistakes made most frequently by trainees and practitioners early in their group therapy experience and to use this information as a guide for determining priorities in the training and supervision of group psychotherapists.

Method

We developed a 43-item questionnaire based on the literature, our clinical experience, and suggestions made by the senior staff at the Vanderbilt outpatient clinic of the Columbia-Presbyterian Medical Center and the New York State Psychiatric Institute.

The items fell into three major categories where the therapist may err: 1) mistakes in technique, e.g., failure to follow up group absences; 2) countertransference problems, e.g., inappropriate openness about therapist's personal life; and 3) personal deficits in the therapist, e.g., difficulty achieving a requisite level of empathy with group members.

The questionnaire was distributed to 52 senior group supervisors (all practicing clinicians) from a variety of institutions in the United States and Canada. Only those actively involved in the training of group therapists were included. An effort was made to include supervisors with varied theoretical orientations in the group field (e.g., psychoanalytic, general systems, group as a whole).

Each item was rated from 1 (occurred not at all) to 4 (occurred very often). The supervisors were asked to base their response on their personal experience in supervising group therapists. An item analysis was performed on the data, and means and standard deviations were derived for each questionnaire item. A ranking of the individual items demarcated those rated highest (greater frequency of occurrence) and those rated lowest (least frequency of occurrence).

Results

Of the 52 supervisors 42 (81%) surveyed completed the questionnaire; of the 42 respondents 73% were physicians. Because an analysis of the response of these 31 physicians and the 11 social workers and psychologists revealed no significant difference, the data were combined for presentation. Table 1 presents the supervisors' ratings of the most common errors and the least common errors committed by beginning therapists. The demarcation between these groups was clear; a statistical comparison between them was highly significant ($p<.001$).

Discussion

The therapist's inability to handle problem patients was the highest rated item in the survey. References to the psychotherapeutic management of patients who were help-rejecting, monopolistic, or otherwise problematic were found with regularity in the group literature (4–6). The results of this study suggest that these patients frequently present a stumbling block for the novice group therapist. Problem patients, especially those who have become adept at interpersonal alienation as a defense against anxiety, pose challenges for the psychotherapist. In the group setting, the leader is charged with the simultaneous tasks of managing individual patients and managing the group as a whole. The successful resolution of this complex task is fundamental for effective group leadership. The trainee's struggle to negotiate a comfortable position between

individual patient management and group management is reflected in the high rating of this item by supervisors.

Many of the highly rated items reflect the trainee's difficulty in adapting to a group orientation rather than a dyadic model, a finding that is consistent with Dies' (3). This can be seen in items such as "defining the most useful focus for the group" and "problems arising in the coleadership relationship." Other highly rated items, such as wanting to be liked by the patient and difficulty with aggressive or angry feelings in patients, are indications of potential blind spots in the therapist's perception and management of transferential and countertransferential phenomena. These results coincide with the findings of Buckley and associates (7) in their survey of mistakes made by beginning residents during individual psychotherapy sessions, and they suggest that there are difficulties in a trainee's work with patients that are independent of the treatment modality.

Many of the highest rated items stand at the interface between mistakes in technique and countertransference problems. The supervisor's comments on the questionnaire support the notion that many of the "technical" errors made by their supervisees are, when explored more fully in supervision, a result of countertransference issues. Characterizing this process, one respondent remarked that his supervisee's ". . . unawareness of countertransference severely interfered with his ability to pursue specific problem areas." The countertransference problem is most often seen with an inability to work comfortably with anger. The therapist may avoid areas of his own anxiety, which invariably reflects itself as a problem in his leadership of the group. "Acting out" on the part of group members is a frequent consequence.

In contrast to the highest rated items, the lowest rated items appear to cluster around the so-called administrative or managerial tasks, e.g., dispensing psychotherapeutic medication or handling hospitalizations. We were encouraged to find that therapists' self-disclosure ("inappropriate openness about therapist's own personal life") and therapists' bias ("unawareness of therapist's bias against group therapy") were among the low rated items. Fee setting was also among the lowest rated items. The difference between this finding and that of Buckley and associates (7), who studied mistakes in individual psychotherapy, may be explained by the general practice of charging uniform fees to all group members. Under group circumstances fee setting may be less of an issue.

The information derived from this survey has helped determine a realistic set of priorities for group therapy supervision. It highlights the awareness of those involved in training other therapists to the most frequently occurring potential pitfalls that face new group leaders. For example, in our outpatient setting the entire 43-item questionnaire is currently being used as a guide or checklist for group therapy supervision. Supervisors monitor their work with trainees to ensure that important training issues are not neglected. The format can be readily integrated into a range of popular group therapy training models described in the literature.

We have found this format useful in identifying potential therapist problems that can be altered, avoided, or remedied by active supervision and case consultation. We will present an evaluation of the long-range effects of this type of group training and supervision on the quality of patient care in the future. It is hoped that over time the likelihood of negative outcomes related to therapist error can be significantly reduced.

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LETTERS TO THE EDITOR

This section contains reactions to Journal *articles, statements of opinion, brief research findings or ideas, short case descriptions, requests for information, etc. Letters should be submitted in duplicate to the Editor, who makes all decisions regarding publication. Letters must be typed double spaced throughout and should not contain more than 500 words and 5 pertinent references. Criticisms of published articles will automatically be sent to the author(s) for response. Letters will be edited for clarity and conformance with* Journal *style. We regret that we cannot inform writers of the disposition of letters or return those which are not printed.*

The Value of the Internship in Psychiatric Training

SIR: I am a psychiatric resident who completed an internship in internal medicine and would like to contribute to the debate over the value of an internship in psychiatric training. This issue has been discussed from various points of view (1–3).

My internship experience proved to be extremely valuable. I consider myself to be first a physician and then a psychiatrist. The internship year was crucial in firmly establishing this professional identity. The value lay not only in solidifying knowledge and confidence in medical judgment and skills, but also in the maturing experience of being a primary care physician with all its inherent responsibilities. The ability to recognize signs of physical illness that can manifest themselves as psychiatric symptomatology is integral to becoming a good physician and psychiatrist. For example, a patient diagnosed as having depression was referred to me. On initial examination, the patient was more physically ill than depressed. Further diagnostic studies revealed that the patient was suffering from a bleeding gastric ulcer. Another example is the importance of recognizing a mental status change that reflects a uremic state rather than an accompanying psychotic process. It is also important to be able to recognize the crisis from the nonemergency. One can then convey a sense of rationality and equanimity to others who may be overreacting to a misperceived crisis. Such abilities are cultivated during an internship experience.

In view of psychiatry's struggle to define its value to the social sciences as well as to the other medical disciplines, it would seem mandatory that a psychiatric trainee become competent and comfortable in dealing with physical as well as psychological illness.

Clinical rotations as an intern are qualitatively different from the bedside experience as a medical student. The essential difference is related to passive versus active learning. There can be no substitute for the latter. The integration of an internship year into the psychiatric residency helps to reduce the mind-body dichotomy and to fulfill the attainment of Engel's biopsychosocial model (4). I would also recommend the integration of psychiatry rotations into medicine and surgery residency programs, particularly because of the many psychiatric problems in everyday medical practice.

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Drug Monitoring and Improved Treatment Response

SIR: In "Monitoring Plasma Nortriptyline Concentrations" (April 1980 issue) Leo E. Hollister, M.D., and associates shed little light on the usefulness of drug monitoring to improve treatment response. The study only underscored the problems of compliance in an outpatient population.

There are several methodological and design problems with this report. The design did not address the authors' question: Do patients in the therapeutic range respond better than do patients outside this range? Instead, the design tested whether feedback from plasma monitoring could help clinicians to adjust dosage successfully. Such monitoring has been shown repeatedly to permit accurate titration for a wide variety of drugs such as lithium.

However, the ability to titrate has limited relevance to the question of whether such titration is desirable. When feedback does not work, researchers must explore the reasons why it did not work—something Dr. Hollister and associates did not do. Successful titration depends on three factors: accurate measurement, altering the dose based on the measurement, and patient compliance. In Dr. Hollister and associates' study patient compliance seems to have been the problem. The assay appeared to be adequate. However, despite repeated feedback—11 samples were assayed—one patient never had a level in the therapeutic range and had a barely detectable mean level for the entire period. Thus, the results suggest noncompliance. Improving compliance, in fact, is one reason for monitoring plasma levels.

Even if the study had been designed to answer the authors' question, problems remain. First, half of the patients had psychotic depression, which typically requires either ECT or combined therapy with neuroleptics and antidepressants. Second, the rating scales of different patients were performed by different raters. Dr. Hollister and associates did not report the number of raters or their interrater reliability. Comparison among patients is therefore not possible. Third, the authors stated that one criterion for admission to

the study was a Hamilton rating score of 20 or more, yet one patient was admitted with a score of 19.

For these reasons, the study failed to prove that monitoring of plasma tricyclic antidepressant concentrations is not justified. Rather, it underlined the problem of compliance in an outpatient population.

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SIR: In "Monitoring Nortriptyline Plasma Concentrations" Leo E. Hollister, M.D., and associates did not make it clear whether they used the information from plasma level determinations in the feedback group to manage those patients. Patients in the feedback group who had plasma levels outside the proposed therapeutic window for nortriptyline do not seem to have had their oral doses adjusted accordingly. One would not expect to see response or length-of-stay differences between this group and the blind group without assay values if the information had not been used. Indeed, (perhaps by chance) the blind group seems to have fallen better into the proposed therapeutic window.

As Dr. Hollister and associates pointed out, plasma assay determinations are not a substitute for management by clinical assessment of the symptomatology of an affective disorder. Rather, they should be used as an adjunct to identify idiosyncratic patients in regard to pharmacokinetics. It is likely that such idiosyncratic patients, if members of the "blind" group, would stay in the hospital and be considered "failures of response." Patients 19 and 18 might be examples of such patients.

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Dr. Hollister Replies

SIR: In a brief report one has to limit words and hope that readers will heed them. We clearly stated in our report that the physicians who treated the feedback group were informed immediately of the laboratory test results and encouraged to make appropriate dosage adjustments so as to maintain the patients in the presumed therapeutic range. That some patients failed to achieve such a range, two doing rather poorly, was undoubtedly due to failure of patient compliance. Such failure is common; in our experience about 25% of patients fail to comply at some stage of treatment or other. However, even when physicians know that patients are not complying, it seems there is little they can do about it. The possibility that such patients had abnormal pharmacokinetics which caused their low plasma levels is remote. Our questions were stated clearly and did not include, "Do patients in the therapeutic range respond better than do patients outside this range?" The fact that some patients have a diagnosis of psychotic depression is not an automatic indication for neuroleptics or ECT. The Hamilton scores of these patients were consistent with definite but moderate depression, confirmed by the fact that they were treated as outpatients. The one patient with a Hamilton score of 19 rather than 20 had a mean score (based on two raters) of 19.5. We probably should have rounded it off to 20, but we did not.

Determining the usefulness of plasma concentrations of drugs in clinical practice is best done in a naturalistic study, such as ours. Our study strongly suggested that routine monitoring of drugs is not mandatory for all patients, but that use of this procedure should be made on the basis of need. Monitoring of lithium levels is well established, but lithium has infinitely simpler kinetics than tricyclic antidepressants have.

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The Metaphysical Nature of Psychotherapy

SIR: While there is much in "Recent Trends in Psychotherapy" (April 1980 issue) by Judd Marmor, M.D., with which I can only concur, there are two points I wish to make in response to the paper. First, Dr. Marmor wrote that "dynamic psychotherapy and behavior therapy are both firmly rooted in scientific tradition. In contrast, the more recent 'third revolution' in psychotherapy represents, for the most part, a radical departure from that tradition." While many would agree with this view, I believe it is important to remember that the distinction between science and nonscience is a matter of convention—and that there is more than one convention extant.

I have argued elsewhere (1) that the application of Karl Popper's criteria makes it clear that psychoanalysis also belongs in the realm of the metaphysical rather than on the "science" side of Popper's line of demarcation. If one accepts Popper's line of demarcation, it follows logically (dictated by the test of the falsifiability of a theory) that all psychotherapy belongs in the realm of the metaphysical, and all psychotherapy rests importantly on the basis of a mutual agreement between therapist and patient as to how reality is to be construed. I agree with Dr. Marmor that many of the newer therapies smack of a forced consensus in this regard—the therapist doing the pushing and the patient seemingly supplying the willingness to be pushed. Second, doing psychotherapy based on any structured framework at all (and I would grant that it is impossible to do psychotherapy otherwise) carries with it a very real risk that human phenomena will be labelled and pigeonholed and then lost to any further possibility of examination. Perhaps this point is best illustrated by an example of a phenomenon not being lost because of the necessary care that was taken. Nemiah and Sifneos (2), in studying affect and fantasy in patients with psychosomatic disorders, found that their patients seemed to have "thoughts (which) appeared shallow, colourless, and lacking in the freedom, excursiveness and richness of intimate, personal associations that are characteristic of most of us." Rather than invoking standard psychological explanations (or labels) such as the mechanism of denial, Nemiah hypothesized as an alternative that "in these patients there is perhaps an absence, or at best only a rudimentary form, of the neuro-mechanisms that normally subserve the experience of feeling and of complex mental associations, and at the same time a lack or incompleteness of connections between these mechanisms to permit their mutual influence" (3). Such a hypothesis would, of course, not have been possible if the authors had not chosen to apply the standard explanation (based on psychoanalytic theory) in this situation.

Thus I feel there are advantages to viewing all psychotherapy as ultimately metaphysical in nature rather than considering the theoretical base of some psychotherapies as being scientific and that of others as being nonscientific. Used in this way, the word "science" all too often invokes an image of a body of knowledge that is entirely valid and that cannot be questioned.

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Material for a History of Lithium Therapy

SIR: I am gathering material for a detailed and fully documented history of lithium therapy. I have already written to many investigators whose work has been of major importance in establishing lithium treatment as a major therapeutic modality in modern psychiatry, but I would be most grateful for the courtesy of your columns to make a wider appeal for information, personal reminiscences, documents, photographs, correspondence, etc., which may have historical relevance. Naturally, all such material will be acknowledged, handled with the greatest care, and returned unmarked in due course. All confidences will be respected.

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Career Choice and Psychiatry

SIR: In "Choosing Psychiatry: The Importance of Psychiatric Education in Medical School" (April 1980 issue) Arthur C. Nielsen, M.D., concluded: "For the time being, psychiatric educators should continue to improve the quality of their educational programs, which we have shown here to be an important determinant of psychiatric career choice." Whether Dr. Nielsen has succeeded in showing the quality of psychiatric education to be an important determinant of psychiatric career choice is questionable. First, his attempt to deal with some of the possible "challenges" to his interpretation of the results is not entirely convincing. For instance, whether the NIMH peer review really measured educational quality is yet to be determined. Langsley and associates (1), commenting on the bases for rating the quality of the programs, cautioned that the final ratings were judgments, the validity of which would be difficult to establish (although there was a high degree of interrater reliability). Second, Dr. Nielsen did not tell us anything about the quality of undergraduate education in other subjects in the medical schools he compared, e.g., surgery or internal medicine, nor did he include the percentages of graduates entering these specialties. Comparative studies of the quality of teaching of several subjects, including psychiatry, and of the number of graduates entering these respective specialties would lend credence to Dr. Nielsen's conclusion.

In a comparative study in Toronto of the effects of teaching on medical students' ranking of career choice (2), we reported favorable changes for psychiatry after the teaching of psychiatry. However, the literature is still inconclusive. This was briefly discussed by Paula F. Eagle, M.D., and Luis R. Marcos, M.D., in "Factors in Medical Students' Choice of Psychiatry," which preceded Dr. Nielsen's article. Career choice is a complex subject influenced by a vast variety of factors, many of which were reviewed by Drs. Eagle and Marcos and some of which were mentioned by Dr. Nielsen in the introduction of his article. In our study we discussed the fact that most teachers would like to think that good teaching will help recruitment into their specialty; however, a causal relationship between good teaching and recruitment remains to be established. Meanwhile, far from being complacent, those of us involved in undergraduate psychiatric education should follow wholeheartedly Dr. Nielsen's suggestion to continue improving the quality of our educational programs. The declining recruitment into psychiatric residency programs has brought to the forefront the psychiatric education of the future physician. The widespread concern and publicity aroused by the declining recruitment should be recognized as an unusual opportunity for enlisting support for undergraduate psychiatric education—an area of psychiatric education of paramount importance that has been characterized by customary benign disinterest.

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Dr. Nielsen Replies

SIR: I welcome the opportunity to clarify certain issues raised by Dr. Voineskos. My study found modest correlations between the percentages of each medical school's graduates who chose psychiatric residencies and the quality of the psychiatric education received by students at the various schools. Quality was measured by priority scores assigned to medical student education grants during NIMH Psychiatry Education Branch peer review. Since publication, additional data have become available for the class of 1978 and the intercorrelations are now best estimated to be .21 (1975), .16 (1976), .36 (1977), and .33 (1978). Such consistent results, including this replication for 1978, argue for the statistical validity of the correlation (1).

What can we infer from this correlation? The hypothesis I favored in my paper is that superior programs induced higher

percentages of graduates to choose psychiatry. Dr. Voineskos appropriately wonders, as I did in my discussion, whether NIMH peer review is a valid assessment of "educational quality." By citing the study of Langsley and associates (2) as evidence favoring the "construct validity" (1) of peer review, I intended to go beyond subjectivity. Although Langsley and associates' actual emphasis was on cautious affirmation of peer review, they remained circumspect about their results by only noting the association between high priority scores and other "process" variables, generally considered to indicate quality. Obviously, no single scale will ever suffice to describe educational programs and their quality. My contention was only that priority scores were valid rough measures of educational quality.

What about the causal interpretation of the observed correlation—that quality influences career choice? Dr. Voineskos' experience with the favorable impact of psychiatric clerkships on student attitudes confirms that of others (3, 4) and supports causation.

Dr. Voineskos correctly wondered whether quality teaching by other medical school departments would also correlate with percentages of graduates choosing these fields. To my knowledge, no such data are available. However, it seems likely that some schools with excellent psychiatric teaching also excel in other fields, and that any competitive edge obtained by psychiatry from excellent teaching might be mitigated by excellence elsewhere. If this were true, the substantial correlations found for psychiatry would seem all the more remarkable.

The possibility that other influences, such as excellence in other departments, might limit psychiatric education's ability to attract recruits leads to my last point: I do not believe that improved education alone can solve the recruitment problem. Certainly the causes seem to lie elsewhere, especially in incorrect views about how much psychiatry can be practiced in the course of other medical specialties (including, but not limited to, family practice) and in the declining numbers of students with strong career interests in psychiatry who seek admission to medical school. Dr. Voineskos appears more sanguine than I am that education can solve the recruitment problem. Nonetheless, my research does indicate some potential in this area and certainly more studies of the microstructure of educational quality are indicated.

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Diagnosis According to Kraepelin

SIR: Back to Kraepelin! In "Schizophrenia to Manic-Depression: Mutation or Misdiagnosis?" (June 1980 issue) Ibe O. Ibe, M.D., and John L. Barton, M.D., referred to Kraepelin in reviewing patients who were initially diagnosed as having schizophrenia but during subsequent attacks were diagnosed as having manic-depression. The authors cited these as examples of mistaken diagnosis rather than mutation from one diagnosis to another. Kraepelin specifically warned against "attributing characteristic significance to a single morbid phenomenon," like catatonia, excitement, hallucinations, delusions of influence, etc. He did attribute some differentiating power to a history of rather complete recovery with insight and stated that "repeated attacks, especially those of contrasted colouring which have brought about no recognizable state of weakness will with rare exceptions justify the diagnosis of manic-depressive insanity" (1).

Still more pathognomonic is the premorbid personality. "An outspoken manic or cyclothymic temperament seldom leads to dementia praecox. . . . The question is more difficult to decide with persons of depressive or irritable temperament. It seems that we must distinguish several outwardly similar forms. Softness, sensitivity, dejection, lack of self-confidence we find more in the past history of manic-depressive insanity; shy, eccentric, withdrawn behavior in that of dementia praecox. Furthermore, the easily inflamed, enthusiastic passionate natures correspond to the former, and the unpredictable, stubborn, rough and violent natures to the latter" (2, my translation). Drs. Ibe and Barton may check these items in their case report.

Kraepelin also reported a study similar to that of Drs. Ibe and Barton. In this study it was found that of 370 patients with dementia praecox, 127 did not have a certain diagnosis. Of these patients, 67 (mainly women) were finally considered probable manic-depressives because they had no abnormalities or only those proper to manic-depressive illness. One wonders if as much diagnostic validity might be gained by a thorough reading of Kraepelin as by using either the RDC (3) or *DSM-III*.

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More on the Case of Anna O.

SIR: In "The Case of Anna O.: A Reformulation" (July 1980 issue) Marc H. Hollender, M.D.'s conclusions about Anna O. and Dr. Breuer were valid, based on the literature he quoted. However, in *Physiologie und Psychoanalyse in Leben und Werk Josef Breuers* (1) Albrecht Hirschmüller has provided us with a great deal more information about the patient and her physician. (This book has not yet been translated from the German.)

Hirschmüller found a copy of a report on Anna O. by Breuer that was sent to Dr. Binswanger's sanitarium in

Kreuzlingen, Switzerland. (Ellinger had established the patient's stay there.) These notes are much more extensive than the Breuer-Freud publication (2) and were taken by Breuer during and after the treatment. There are also letters written by Breuer to Anna O.'s mother, a cousin, and Dr. Binswanger, and notes by a Dr. Laupus, in whose charge she was at Kreuzlingen.

We learn that among other complaints Anna O. suffered from trigeminal neuralgia. For this and "convulsions and chorea minor," she had received increasing doses of morphine and chloral hydrate. Breuer tried to withdraw her during a stay at Sanitarium Inzersdorf near Vienna (the "country house" in the Breuer-Freud report), but she responded with delirium tremens.

Anna O. improved at Kreuzlingen, but was not cured. When she left in October 1882, she was still addicted. The duration of her addiction remains unknown. From 1882 to 1887 she readmitted herself four more times to Inzersdorf. The reasons are unknown, the diagnosis was hysteria.

The highlights of Hirschmüller's report are as follows:

1. Breuer did not abandon Anna O. suddenly. He transferred her to a sanitarium and kept in touch with her physicians and her family. He saw her at least once more in 1884.

2. Breuer's reluctance to publish the material does not appear to stem from sexual squeamishness. Like others before him, he was well aware of sexual disturbances in hysteria. Freud himself reported this, adding that he saw the importance of a sexual etiology for the neuroses much later. Breuer expressed his surprise about the lack of sexual content in Anna O.'s productions, but added "unless her passionate attachment to an overindulgent father served as a replacement" (1). Most likely Breuer was concerned about revealing the identity of his patient because she belonged to a prominent Viennese family. His daily visits to her house hardly went unnoticed.

3. The hysterical childbirth scene is never mentioned before 1932. Freud (3) wrote to Stefan Zweig that he "reconstructed" the abrupt termination of Breuer's treatment. Freud added that Breuer took flight on a second honeymoon with his wife and during that time his youngest daughter was conceived. Actually, Breuer did not leave Vienna at that time and his youngest child had already been born in 1882. (In 1914 Freud [4] only quoted Breuer as having mentioned an "untoward incident.")

4. Anna O. was not Breuer's first or last hysterical patient. He treated others until 1912 and, like Freud, admitted such patients to Kreuzlingen. However, Anna O. may have been the only one Breuer treated with the cathartic method.

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Dr. Hollender Replies

SIR: Several of the points made in Dr. Pappenheim's letter were new to me, although I had already gleaned some information from Ellenberger's fascinating article (1). While the material helps to fill in a few gaps, I don't think it alters the conclusions I reached—that Anna O. was a bright woman born at a time and in a place where there were few outlets for her talents, that her treatment by Breuer temporarily relieved the tedium and boredom of her confined life, and that her final solution was reached when she became an effective and successful social worker and feminist.

I do not believe it was Freud who stated that Breuer took flight on a second honeymoon. That statement seems to have originated with Jones (2).

I greatly appreciate Dr. Pappenheim's comments.

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MARC H. HOLLENDER, M.D.
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Psychotherapy: A Laboratory of Preventive Knowledge

SIR: Perhaps an organic synthesis of psychotherapy as a whole rather than encyclopedic eclecticism should be applied to the question of whether psychotherapy should receive government support.

In order to solve life's problems the individual must constantly integrate here-and-now, ephemeral interactions with his environment on the one hand and his lifelong process of personality formation or self-definition on the other. A workable "common sense" capacity for problem solving derives from a favorable character formation and a knowledge about one's changing social environment. Concern with mental health is meaningless without the study of personality formation. If grant support were restricted only to categories of "problem" definition (addiction, rape, teenage pregnancy, child abuse, delinquency, etc.), the overall knowledge of personality resources would decline to a dangerous point, which our society can hardly afford.

In addition to providing trouble-shooting expertise for symptoms of disturbed behavior, psychotherapy has been an empirical testing ground for the study of personality formation. There are many paths toward this complex goal. Child therapists deal with the manifestations of individual behavior while character is being formed. Adult therapists have been interested in the patient's ingrained identifications and patterns of behavior. Family therapists work in the laboratory of ongoing reactions that occur between the formative parental environment and the offspring's emerging personality.

The results of this complex and overwhelming undertaking are emerging even slower than did Pasteur's first discoveries about microorganisms. If bacteriologists had been constantly held accountable for accomplishing therapeutic effectiveness, the discovery of penicillin and other drugs might have been handicapped or prevented.

Yet the test of psychotherapeutic results is only partly in

the removal of visible malfunction. Prevention of malfunction in the offspring is causally, if complexly, connected with helping the parent generation, as it has become explicit in the evaluation of the family therapy approach. However, all current forms of psychotherapy are potentially preventive interventions for the next generation. Consequently, the burden of proof of cause and effect is overwhelming at this time.

Is it fair to threaten withdrawal of funds from psychotherapy, the applied research laboratory of preventive knowledge about effective leverages of future mental health? Is anyone in a position to determine whether such a vast effort works? We know that rapid technological progress has led to disintegrative results in human relationships. We know that explosive societal changes have escalated self-destructive behavior and erosion of parenting responsibility. Will Congress, and society as a whole, have the wisdom to support psychotherapy as the living laboratory of preventive knowledge?

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Questioning the Board Certification Process

SIR: In "Board Certification Anxiety" (July 1980 issue) Franklin G. Maleson, M.D., and associates provided some rich, useful anecdotes pertinent to any candidate for Board certification in psychiatry. However, I must take issue with their assertion that "it is clear that the examiner team functions with a satisfactory operational definition of competence." Examiners do not function as two separate observers; rather, they sit in the same room with easy exposure to conscious and unconscious cues from each other. Sometimes they may be familiar colleagues. This circumstance makes it far from clear that there is an operational definition of competence in the Board certification process.

Psychiatrists have responded to the need for operationalism in psychiatric diagnosis by stressing phenomenological criteria in *DSM-III*. This addressed itself to the problem of psychiatrists defining schizophrenia and other mental illness diagnoses on the basis of their personalized notions. There has been an increased awareness that it is not adequate for psychiatrists to simply agree on who should be labelled schizophrenic, but that they must define their criteria. Those involved with the Board certification process should borrow from these leads. With better defined criteria, they might strive for a more operational definition.

Finally, I question whether this article solely addressed candidate anxiety. It seems Dr. Maleson and associates attempted to reassure all psychiatrists, including Board examiners, that the system is essentially fair. Is this an attempt to assuage the anxiety of examiners, some of whom might not be comfortable in acknowledging that the Board certification process is not as fair as it should or could be?

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Dr. Maleson and Associates Reply

SIR: During the orientation for examiners each examiner is given a description of performance areas to be evaluated. These include patient-physician interaction; conduct of the clinical examination; capacity to elicit clinical data; formulation, differential diagnosis, and prognosis; therapeutic management and knowledge of therapies. Each of these areas is further defined in detailed and explicit ways. These performance criteria are discussed by each examining team.

Examiner assessments about candidate performance tend to be surprisingly similar, even at the beginning of their evaluation discussions. This suggests that the examiners, although maintaining different areas of subspecialty interest, are observing similar candidate qualities and are using similar criteria for competence. This was what we meant in a general, if not "pure science," sense by "a satisfactory operational definition of competence." Although examiners are not usually close colleagues, examiners do, of course, influence one another. This occurs somewhat indirectly during the 30-minute question-and-answer period and quite directly during the evaluation discussion. This mutual influence would seem to provide a safeguarding function by moderating more extreme, perhaps idiosyncratic, examiner positions in regard to a candidate's performance, a safeguard of fairness that completely independent assessments would sacrifice. Even *DSM-III*, a model striving for greater objectivity as Dr. Garrick noted, still contains the potential for independent observers to see quite different things after observing the same phenomena. The objectivity in grading so rightly valued by Dr. Garrick is approached in the written examination. The existence of an additional oral examination reflects a sentiment that a written examination alone is not sufficient to measure clinical competence in psychiatry.

FRANKLIN G. MALESON, M.D.
PAUL J. FINK, M.D.
HOWARD L. FIELD, M.D.
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Psychiatric Patients and Sudden Death

SIR: It was gratifying to me to discover that Robert E. Smith, M.D., and associates, in their article "Haloperidol and an Unrelated Sudden Death" (July 1980 issue), had adopted a viewpoint that is essentially in accord with the one I have emphasized in my book *Sudden Death and Psychiatric Illness* (1). Dr. Smith and associates averred, "Because coronary heart disease is the leading cause of sudden death, it must be carefully ruled out before one reports a death as having no demonstrable natural causes and before one questions whether previously administered medications contributed to the death in an unspecified manner."

Quite properly, then, they added, "Because asymptomatic coronary heart disease is the leading cause of sudden natural death in young adults, this diagnosis must be carefully ruled out in any sudden unexpected death involving psychiatric patients receiving psychotropic medication." However, their description of the events preceding their patient's cardiac arrest suggested that "restraint stress," a syndrome I have discussed at some length in my book, could have been responsible for this sudden, unexpected death. In discussing the pathogenesis of the fatality, Dr. Smith and associates had not overlooked the possibility that "psychiatric patients with asymptomatic coronary heart disease may be at increased risk for sudden death because of agitation and heightened arousal secondary to their psychotic state:" However, in my

opinion it is equally important for psychiatrists to recognize that imposed physical restraints on agitated psychiatric patients may be a meaningful risk factor in sudden, unexplained deaths of even relatively young adults who exhibit manic behavior, regardless of the psychotropic drugs such decedents may have received prior to their deaths.

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MARTIN H. WENDKOS, M.D.
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Opiate Use and Sexual Function in Women

SIR: Steven M. Mirin, M.D., and associates are to be congratulated for their thorough and thoughtful investigation reported in "Opiate Use and Sexual Function" (August 1980 issue) and for the support it provides for the drive reduction theory of heroin addiction.

Mendelson and Mello's work (1) on testosterone and luteinizing hormone levels in alcohol users and the known dysphoric effects of chronic alcohol intake (2) may provide an interesting parallel for alcohol addiction. However, almost all of these types of studies have been done only on men. It would be interesting to see if a similar relationship could be established between opiate use and sexual function in women. Certainly the effects of alcohol on pituitary-gonadal function in women have been shown to be completely dissimilar to the effects that occur in men (3).

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Oculogyric Crises and Tardive Dyskinesia

SIR: In "Oculogyric Dystonia in Tardive Dyskinesia" (July 1980 issue) Henry A. Nasrallah, M.D., and associates raised the possibility that dystonia may be an acute manifestation of dopamine hyperactivity and therefore its presence could predict a future predisposition to tardive dyskinesia. Yet their patient's tardive dyskinesia was long standing and his dystonic symptoms occurred relatively late in the course of treatment. We have treated a patient who had typical but recurrent oculogyric crises and subsequently developed tardive dyskinesia.

Mr. A, a 54-year-old man with a 31-year history of schizophrenia, had been in outpatient treatment in our clinic for more than 2 years. While maintained on fluphenazine, 15 mg/day, and benztropine, 2 mg/day, Mr. A began to experience recurrent dystonic reactions characterized by a fixed upward gaze and a hyperextended neck. These apparent oculogyric crises were relieved by intramuscular benztropine and occurred much less frequently when we decreased the fluphenazine dosage to 10 mg/day. Six months later, Mr. A continued to report about two episodes of oculogyric crisis per month. At that time we also noted rolling of the tongue and lip smacking for the first time. We changed Mr. A's medication to 300 mg of thioridazine and his oculogyric crises completely stopped. However, an AIMS exam the following month revealed moderate abnormal movements of the lips, jaw, and tongue.

This case of oculogyric crisis coexisting with tardive dyskinesia differs from Dr. Nasrallah and associates' case report because of the differing temporal relationships. Our patient's oculogyric crises were long standing and his tardive dyskinesia was not detected until later.

This cannot be taken as evidence for the hypothesized predisposition to tardive dyskinesia in patients with dystonic reactions. While parkinsonian symptoms can clearly coexist with tardive dyskinesia, their significance as possible predisposing or etiologic factors is still very much in question (1, 2, 3). The possible connections between the hyperkinetic symptoms of parkinsonism, including oculogyric crisis, akathisia, and tremor, and the hyperkinetic syndrome of tardive dyskinesia need to be thoroughly investigated (4).

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The Omnipresence of Culture

SIR: I applaud Edward F. Foulks, M.D., Ph.D., for emphasizing culture as an integral part of psychiatric education in "The Concept of Culture in Psychiatric Residency Education" (July 1980 issue). As Dr. Foulks illustrated with his clinical examples of certain ethnic minorities, such training may help to improve the care of underserved groups of patients. Although cultural issues in psychiatric education have increased, too often they are an exotic appendage to the "real" training in psychodynamics or psychobiology. Yet culture seems best understood as pervading all aspects of the patient's life, from the unconscious to the biological response to medication (1).

However, in subtle ways Dr. Foulks also seemed to present culture as an exotic appendage to mainstream psychiatry. Although he stated that "clinical judgments . . . are never free from the influence of the therapist's cultural values," he also stated that when the culture of patient and therapist are matched "therapy is often successful," while a mix of cultures usually produces problems. This ignores therapeu-

tic difficulties, such as overidentification, when the patient and therapist are of similar cultural background (2, 3). The relationship of such matches to interminable psychotherapy is one area that needs further research. The effects of culture may seem most apparent to an observer from a different frame of reference; when therapist and patient have syntonic backgrounds, cultural effects may go unnoticed.

Foulks' emphasis may belie the importance of culture in the treatment of all patients, including VIPs, women, homosexuals, and many ignored ethnic groups. (When was the treatment of Cajun patients last discussed?) Without such a broad perspective on culture, psychiatric education may be unconsciously devaluing certain sociodemographic groups and limiting the relevance of cultural psychiatry.

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H. STEVEN MOFFIC, M.D.
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Dr. Foulks Replies

SIR: I am grateful to Dr. Moffic for pointing out the possibility of problems arising in psychotherapy when patient and therapist have common cultural values. There are indeed obvious pitfalls in the psychotherapeutic process when the degree of shared perspective results in a mutual avoidance of areas of unconscious conflict. Antitherapeutic alliances with unrecognized countertransference problems, such as overidentification with the patient, are encountered frequently enough by clinical supervisors to warrant explicit attention in most residency and psychoanalytic training programs. On the other hand, Glass and associates (1) recently analyzed the results of 475 controlled studies involving the efficacy of psychotherapy. Using the statistical method of meta-analysis, they were able to compare 78 different forms of psychotherapy. A major conclusion of this research was that the studies with the best results were those in which the ethnic group, age, social, and educational status of patients and therapists were similar.

The relevance of cultural factors in the psychotherapeutic process raises additional concerns, not only in the treatment of ethnic minorities but of social minorities as well. Perceptions of patients' thoughts and behavior as bizarre or exotic might be replaced by therapeutic understanding when seen in social and cultural contexts. This is a major benefit derived from using the concept of culture in treating all patients, medical or psychiatric, Cajun or Cuban, homosexual or heterosexual, male or female.

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An Omission

SIR: In "Hemodialysis, Endorphins, and Schizophrenia" (April 1980 issue) Milton T.W. Hearn, Ph.D., and I inadvertently omitted an important work (1).

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1. Ross M, Berger PA, Goldstein A: Plasma β-endorphin immunoreactivity in schizophrenia. Science 205:1163–1164, 1979

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Correction

In Philip E. Veenhuis, M.D.'s book review of *The Broken Connection* by Robert Jay Lifton in the August 1980 issue, the second sentence of the first paragraph on page 1000 should read, "Lifton extensively quotes a 1963 article by Elliot Luby; he sees Luby's work as 'very compatible with our paradigm.' " The staff regrets this error.

BOOK FORUM

NANCY C. ANDREASEN, M.D., EDITOR

DSM-III: A British Perspective

Diagnostic and Statistical Manual of Mental Disorders, 3rd ed., *by the American Psychiatric Association. Washington, D.C., APA. 1980, 481 pp., $25.00; $20.00 (paper).*

Attitudes toward the classification of mental disorders have changed radically in the last two decades, and there can be no more vivid illustration of this renaissance than the difference between the second (1968) and third (1980) editions of this manual. *DSM-III* is five times the size of its predecessor, the product of nearly six years' labor by an energetic task force with 14 subcommittees. A vast amount of work, discussion, argument, and persuasion—in what proportions one can only guess—has gone into its preparation. The reliability studies and field trials alone involved over 500 clinicians, drawn from all parts of the United States, and nearly 13,000 patients.

The importance of this manual does not lie, like the Great Pyramid of Giza, in its size or the man hours it represents. It lies in a series of major changes in format and philosophy, and it is these which have made it so controversial. For the first time in any national or international classification every diagnostic category is given an operational definition to make it as clear as possible which patients do and do not qualify for inclusion under that rubric. The classification is "multiaxial," with independent recording systems for five different kinds of information, making it much more complicated than its predecessor. The grouping of clinical syndromes has also been extensively changed. All affective disorders are brought together, for example, and the old groupings of psychotic and neurotic illness disregarded. Finally, and most provocatively of all, many of the hallowed terms of psychiatry, like hysteria, manic-depressive illness, and even psychosis and neurosis, have been discarded and replaced by unfamiliar and unattractive but also unambiguous and unsullied terms like somatoform disorder, substance use disorder, and paraphilia.

These radical changes have involved breaking ranks with the World Health Organization because they commit American psychiatry for the next decade to using a quite different classification from the ninth revision of the *International Classification of Diseases* (*ICD-9*), which most other countries will be using. This is obviously a matter for regret and will at least temporarily hinder international communication, but it is difficult to imagine WHO ever being able to introduce radical changes into the *ICD* on its own initiative. As Stengel pointed out 20 years ago, any international classification is bound to be "conservative and theoretically unenterprising." Like a convoy its speed is dictated by its slowest members. If major changes were ever to come, an influential association had to show the way first, and the architects of *DSM-III* have done all they could to minimize the damage by providing a rough translation into *ICD-9* categories that will serve for basic statistical purposes, and by reprinting the whole of the mental disorders section of *ICD-9*, including its built-in glossary, as an appendix to the manual.

DSM-III has been and will continue to be criticized on many different scores, both reasonable and unreasonable. To my eye there are several minor anomalies or misjudgments and at least one major opportunity missed. The use of five different axes represents a considerable increase in complexity, which is justified only if substantial benefits are gained. The fourth axis (severity of psychosocial stressors) and the fifth (highest level of adaptive functioning past year) are relatively uncontroversial; they are optional anyway, and pilot work has shown them to have reasonable interrater reliability. The problems lie with the first three. Axes I and II could have been used to distinguish between lifelong but stable handicaps (personality disorders, mental retardation, etc.) and disorders that are either progressive or potentially reversible (clinical syndromes). Clinical syndromes are indeed coded under axis I and personality disorders and specific developmental disorders under axis II. However, mental retardation is coded under axis I, without any explanation, and to increase the confusion neither of the two axes has either a title or any discernible unifying theme. Since multiple diagnoses are possible on both, it is difficult to see any advantage in keeping them separate. The system could be reduced to four axes without any loss. The third axis (physical disorders and conditions) could have been used for all etiological factors, both proven and suspected, thus allowing the first two to be purged of all etiological implications and assumptions in the way that Essen Möller has suggested. Dr. Spitzer and his colleagues, however, have done this only halfheartedly. Despite their avowed commitment to an atheoretical approach to etiology, several of the terms they have chosen for their clinical syndromes embody quite explicit etiological assumptions—adjustment disorder, brief reactive psychosis, and conversion disorder, for example.

I also doubt the wisdom of the decision to define schizophrenia very tightly and restrict the diagnosis to patients under age 45 who have been continuously ill for at least six months. Many patients who would formerly have been regarded as having schizophrenia will now be displaced into other categories, mainly schizophreniform or schizoaffective disorder and atypical psychosis. Since no diagnostic criteria are provided for either schizomanic or schizodepressive disorders, this important group of illnesses is thus left to serve, with atypical psychosis, as the ragbag of the functional psychoses. Indeed, if many patients fail to fulfill the criteria for a diagnosis of either schizophrenia or affective disorder, a high proportion of all patients with psychoses will end up in this unsatisfactory limbo. Although this failure to define schizoaffective disorder may perhaps be based on a realistic appraisal of current uncertainties, it is, nonetheless, a serious shortcoming, and one that has been exacerbated by this restrictive approach to the diagnosis of schizophrenia.

Finally, there were places where I could not follow the

authors' logic. I do not understand, for example, why a man who becomes depressed after losing his job has an adjustment disorder, but one who develops the same symptoms after losing his brother is not ill at all, though both are apparently equally worthy of treatment. Nor do I understand why pedophilia should be regarded as a disorder if homosexuality is not. But these and other criticisms must be seen in perspective. Anything so complex and so laden with overtones as a classification of mental disorders is bound to be criticized in one way or another by almost all its users. Indeed, I imagine that most of the members of Dr. Spitzer's task force would like to see changes themselves, perhaps quite radical ones. Real classifications are always imperfect, and the authors have taken pains to emphasize that they anticipate the need for extensive and more or less continuous revision in the future. In their own words, "This final version of *DSM-III* is only one still frame in the ongoing process."

By any standards *DSM-III* is an impressive document, amply justifying the time and effort involved. Its principal architects have a sound grasp of the fundamentals of psychiatric nosology, and every section of the classification is obviously the product of a thoughtful and well-informed consideration of the issues involved, sustained through several drafts. The levels of interrater reliability obtained in the field trials were far higher for most of the major syndromes than anyone has achieved before outside the restricted confines of academic research. The manual is well laid out and accompanied, for those who find its bulk intimidating, by a pocket-sized booklet, the *Mini-D*, describing the framework of the classification and the operational criteria and main differential diagnosis to be considered for each disorder.

Everything now depends on the response of the APA membership as a whole over the next few years. If most clinicians ignore or reject this classification and the operational definitions that are its most important component, or even if they merely fail to master its complexities, the whole enterprise will be seen as a failure, a psychiatric Concorde. If, on the other hand, the majority of clinicians recognize the fundamental importance of these innovations and make the effort needed to adapt to them, psychiatry will have taken a crucial step on the road to becoming a more rational and scientific discipline, and WHO and the psychiatric community as a whole will have good reason to be grateful to their American colleagues. Whatever the final outcome, Dr. Spitzer and his colleagues, and the American Psychiatric Association itself, deserve a hearty vote of thanks for their imagination, their energy, and, above all, their determination in bringing this great enterprise to fruition.

R.E. KENDELL, M.D.
Edinburgh, Scotland

How to Write and Publish a Scientific Paper, *by Robert A. Day. Philadelphia, Pa., ISI Press, 1979, 154 pp., $15.00; $8.95 (paper).*

Freud once remarked that there were three impossible professions—parenthood, diplomacy, and psychoanalysis. He might well have added editing, for the editor, especially one who watches over a scientific journal, must bring to his work the talents of parent, diplomat, and analyst all rolled into one. The insight of the analyst is often required to ferret out the message buried behind the verbiage of an opaque manuscript. In loco parentis, he must combine friendly encouragement to a whole family of authors with the ability to say no with firm assurance. And if he cannot be diplomatic, he will have few friends in this life and sparse attendance at the memorial service after his demise.

Perhaps the greatest source of despair for editors and their staffs lies in manuscripts submitted for their judgment that all too often are poorly written and prepared. No one knows this better than Robert A. Day, for many years the managing editor of the American Society for Microbiology. In his splendid book, written with clarity, conciseness, and a leavening of wit, he lets would-be authors in on the secrets of the composition of scientific manuscripts. Step-by-step he leads the reader through the mysteries of titling, organizing, writing, and typing his text, and of properly preparing his references, tables, and figures. He describes where and how to submit manuscripts and gives useful hints for dealing with editors and publishers. And in a series of chapters he provides invaluable instructions for writing simple, intelligible, jargon-free prose. Indeed, nothing has been omitted that an aspiring author will need or want to know.

One fleeting, frightening thought crossed my mind as I read this eminently practical book. If every author mastered Mr. Day's precepts, if every paper that came across the editorial desk were crisply written, well organized, scientifically sound, cogently argued, clearly typed, and perfectly arranged in conformity with his journal's specifications, what grounds would the sorely besieged editor have for rejecting a sufficient number of papers to stay within the bounds of his limited space and budget? An unlikely situation, no doubt, in an age of solecisms, and editors everywhere can, without a sense of peril, join their authors in gratitude to Mr. Day for his important contribution to scientific writing.

J.C.N.

BRIEF MENTION

Living with Schizophrenia: A Guide for Patients and Their Families, *by Alexander P. Hyde, M.D. Chicago, Ill., Contemporary Books, 1980, 128 pp., $8.95.*

The title of this book is promising. Many clinicians would like to know about good books addressed to lay people on the subject of living with mental illness. Unfortunately, this book will not fill their needs. It is full of opinion masquerading as fact. (There are three types of schizophrenia: acute, process, and paranoid.) The suggestions of recent research, when they are noted at all, are presented as unquestionable dogma. (Schizophrenia is a brain disease; schizophrenia is inherited.) It also contains a fair amount of simple misinformation or bad advice. (Schizophrenia is made worse by eating white sugar and white flour.) It is written in the popular question-and-answer format, but the questions and answers do not always follow in a clear and logical manner. This book is likely simply to confuse most of its readers.

Mental Illness in the United States: Epidemiological Estimates, *by Bruce P. Dohrenwend, Barbara Snell Dohrenwend, Madelyn Schwartz Gould, Bruce Link, Richard Neuge-*

bauer, and Robin Wunsch-Hitzig. New York, N.Y., Praeger, 1980, 157 pp., $18.95.

This slim volume grew out of the investigators' work in conjunction with the President's Commission on Mental Health, for which they prepared one of the task panels, the Panel on Nature and Scope of the Problem.

The book reviews all epidemiological field studies that examined the prevalence of psychiatric disorders in the United States and Europe after 1950. Consequently, it provides a useful compendium of a large amount of information on the epidemiology of psychiatric disorders. The problems in comparing studies that use a variety of methods are reviewed, and appropriate cautions are provided concerning interpretation of the rates presented, usually in the form of medians rather than means. Issues in definition are also discussed, such as the relationship between broad and narrow definitions of schizophrenia or between affective psychoses versus neuroses. Sometimes the conclusions to which the investigators are led seem questionable. For example, they estimate that 3% may represent the best estimate of the prevalence of schizophrenia, a rate that seems rather high. On the other hand, they estimate a rate of .3% for affective psychoses, which seems quite low, especially when compared with more recent rates found by Weissman. Rates in children and the elderly are also reviewed. As the investigators point out, the problems inherent in these data are even greater than those dealing with psychiatric disorders among adults.

The volume is useful for its discussion of the methodology of epidemiological field studies. The authors discuss such issues as the relationship between rates of treated and untreated cases and definitional problems related to imprecise or changing nosology.

This book should be of interest to researchers who want to learn more about the rates of mental illness in the United States. It contains a thorough review of past studies, usually presented with appropriate caution, and a careful and reasoned discussion of possible future approaches.

Schizophrenia and Civilization, *by E. Fuller Torrey, M.D. New York, N.Y., Jason Aronson, 1980, 221 pp., $20.00.*

Many people have wondered if mental illness is more common in civilized society than it was in more primitive times. Torrey attempts to explore the relationship between schizophrenia and civilization in order to examine the hypothesis that schizophrenia is more common in developed or industrialized countries and therefore could be considered a "disease of civilization."

He notes early in the book that most textbooks contain a global statement that schizophrenia is a common disorder that has occurred in all periods of time and in all parts of the world. Torrey has amassed a substantial amount of evidence to indicate that the truism should be abandoned. He has reviewed a substantial number of epidemiological studies and anthropological field studies. These imply that schizophrenia is unevenly distributed. For example, it is three times more common in western Ireland than in England. In India it is more common among the higher castes than the lower castes. The distribution of schizophrenia is uneven in Papua New Guinea, with a higher prevalence in those areas which have had longer contact with Western civilization.

Although Torrey's review of studies clearly attempts to be comprehensive, one gets the impression that he is marshaling it in order to build the case that schizophrenia is indeed a disease of civilization. Sometimes the case is rather weak. For example, he tries to prove that schizophrenia was rare until the nineteenth century. To do this, he overlooks or reinterprets some evidence that schizophrenia has been around for many years. For example, mania, recognized since classical times, was typically described as a psychotic disorder with many symptoms characteristic of schizophrenia. Various literary and historical documents suggest that a syndrome similar or identical to schizophrenia was present. For example, much of the Renaissance witchcraft literature describes rather classic schizophrenic syndromes. Shakespeare's Ophelia and Poor Tom are probably examples of schizoaffective disorder and hebephrenic schizophrenia.

One can find a variety of things to criticize or complain about, but nevertheless this book makes fascinating reading. Even when viewed through Torrey's interpretative eyes, the epidemiological data are absorbing and thought-provoking. Torrey attempts to be fair and present a variety of interpretations for the data. Any psychiatrist interested in schizophrenia, epidemiology, or cross-cultural psychiatry will want to read this book.

Art Psychotherapy, *by Harriet Wadeson. New York, N.Y., John Wiley & Sons, 1980, 352 pp., $26.50.*

Wadeson has spent 18 years at NIMH and in other clinical settings working as an art therapist. This book is a distillation of her experiences. Its orientation is practical and descriptive. Wadeson herself began as an artist and became a therapist, learning about psychopathology and treatment as she progressed. While this approach has clearly been productive for her, she advocates that formal training programs should include a solid grounding in the same materials that any psychotherapist must study, such as human development, abnormal psychology, and systems of psychotherapy. She describes her own therapy programs from a very practical orientation, discussing such issues as institutional politics, relationship with staff, structuring therapy sessions, and the physical environment needed.

Early in the book she discusses the various purposes and uses for art therapy. These include decreasing defenses, bringing imagery and fantasy materials to the fore, providing catharsis, improving communication, enhancing contact with others, providing pleasure and relaxation, and building self-esteem. Wadeson, rather wisely, minimizes the usefulness of art therapy as a diagnostic tool. Much of the book contains detailed descriptions of work with individual patients, illustrations of the art produced, and a discussion of therapeutic management. Many different diagnoses and problems are presented, including schizophrenia, depression, bipolar affective disorder, neuroses, and suicide.

This volume represents a pioneer effort. As a consequence, it should be of value to psychiatrists interested in art, creativity, and approaches to psychotherapy through various artistic media. Although the author is wise in her attempt to avoid excessive generalization, the book is marred by an excessive emphasis on detail and specificity. One would like her to step back from time to time and use the large canvas in addition to the miniature.

N.C.A.

Neurology for Psychiatrists, *by Charles E. Wells, M.D., and Gary W. Duncan, M.D. Philadelphia, Pa., F.A. Davis Co., 1980, 233 pp., $22.00.*

This is a good and valuable book. It covers an area of importance to every psychiatrist and neurologist—the behavioral aspects of neurologic disorders and the practical aspects of the differentiation of neurologic disorders from psychiatric disorders. This book represents one of several books and journal articles that signal the increasing importance of knowledge of the CNS for psychiatrists.

The most crucial decision confronting an author who intends to cover such a vast subject as neurology is that of the organization and selection of his material. Drs. Wells and Duncan have organized their material into topics relating to disturbed function. Chapters dealing with such things as dementia, delirium, syncope and dizziness, seizure disorders, and head trauma help organize this potentially vast area into manageable categories. Consequently, the topics lend themselves to categorization into clinical problems; in this sense the text will be extremely useful to the clinician.

The subject matter of this book is clinical. The material should be mastered by every psychiatric resident. Practicing clinicians will also find this book a useful consultant as they struggle with patients who present difficult diagnostic problems. Almost every page is full of clinical "pearls" that are useful either diagnostically or for management. The clinical and practical orientation of this book is its major strength. It is also the basis on which this book should be judged. Reviewers often use their review of another's book to discuss the book they would have written; in this sense there are many paths that the authors of this volume did not tread. There is little discussion of etiology, which may reflect the lack of knowledge of etiology of most neurologic conditions; the basic descriptions and natural history of most neurologic conditions are very briefly covered; and neurochemistry and neuroanatomy are almost nonexistent in the text. However, the topics left out are in large part not necessary to the book because it is a clinical aid for the psychiatrist (and the neurologist) in differential diagnosis. As such it is excellent.

Certainly this volume, especially for trainees, must be used along with other texts, but the book and its valuable bibliography are unique, pulling together in one place a diverse and confusing literature for the clinician. Although this book is somewhat difficult to read at one sitting because it is so full and because of some rather involved sentence structure, it is the kind of book that should be kept at hand and read in relation to specific cases. Used in this manner it will help fulfill the intent of the authors' statement, "Psychiatrists should be wary of accepting the diagnostic conclusions of referring physicians and should refine their own diagnostic skills so that costly diagnostic and therapeutic errors are avoided" (p. 75).

GARY J. TUCKER, M.D.
Hanover, N.H.

Transsexuality in the Male: The Spectrum of Gender Dysphoria, *by Erwin E. Koranyi, M.D. Springfield, Ill., Charles C Thomas, 1980, 191 pp., $17.50.*

In the 1960s, when transsexuality was a subject of relatively recent medical interest, extensive monographs were produced by Benjamin and later by Green and Money. At that time gender change through surgical transformation and hormone administration was more startling and aroused more resistance in the medical profession than it does today. In undertaking this book, Dr. Koranyi proposes to update the readers' knowledge concerning the medical and legal aspects of transsexuality in the male and the interrelations with transvestism and homosexuality. Since he confines himself to only 140 pages (not counting surgical plates, glossary, and bibliography), readers may disagree with his priorities. However, the chapters on sexual dimorphism, genetics of sexuality, and clinical cases are thorough and timely. A long chapter on the medical-legal aspects of the sexual dysphorias and legal complications of sex reassignment surgery widens the interest of this volume to the fields of social work and the law.

The major disappointment in this book is the author's bias in favor of surgical treatments. He dismisses psychological forms of treatment "because the reported successes are few in number and represent a self-selected subgroup remote from the typical cases." The explanation offered for this exclusive view is that transsexuals are rarely interested in what these treatments can offer and, by contrast, they welcome the sex-change operation. Although this consumer approach is enjoying a vogue in clinical practice nowadays, it would still be more appropriate for a medical writer to present the whole scope of treatments available even if some are not popular with patients.

Given the limitations of our present knowledge of the etiology of transsexualism, a good argument can be presented that the disorder is a gender disorientation. A behavioral approach, which deals with all the ramifications of gender in regard to mannerism, gait, and so forth as well as sexual arousal stimuli, gives promise of successful outcome. The omission of medical reports of these therapies from the monograph and even from the bibliography is a disservice to the reader, who has a right to expect complete coverage. Despite this shortcoming, readers will be pleased with the conciseness and readability of this monograph.

FRANK G. BUCKNAM, M.D.
Honolulu, Hawaii

The Neurobiology of Dopamine, *edited by A.S. Horn, J. Korf, and B.H.C. Westernik. London, England, Academic Press, 1979, 677 pp., $83.00.*

This volume brings together contributions covering a wide area of dopamine research. The book covers topics that range from the chemistry of dopamine to the biology, pharmacology, behavioral aspects, and, finally, the functional roles of dopamine in the CNS of man. The editors' aim was "to bring together in one volume as many important facets of dopamine research as possible." To this end, 36 chapters were assembled by 43 contributors and a massive volume emerged. The book is well edited, the chapters are clear, and the coverage is comprehensive, yet the question remains for whom this was put together. Certainly the psychiatrist would need a sincere interest in neurochemical, physiological, and behavioral research to wade into such a volume. The researcher, on the other hand, might find that his or her area is not sufficiently detailed or timely enough to be of help. Thus the volume appears to me to be a reference work for people

interested in dopamine who wish to know what is going on beyond the flanks of their specialized interests. In general this book is an excellent source for such a person.

The volume begins with a short review by Anden followed by a section on chemistry and the analysis of dopamine. The chapters on methods of analysis are an especially helpful source of information about the various techniques used to measure dopamine. They are followed by a chapter on the enzymes involved in dopamine metabolism. It was in this chapter that the tremendous problems remaining in dopamine research began to be apparent. The fine chapter on tyrosine hydroxylase by Roth illustrates the puzzles that remain in understanding the regulation of this crucial enzyme. The chapter on dopamine-sensitive adenylate cyclase coupled with the later review of dopamine receptors illustrates the problems in attempting to understand the functional consequences of receptor binding. Unfortunately, the rapid developments in receptor work make this section out of date. The current concepts of D_1 and D_2 receptors (1) are not mentioned, nor is the role of GTP (2), which appears to be important in determining receptor behavior.

The section on anatomy provides an excellent review of fluorescence staining and pathways but, unfortunately, does not address the problems of microanatomy; namely, where do boutons synapse and how are they distributed? The neurophysiology chapter by York defining dopamine as an excitatory neurotransmitter in the striatum appears juxtaposed with Bunney's account of dopamine's inhibition and its modulation by drugs with little or no straightforward explanation of how to relate these two sets of results. The problems of defining the role of cyclic AMP in dopamine's postsynaptic action is highlighted in Libet's chapter. Thus the middle section of the book, in addition to reviewing a wealth of data, illustrates the problems and ignorance behind many of the explanations of dopamine's actions found in textbooks.

The final sections deal with dopamine and behavior and clinical aspects of dopaminergic systems. The behavioral section begins with a discussion of agonists, antagonists, and the behavioral classification of D_1 (hyperactivity) and D_2 (dyskinesia) receptors. Ungerstedt reviews the effect of dopamine on unconditioned behavior; finally, neural mechanisms of reinforcement are examined. The chapter on clinical aspects of dopamine is an excellent review of the measurement of dopamine in human fluids and tissues. This is followed by two chapters that would be of special interest to psychiatrists: Hornykiewcz on dopamine in neurological conditions and van Praag on dopamine in psychiatric conditions. This finale emphasizes the enormous impact of dopamine research on clinical practice. In summary, this book is an excellent reference text illustrating the current problems in trying to understand dopamine's actions at the tissue and cellular levels in addition to pointing to the solid achievements that past research has made in solving clinical problems.

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FRITZ HENN, M.D., PH.D.
Iowa City, Iowa

The Interface Between the Psychodynamic and Behavioral Therapies, *edited by Judd Marmor, M.D., and Sherwyn M. Woods, M.D., Ph.D. New York, N.Y., Plenum Medical Book Co., 1980. 377 pp., $22.50.*

Investigation of the nature of the therapeutic action of psychotherapy is one of the more urgent and interesting tasks of psychiatry at the present time. This interest is fueled by the evident lack of information in a crucial area of endeavor and by the proliferation of psychotherapeutic techniques, both traditional and "far-out." Without some better knowledge of the mechanisms of psychotherapy, it is difficult or hopeless to attempt to make sense of the clamor of claims for therapeutic success that assault us. This fine book presents an approach to this task by bringing together 25 important papers that have appeared during the last two decades concerning the relationship of psychodynamic to behavioral therapy. The papers discuss the two main streams of psychotherapy today, their common and different elements of therapeutic technique, the nature of their theoretical bases, and methods of combining their clinical techniques to enhance the therapeutic benefit. Marmor and Woods have organized the chapters into theoretical and clinical sections and have provided excellent brief introductions to each paper.

Although the papers vary in quality, they have been selected for a consistency of approach (at times a bit repetitious) and succeed in establishing a number of important points. Several excellent papers deal with the claims of some behaviorists that theirs is a psychotherapy derived entirely from scientifically validated, controlled laboratory investigation and that their clinical claims represent a similar degree of control and validity. The authors of these chapters make it clear that the leap from laboratory experiment to the treatment of human neurosis is an act of faith rather than a simple scientific application and that the therapeutic regimens derived by behavioral theorists are far more complex and "tainted" by interpersonal events than is accounted for in their terminology or data. Techniques such as relaxation or the imagining of frightening stimuli do not lend themselves to description in stimulus-response terms. As Breger and McGaugh point out, "The laws or principles of conditioning and reinforcement which form the basis of their [Wolpe, Eysenck, Skinner, and their followers] learning theory are insufficient explanations for the findings from laboratory experiments, let alone the complex learning phenomena that are encountered in psychotherapy" (p. 52).

After laying to rest the misleading issue of "true science" versus "false dogma," the editors present a group of papers attempting to outline the kinds of epistemologic, research, and clinical efforts that would enrich both schools of thought. These papers, of varying degrees of sophistication, attempt to find a common ground in an expanded learning theory that would explain the phenomena observed in the different psychotherapeutic situations. Wolf's paper, following a superb discussion of the differences between laboratory learning and the ordinary human circumstances in which neurotic behavior develops and becomes self-sustaining, begins the necessary task of attempting to understand the nature of the clinical research that would help us to develop a better learning theory, taking into account the events occurring in psychotherapeutic situations. There is general agreement that the core theoretical problem is to formulate a better learning theory, but we are not given a discussion of the nature of learning theory itself. Is all behavior learned? Is

there anything in psychological maturation and development that is not subsumed by learning theory? Many would answer yes, but the problem is not addressed.

The clinical portion of the book presents a sampling of the literature on combined psychodynamic and behavioral therapies, with clinical reports of integrated, concurrent but separate, alternating, and sequential therapies. The evidence indicates that selected patients will benefit from combined therapies and that problems are likely to arise from failures of cooperation and communication between therapists rather than from difficulties created by patients. In the psychotherapeutic situations described, the predicted problems of split transferences and special transference resistances seemed minimal. These reports of combined therapy are sufficient in quantity and quality of the work that there can be no doubt there are clinical situations in which it is the treatment of choice.

Although there are polemic papers in the volume, the tone is empirical and ecumenical, steering clear of "true believers" and trying to make a contribution to theory and therapy from the interaction of these powerful points of view. The book reminds dynamic psychotherapists of the need to clarify their terminology and tease apart the great variety of techniques they use in their work. Equally, the myth of purely scientific behaviorists is dispelled, and they are helped to bring back the interest and complexity of the psyche to their field of awareness. Anyone doing any variety of psychotherapy will benefit from reading this book and is likely to approach his or her work with a keener appreciation and questioning of his or her methods. I have a suggestion for the second edition: place the dates of the papers in the table of contents so that the reader can better appreciate the historical development of this line of thought.

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New York, N.Y.

The Use and Misuse of Sleeping Pills: A Clinical Guide, *by Wallace B. Mendelson. New York, Plenum Medical Book Co., 1980, 220 pp., $22.50.*

In this clinical guide on sleeping pills Dr. Mendelson presents a basic but comprehensive overview of the state of the art with respect to insomnia and the use of hypnotics. This is no small task, given both the present controversy surrounding these topics and our ever-increasing awareness that the insomnias and treatment by sleeping medication are neither simply nor directly related. That this book tends overall to further highlight this disparity may, in fact, represent its single most important contribution as a clinical guide.

The book is written in a manner that allows both student and experienced professional alike to read it. Each of the eight chapters devoted to hypnotic use and abuse uses a format that first provides interesting historical and essential background data, followed by an extensive and detailed review of relevant research data, and ending with a brief but concise summary of the major points covered. The work opens with a discussion of the basic concepts of sleep that emphasizes the current approach to insomnia as a complaint rather than as a primary diagnosis. Mendelson then presents data on the prevalence of sleep disturbance and hypnotic use. These first two chapters probably could have been expanded to place greater emphasis on the lack of clarity we find in the definition of insomnia. The major prescription hypnotics are then discussed in terms of basic pharmacology, efficacy, and specific risk factors.

Although these chapters are readable and complete, they fail to identify, especially for the student, those drugs which are most often prescribed not as sleeping medication but for the treatment of anxiety, agitation, and depression. The daytime sedative and hypnotic effects of these compounds are often secondary to the major purpose for which they are prescribed. A discrimination between these drugs and compounds administered specifically as sleeping pills needs to be underlined. In addition, the text might have benefited from further discussion of sleeping problems related to medical disease and potential drug interactions resulting from treatments of the medical diseases.

The author is to be complimented for stating in the section on hypnotic dependence that 15%–20% of patients who are started on hypnotics might very well be beginning the long road to chronic hypnotic use. Of particular note in the latter regard is a chapter dealing with some relatively new findings on the residual daytime effects of these compounds. Of perhaps even greater pragmatic importance is a chapter dealing with the use of hypnotics in the elderly, for whom a disproportionately high number of these agents are prescribed. Dr. Mendelson also includes a chapter on other pharmacologic approaches to insomnia and provides some interesting and little-publicized information on the use of over-the-counter hypnotics.

Nonpharmacologic approaches in the treatment of insomnia are rather sparsely reviewed and discussed, but since this text primarily deals with pharmacologic interventions, the bias can be condoned. Dr. Mendelson concludes with a discussion of the implications of the information presented for medical practice and offers some clinical guidelines for the prescribing of hypnotics that emphasize not only a more comprehensive assessment of the complaints but also increased attention to the risk-benefit ratios of pharmacologic treatment strategies.

In summary, *The Use and Misuse of Sleeping Pills* is an informative book that provides basic information for the student and a comprehensive review of the latest technical information for the more experienced clinician or researcher. As a clinical guide it does provide some practical advice, but it may best serve as a testament to the relative lack of scientific knowledge in this area.

DAVID J. KUPFER, M.D.
PATRICIA A. COBLE, R.N.
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Psychiatric Emergencies in Pediatrics, *by Aman U. Khan, M.D. Chicago, Ill., Year Book Medical Publishers, 1979, 244 pp., $21.95.*

Child psychiatry and pediatrics are extending tentative fingers toward one another, hoping to preserve their autonomy while expanding their horizons. The links formed to date are "pediatric psychiatry" and "behavioral pediatrics." As pediatrics, facing a declining birthrate and increasingly healthy children, increases its commitment to improving the emotional lot of children, we can expect a spate of books to be published aimed at this market. *Psychiatric Emergencies in Pediatrics* is one of the growing number of these.

Designed to help physicians and other health care providers to understand and to manage the psychiatric problems commonly encountered in emergency rooms and acute care clinics, this book uses the time-honored technique of reviewing the literature. Despite a few glaring omissions such as amphetamine psychosis, this book reviews the literature in a thorough, knowledgeable, and informative fashion. It is a cornucopia of information, assembled for thoughtful reading and for leisurely digestion. Yet in just that strength lies its difficulty: it is not focused and organized in a format that stresses emergencies, a format that would make it truly useful to the provider of pediatric services on the front lines.

One of the book's chapters, "Management of the Rape Victim," illustrates in its excellence the dilemma of other sections. The main thrust of this chapter is a description of raped patients who are brought to the emergency room, what the physician should and should not do immediately, and how thoughtful follow-up can help prevent later complications. Were the entire book written as this one is, it would serve its end far better.

As a book for emergency room and acute care personnel, this book does not meet its goal. Yet it is worthy of mention because, as a review of the literature for the beginning child psychiatry resident or other mental health professional, it does more than an adequate job. The book's publication also raises several important questions: What behavioral interventions should pediatricians make? Where are the boundaries between the disciplines? It would be a tragedy if, instead of increasing collaborative efforts between the disciplines, books such as this led one or the other field to think the two were interchangeable.

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This Month's Reviewers

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Books Received

This listing acknowledges the receipt of recent books. Books of particular interest to the readers of this journal will be reviewed as space permits, and copies will be sent to the publishers. Books cannot be returned to the publishers.

Dimensions of Family Therapy, *edited by Maurizio Andolfi and Israel Zwerling. New York, N.Y., Guilford Press, 1980, 280 pp., $20.00.*

The Skills of Diagnostic Planning: Psychiatric Rehabilitation Series, Book 1, *by William A. Anthony, Ph.D., Richard M. Pierce, Ph.D., Mikal R. Cohen, Ph.D., and John R. Cannon, Ph.D. Baltimore, Md., University Park Press, 1980, 177 pp., $8.95 (paper).*

The Skills of Rehabilitation Programming: Psychiatric Rehabilitation Practice Series, Book 2, *by William A. Anthony, Ph.D., Richard M. Pierce, Ph.D., Mikal R. Cohen, Ph.D., and John R. Cannon, Ph.D. Baltimore, Md., University Park Press, 1980, 114 pp., $7.50 (paper).*

Body Symbolism and the Somatic Inkblot Series, *by Wilfred A. Cassell, M.D. Vancouver, B.C., Canada, Northern Lights Publishing, 1980, 216 pp., no price listed (paper).*

Manual for Clinical Psychology Practicums, *by James Choca, Ph.D. New York, N.Y., Brunner/Mazel, 1980, 158 pp., $15.00; $9.95 (paper).*

Psychological Models and Neural Mechanisms, *by Austen Clark. Oxford, England, Clarendon Press, 1980, 201 pp., $34.50.*

The Lineaments of Mind in Historical Perspective, *by John Cohen. San Francisco, Calif., W.H. Freeman and Co., 1980, 305 pp., $19.95.*

The Skills of Professional Evaluation: Psychiatric Rehabilitation Practice Series, Book 3, *by Mikal R. Cohen, Ph.D., William A. Anthony, Ph.D., Richard M. Pierce, Ph.D., Leroy A. Spaniol, Ph.D., and John R. Cannon, Ph.D. Baltimore, Md., University Park Press. 1980, 72 pp., $5.95 (paper).*

Psychopharmacology Updated, *edited by Jonathan O. Cole, M.D. Lexington, Mass., Collamore Press, 1980, 195 pp., no price listed.*

What Is a Doctor? Essays on Medicine and Human Natural History, *by Alex Comfort, Ph.D. Philadelphia, Pa., George F. Stickley Co., 1980, 232 pp., $10.95.*

Clifford W. Beers: Advocate for the Insane, *by Norman Dain. Pittsburgh, Pa., University of Pittsburgh Press, 1980, 384 pp., $19.95.*

Anorexia Nervosa, *by Peter Dally and Joan Gomez. London, England, William Heinemann Medical Books, 1980, 209 pp., $24.00.*

Obesity and Anorexia Nervosa: A Question of Shape, *by Peter Dally and Joan Gomez. London, England, Faber and Faber, 1980, 117 pp., $6.95 (paper).*

Suicide and Bereavement, *edited by Bruce L. Danto and Austin H. Kutscher. New York, N.Y., MSS Information Corp. (Arno Press), 1980, 260 pp., $15.50.*

Thomas Willis's Oxford Lectures, *by Kenneth Dewhurst. Oxford, England, Sandford Publications, 1980, 161 pp., no price listed.*

The Human Psyche: The Gifford Lectures, University of Edinburgh 1978–1979, *by John Eccles. Secaucus, N.J., Springer-Verlag New York, 1980, 268 pp., $25.00.*

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Each entry in the subject index is identified by the surname of the first author only; semi-colons between names of authors of letters to the editor indicate multiple letters. All entries are in abbreviated style, and two-letter abbreviations are used for months. Entries other than regular articles, brief communications, and clinical and research reports are given the following designations: book review (bk rev), letter to the editor (ltr to ed), editorial (edtl), in memoriam (in mem), and official actions (off acts).

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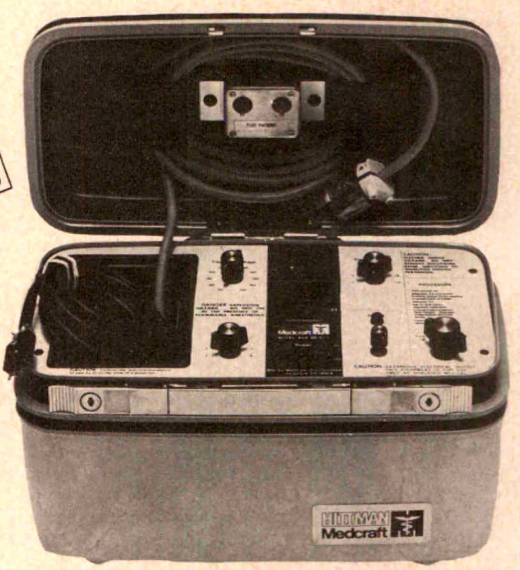
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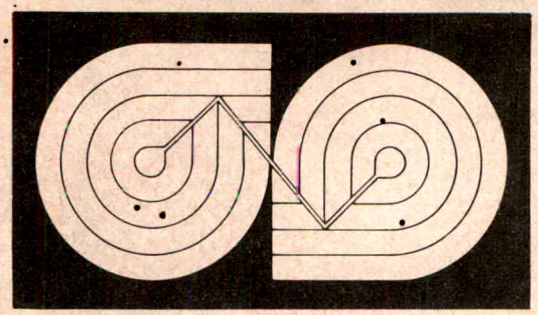
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1. Talley JH, in "Treat depression as the curable disease it is." *Patient Care* 11:20-41, 1977.

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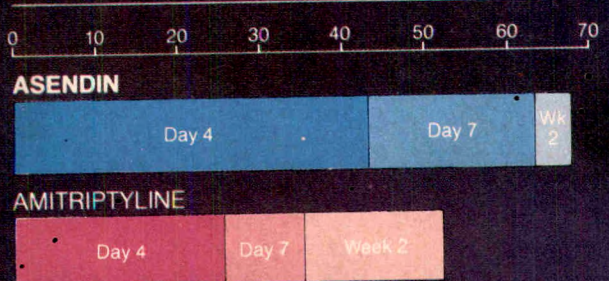
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Rapidly relieves important symptoms of depression, often in the early days of therapy—
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ASENDIN® amoxapine provided greater relief than amitriptyline for key anxiety-depression symptoms on days 4 and 7 of therapy

PERCENT (%) MAXIMUM POSSIBLE IMPROVEMENT IN A CLUSTER OF ANXIETY-DEPRESSION SYMPTOMS–DOUBLE-BLIND 71-PATIENT STUDY*



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Rapidly relieves sleep disturbances in most patients, usually without impairment of daytime alertness...

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Now...in depression

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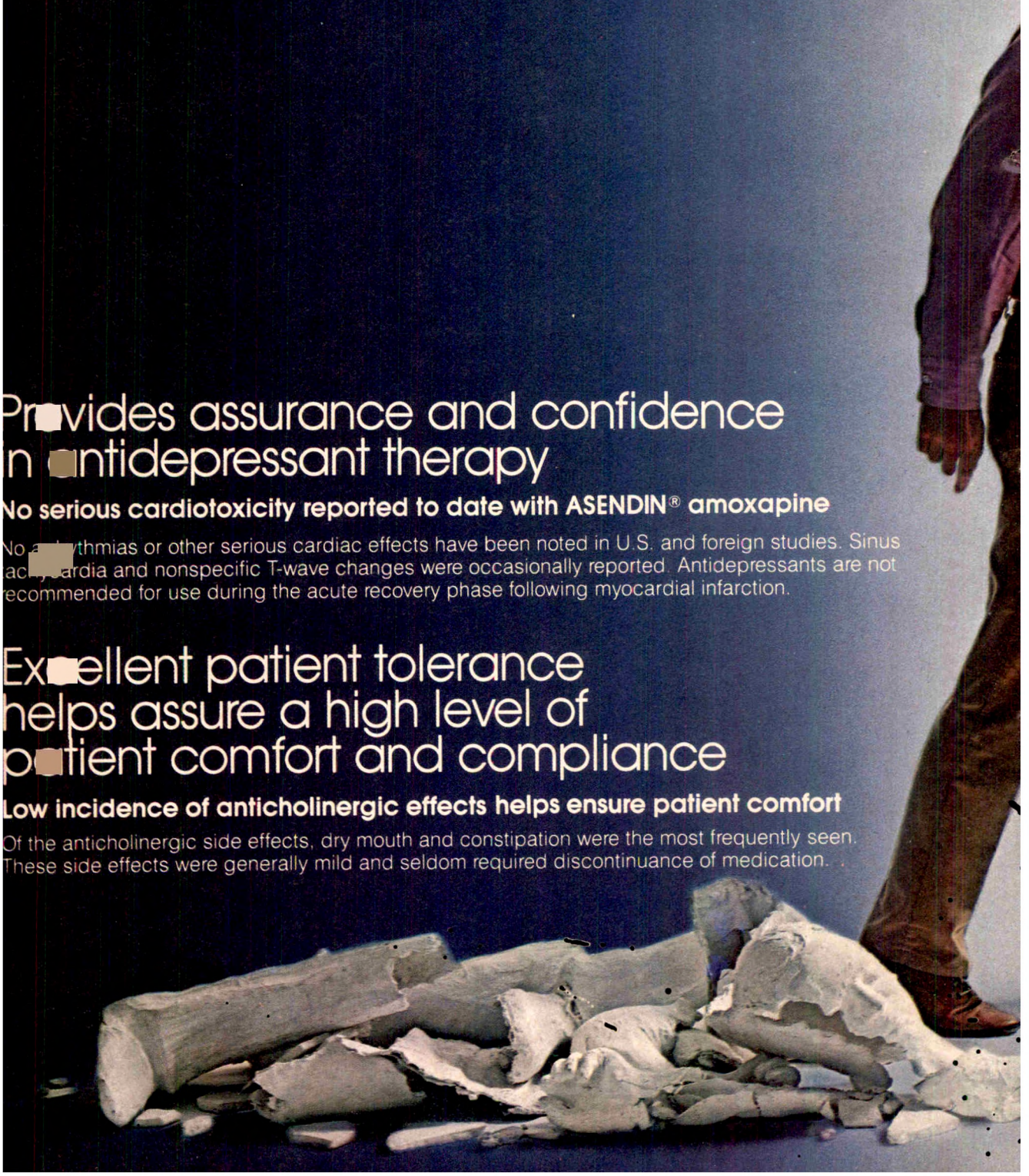
No serious cardiotoxicity reported to date with ASENDIN® amoxapine

No arrhythmias or other serious cardiac effects have been noted in U.S. and foreign studies. Sinus tachycardia and nonspecific T-wave changes were occasionally reported. Antidepressants are not recommended for use during the acute recovery phase following myocardial infarction.

Excellent patient tolerance helps assure a high level of patient comfort and compliance

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Recommended adult daily dosage	
Usual initial dosage Depending on age and tolerance	50 mg + 50 mg + 50 mg
Optional initial dosage Suitable for outpatients	50 mg + 50 mg
Usual effective dosage by end of first week	200 mg to 300 mg in divided doses
Usual maintenance dosage	Lowest dose that will maintain remission

When effective dosage is established, the drug may be given in a *single daily dose at bedtime*—not to exceed 300 mg.

See ASENDIN prescribing information on last page for more detailed information concerning side effects.

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Because every day counts in the relief of depression



DESCRIPTION
ASENDIN **amoxapine** is an antidepressant of the dibenzoxazepine class, chemically distinct from the dibenzazepines, dibenzocycloheptenes, and dibenzoxepines.

It is designated chemically as 2-chloro-11-(1-piperazinyl) dibenz[b,f]-[1,4] oxazepine. The molecular weight is 313.8. The empirical formula is $C_{17}H_{16}ClN_3O$.

ASENDIN is supplied for oral administration as 50 mg, 100 mg, and 150 mg tablets.

CLINICAL PHARMACOLOGY
ASENDIN is an antidepressant with a mild sedative component to its action. The mechanism of its clinical action in man is not well understood. In animals, amoxapine reduced the uptake of norepinephrine and serotonin and blocked the response of dopamine receptors to dopamine. Amoxapine is not a monoamine oxidase inhibitor.

ASENDIN is absorbed rapidly and reaches peak blood levels approximately 90 minutes after ingestion. It is almost completely metabolized. The main route of excretion is the kidney. *In vitro* tests show that amoxapine binding to human serum is approximately 90%.

In man, amoxapine serum concentration declines with a half-life of 8 hours. However, the major metabolite, 8-hydroxyamoxapine, has a biologic half-life of 30 hours. Metabolites are excreted in the urine in conjugated form as glucuronides.

Clinical studies have demonstrated that ASENDIN has a more rapid onset of action than either amitriptyline or imipramine. The initial clinical effect may occur within four to seven days and occurs within two weeks in over 80% of responders.

INDICATIONS AND USAGE
ASENDIN is indicated for the relief of symptoms of depression in patients with neurotic or reactive depressive disorders as well as endogenous and psychotic depressions. It is indicated for depression accompanied by anxiety or agitation.

CONTRAINDICATIONS
ASENDIN is contraindicated in patients who have shown prior hypersensitivity to dibenzoxazepine compounds. It should not be given concomitantly with monoamine oxidase inhibitors. Hyperpyretic crises, severe convulsions, and deaths have occurred in patients receiving tricyclic antidepressants and monoamine oxidase inhibitors simultaneously. When it is desired to replace a monoamine oxidase inhibitor with ASENDIN, a minimum of 14 days should be allowed to elapse after the former is discontinued. ASENDIN should then be initiated cautiously with gradual increase in dosage until optimum response is achieved. The drug is not recommended for use during the acute recovery phase following myocardial infarction.

WARNINGS
ASENDIN should be used with caution in patients with a history of urinary retention, angle-closure glaucoma or increased intraocular pressure. Patients with cardiovascular disorders should be watched closely. Tricyclic antidepressant drugs, particularly when given in high doses, can induce sinus tachycardia, changes in conduction time, and arrhythmias. Myocardial infarction and stroke have been reported with drugs of this class.

There have been rare reports of grand mal seizures with dosages of ASENDIN above recommended limits. Extreme caution should be taken in patients with a history of convulsive disorders.

PRECAUTIONS
General:
In prescribing the drug it should be borne in mind that the possibility of suicide is inherent in any severely depressed patient, and persists until a significant remission occurs; the drug should be dispensed in the smallest suitable amount. Manic depressive patients may experience a shift to the manic phase. Schizophrenic patients may develop increased symptoms of psychosis; patients with paranoid symptomatology may have an exaggeration of such symptoms. This may require reduction of dosage or the addition of a major tranquilizer to the therapeutic regimen.

Information for the patient:
Patients should be warned of the possibility of drowsiness that may impair performance of potentially hazardous tasks such as driving an automobile or operating machinery.

Drug interactions:
See "Contraindications" about concurrent usage of tricyclic antidepressants and monoamine oxidase inhibitors. Paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic drugs. ASENDIN may enhance the response to alcohol and the effects of barbiturates and other CNS depressants.

Therapeutic interactions:
Concurrent administration with electroshock therapy may increase the hazards associated with such therapy.

Carcinogenesis, impairment of fertility:
In a 21-month toxicity study at 3 dose levels in rats, pancreatic islet cell hyperplasia occurred with slightly increased incidence at doses 5-10 times the human dose. Pancreatic adenocarcinoma was detected in low incidence in the mid-dose group only, and may possibly have resulted from endocrine-mediated organ hyperfunction. The significance of these findings to man is not known.

Treatment of male rats with 5-10 times the human dose resulted in a slight decrease in the number of fertile matings. Female rats receiving oral doses within the therapeutic range displayed a reversible increase in estrous cycle length.

Pregnancy. Pregnancy category C:
Studies performed in mice, rats and rabbits have demonstrated no evidence of teratogenic effect due to ASENDIN. Embryotoxicity was seen in rats and rabbits given oral doses approximating the human dose. Fetotoxic effects (intrauterine death, stillbirth, decreased birth weight) were seen in animal studies at oral doses 3-10 times the human dose. Decreased postnatal survival (between days 0-4) was demonstrated in the offspring of rats at 5-10 times the human dose. There are no adequate and well-controlled studies in pregnant women. ASENDIN should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing mothers:
It is not known whether this drug is excreted in human milk, but amoxapine and/or its metabolites have been shown to be freely transported into milk of lactating rats.

Pediatric use:
Safety and effectiveness in children below the age of 16 have not been established.

ADVERSE REACTIONS
Adverse reactions reported in controlled studies in the United States are categorized with respect to incidence below. Following this is a listing of reactions known to occur with other antidepressant drugs of this class but not reported to date with ASENDIN.

INCIDENCE GREATER THAN 1%

The most frequent types of adverse reactions occurring with ASENDIN in controlled clinical trials were sedative and anticholinergic: these included drowsiness (14%), dry mouth (14%), constipation (12%), and blurred vision (7%).

Less frequently reported reactions were:
CNS and Neuromuscular—anxiety, insomnia, restlessness, nervousness, palpitations, tremors, confusion, excitement, nightmares, ataxia, alterations in EEG patterns.
Allergic—skin rash, edema.
Gastrointestinal—Nausea.
Other—dizziness, headache, fatigue, weakness, excessive appetite, increased perspiration.

INCIDENCE LESS THAN 1%
Anticholinergic—disturbances of accommodation, mydriasis, delayed micturition, nasal stuffiness.
Cardiovascular—hypotension, hypertension, syncope, tachycardia.
Allergic—drug fever, photosensitization, pruritus.
CNS and Neuromuscular—tingling, paresthesias of the extremities, tinnitus, disorientation, extrapyramidal symptoms, seizures, hypomania, numbness.
Hematologic—Leukopenia.
Gastrointestinal—epigastric distress, vomiting, flatulence, abdominal pain, peculiar taste, diarrhea.
Endocrine—increased or decreased libido, impotence, menstrual irregularity, breast enlargement and galactorrhea in the female.
Other—lacrimation, weight gain or loss, altered liver function.

DRUG RELATIONSHIP UNKNOWN
Other reactions have been reported rarely but occurred under circumstances where a drug relationship was unknown. These observations are listed to serve as alerting information to physicians.

Cardiovascular—stroke.

Other—urinary frequency, testicular swelling, anorexia.

ADDITIONAL ADVERSE REACTIONS REPORTED WITH OTHER ANTIDEPRESSANT DRUGS
Anticholinergic—Sublingual adenitis, paralytic ileus, urinary retention, dilation of urinary tract.

Cardiovascular—arrhythmias, myocardial infarction, heart block.

Allergic—urticaria, petechiae.

CNS and Neuromuscular—disturbed concentration, delusions, hallucinations, peripheral neuropathy, incoordination, syndrome of inappropriate ADH (antidiuretic hormone) secretion.

Hematologic—agranulocytosis, eosinophilia, purpura, thrombocytopenia.

Gastrointestinal—stomatitis, parotid swelling, black tongue.

Endocrine—gynecomastia, elevation and lowering of the blood sugar levels.

Other—alopecia, hepatitis (including jaundice).

OVERDOSAGE
There has been limited experience with overdosage of ASENDIN. One young female patient experienced a convulsive seizure following ingestion of 1000 mg; no other serious adverse reactions or sequelae were reported in this case. Another young woman ingested 2700 mg ASENDIN together with 325 mg imipramine and 210 mg flurazepam without serious adverse effect. She reportedly slept for 14 hours and required no treatment. Electrocardiographic and laboratory findings obtained 26 hours post drug were normal.

In general, treatment of overdosage must be symptomatic and supportive. If the patient is conscious, induced emesis followed by gastric lavage with appropriate precautions to prevent pulmonary aspiration should be accomplished as soon as possible. Following lavage, activated charcoal may be administered to reduce absorption. An adequate airway should be established in comatose patients and assisted ventilation instituted if necessary. The possibility of occurrence of seizures should be kept in mind. Convulsions, should they occur, may respond to standard anticonvulsant therapy; however, barbiturates may potentiate any respiratory depression. Specific treatment should be guided by the predominant symptoms which may suggest use of a particular pharmacologic agent. For example, the slow intravenous administration of physostigmine salicylate has been reported to reverse most of the serious cardiovascular and CNS effects of overdosage with tricyclic antidepressants, such as cardiac arrhythmias and convulsions. Avoid rapid injection to reduce the possibility of physostigmine-induced convulsions.

A patient who has ingested a toxic overdose of a tricyclic antidepressant may remain medically and psychiatrically unstable for several days due to sustained excessive drug levels. Unexpected cardiac deaths have occurred up to six days post overdose with other antidepressants. The QRS interval of the electrocardiogram appears a reliable correlate of the severity of overdosage. If the QRS interval exceeds 100 milliseconds anytime during the first 24 hours after overdose, cardiac function should be continuously monitored for five or six days.

DOSAGE AND ADMINISTRATION
Effective dosage of ASENDIN may vary from one patient to another. Usual effective dosage is 200 mg to 300 mg daily. Three weeks constitutes an adequate period of trial providing dosage has reached 300 mg daily (or a lower level of tolerance) for at least two weeks. If no response is seen at 300 mg, dosage may be increased, depending upon tolerance, up to 400 mg daily. Hospitalized patients who have been refractory to antidepressant therapy and who have no history of convulsive seizures may have dosage raised cautiously up to 600 mg daily in divided doses.

ASENDIN may be given in a single daily dose, not to exceed 300 mg, preferably at bedtime. If the total daily dosage exceeds 300 mg, it should be given in divided doses.

Initial Dosage for Adults—Usual starting dosage is 50 mg three times daily. Depending upon tolerance, dosage may be increased to 100 mg three times daily on the third day of treatment. (Initial dosage of 300 mg daily may be given, but notable sedation may occur in some patients during the first few days of therapy at this level.) Increases above 300 mg daily should be made only if 300 mg daily has been ineffective during a trial period of at least two weeks. When effective dosage is established, the drug may be given in a single dose (not to exceed 300 mg) at bedtime.

Elderly Patients—In general, lower dosages of the tricyclic antidepressants are recommended for these patients. Recommended starting dosage of ASENDIN is 25 mg three times daily. If no intolerance is observed, dosage may be increased after three days to 50 mg three times daily. Although 150 mg daily may be adequate for many elderly patients, some may require higher dosage. Careful increases up to 300 mg daily are indicated in such cases.

Once an effective dosage is established, ASENDIN may conveniently be given in a single bedtime dose, not to exceed 300 mg.

Maintenance—Recommended maintenance dosage of ASENDIN is the lowest dose that will maintain remission. If symptoms reappear, dosage should be increased to the earlier level until they are controlled.

For maintenance therapy at dosage of 300 mg or less, a single dose at bedtime is recommended.

HOW SUPPLIED
ASENDIN *amoxapine* tablets are supplied as follows:
50 mg heptagonal, scored tablets, orange —bottles of 100.
100 mg heptagonal, scored tablets, blue —bottles of 100.
150 mg heptagonal, scored tablets, peach —bottles of 30.

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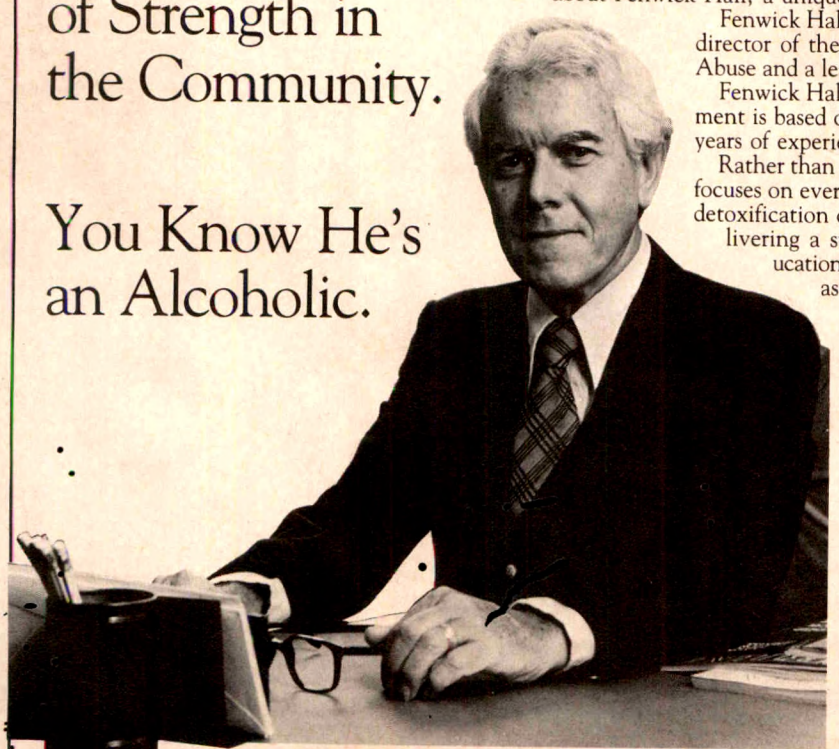
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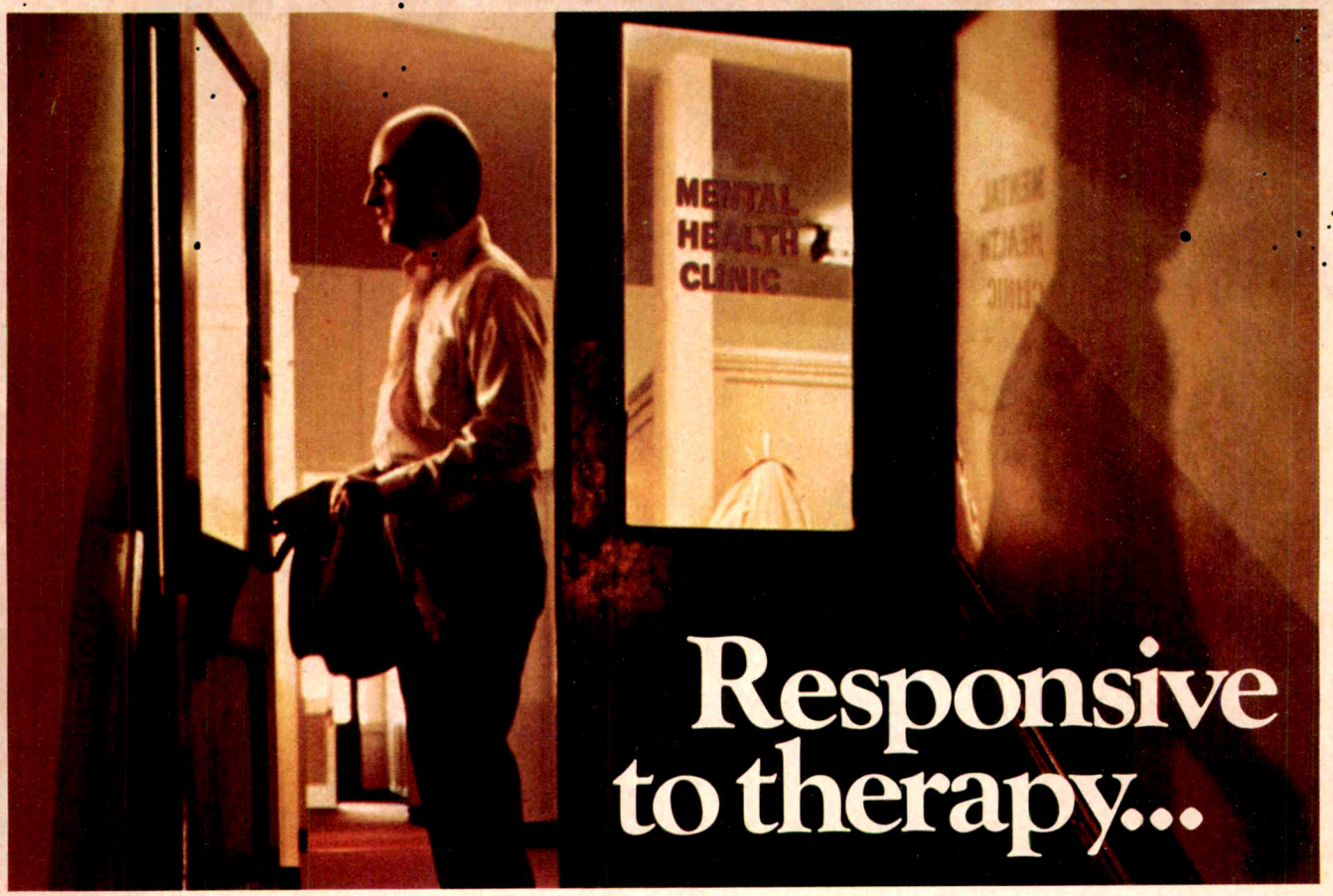
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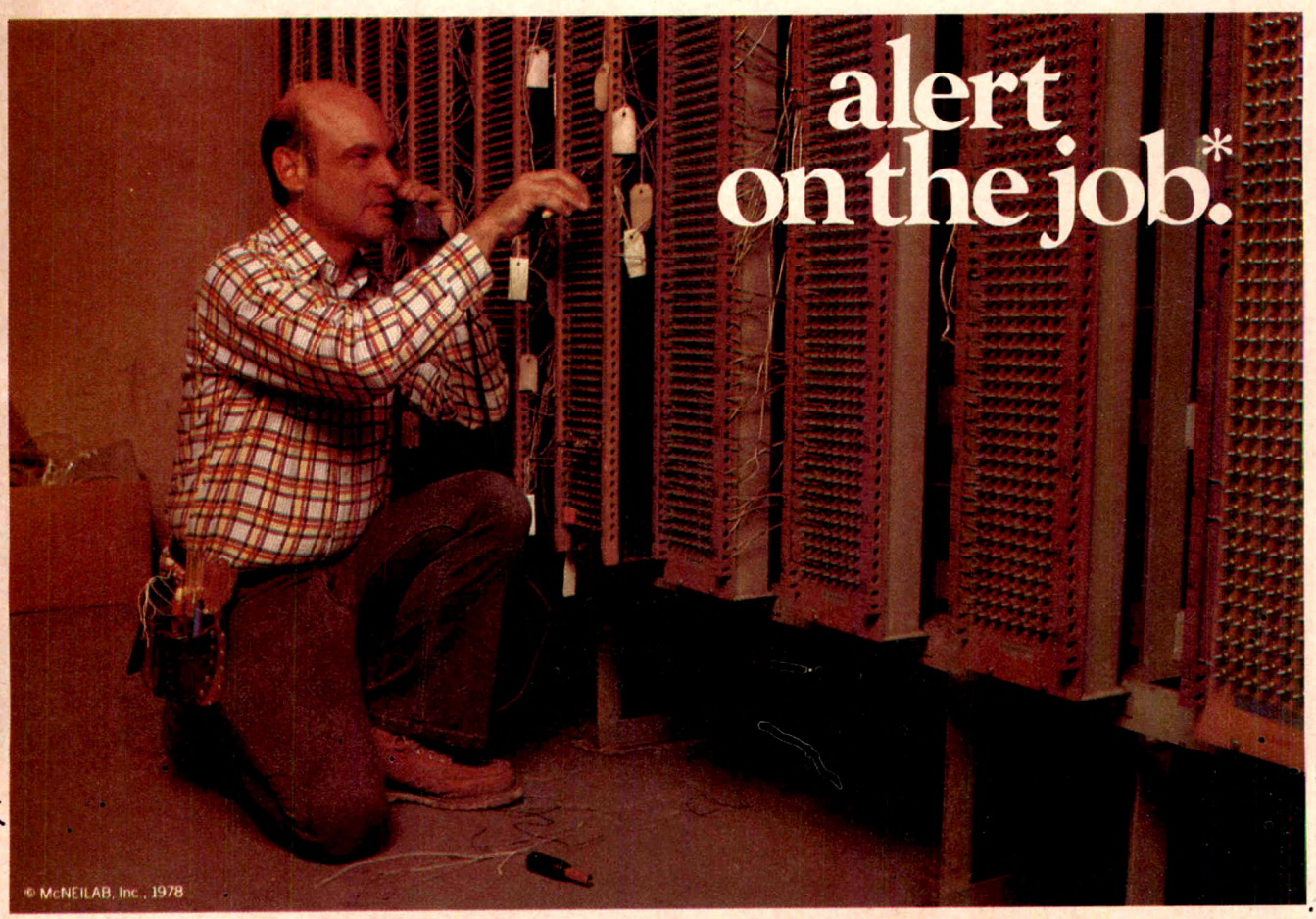
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*Not an actual case history, this situation illustrates the action of HALDOL haloperidol as reported in various clinical studies (available on request).

Minimal risk of hypotension, oversedation, or troublesome anticholinergic effects[3-5,7-9]
Transient hypotension occurs rarely; severe orthostatic hypotension has not been reported. Although some instances of drowsiness have been reported, marked sedation is rare.

Common side effects easily controlled[1,3-7]
Although extrapyramidal symptoms (EPS) have been reported frequently, they are usually dose-related and readily controlled with dose adjustment or antiparkinson drugs. EPS often diminish spontaneously with continued use of HALDOL haloperidol.

References: 1. Man, P.L.: Dis. Nerv. Syst. *34*:113 (Feb.) 1973. 2. Sugerman, A.A., et al.: Am. J. Psychiatry *129*:1190 (June) 1964. 3. Ayd, F.J., Jr.: Med. Sci. *18*:55 (Oct.) 1967. 4. Howard, J.S.: Dis. Nerv. Syst. *35*:458 (Oct.) 1974. 5. Abuzzahab, F.S., Sr.: Psychosomatics *11*:188 (May-June) 1970. 6. Darling, H.F.: Dis. Nerv. Syst. *34*:364 (Oct.-Nov.) 1973. 7. Gerle, B.: Clin. Trials J. *3*:380 (Feb.) 1966. 8. Snyder, S.H., et al: Science *184*:1243 (June 21) 1974. 9. Stimmel, C.L.: Dis. Nerv. Syst. *34*:219 (Apr.) 1976.

Please turn page for summary of prescribing information.

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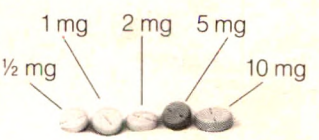
injection

A rapid-acting injection for psychiatric emergencies: 5 mg haloperidol (as the lactate) with 1.8 mg methylparaben and 0.2 mg propylparaben per ml, and lactic acid for pH adjustment to 3.4 ± 0.2.



tablets

5 tablet strengths for convenience in individualizing dosage:



HALDOL® (haloperidol)

tablets / concentrate / injection

A dosage form for every therapeutic need

Summary of Prescribing Information

Contraindications: Severe, toxic CNS depression or comatose states from any cause, hypersensitivity to the drug, Parkinson's disease.

Warnings: ***Usage in Pregnancy:*** Safe use in pregnancy or in women likely to become pregnant has not been established; use only if benefit clearly justifies potential hazards. Infants should not be nursed during drug treatment.

Combined Use With Lithium: Patients receiving lithium plus haloperidol should be monitored closely for early evidence of neurological toxicity.

General: Bronchopneumonia, sometimes fatal, has followed use of major tranquilizers, including haloperidol. Prompt remedial therapy should be instituted if dehydration, hemoconcentration or reduced pulmonary ventilation occurs, especially in the elderly. Decreased serum cholesterol and/or cutaneous and ocular changes have been reported with chemically-related drugs, although not with haloperidol. Mental and/or physical abilities required for hazardous tasks or driving may be impaired. Alcohol should be avoided due to possible additive effects and hypotension.

Precautions: Administer cautiously to patients: (1) with severe cardiovascular disorders, due to the possibility of transient hypotension and/or precipitation of anginal pain (if a vasopressor is required, epinephrine should not be used since HALDOL haloperidol may block its vasopressor activity and paradoxical further lowering of blood pressure may occur); (2) receiving anticonvulsant medication since HALDOL haloperidol may lower the convulsive threshold; (3) with known allergies or a history of allergic reactions to drugs; (4) receiving anticoagulants. Concomitant antiparkinson medication, if required, may have to be continued after HALDOL haloperidol is discontinued because of different excretion rates; if both are discontinued simultaneously, extrapyramidal symptoms may occur. Intraocular pressure may increase when anticholinergic drugs, including antiparkinson drugs, are administered concomitantly with HALDOL haloperidol. When HALDOL haloperidol is used for mania in cyclic disorders, there may be a rapid mood swing to depression. Severe neurotoxicity may occur in patients with thyrotoxicosis receiving antipsychotic medication, including HALDOL haloperidol. Neuroleptic drugs elevate prolactin levels; the elevation persists during chronic administration. Tissue culture experiments indicate that approximately one-third of human breast cancers are prolactin dependent *in vitro*, a factor of potential importance if the prescription of these drugs is contemplated in a patient with a previously detected breast cancer. Although disturbances such as galactorrhea, amenorrhea, gynecomastia, and impotence have been reported, the clinical significance of elevated serum prolactin levels is unknown for most patients. An increase in mammary neoplasms has been found in rodents after chronic administration of neuroleptic drugs. Neither clinical studies nor epidemiologic studies conducted to date, however, have shown an association between chronic administration of these drugs and mammary tumorigenesis: the available evidence is considered too limited to be conclusive at this time. The 1, 5, 10 mg tablets contain FD&C Yellow No. 5 (tartrazine) which may cause allergic-type reactions (including bronchial asthma) in certain susceptible individuals, especially in those who have aspirin hypersensitivity.

Adverse Reactions: ***CNS Effects:*** *Extrapyramidal Reactions:* Neuromuscular (extrapyramidal) reactions have been reported frequently, often during the first few days of treatment. Generally they involved Parkinson-like symptoms which were usually mild to moderately severe and usually reversible. Other types of neuromuscular reactions (motor restlessness, dystonia, akathisia, hyperreflexia, opisthotonos, oculogyric crises) have been reported far less frequently, but were often more severe. Severe extrapyramidal reactions have been reported at relatively low doses. Generally, extrapyramidal symptoms are dose-related since they occur at relatively high doses and disappear or become less severe when the dose is reduced. Antiparkinson drugs may be required. Persistent extrapyramidal reactions have been reported and the drug may have to be discontinued in such cases.

Withdrawal Emergent Neurological Signs: Abrupt discontinuation of short-term antipsychotic therapy is generally uneventful. However, some patients on maintenance treatment experience transient dyskinetic signs after abrupt withdrawal. In certain cases these are indistinguishable from "Persistent Tardive Dyskinesia" except for duration. It is unknown whether gradual withdrawal will reduce the occurrence of these signs, but until further evidence is available haloperidol should be gradually withdrawn.

Persistent Tardive Dyskinesia: Although rarely reported with HALDOL haloperidol, tardive dyskinesia may appear during or after long-term therapy. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and sometimes appear irreversible; there is no known effective treatment and all antipsychotic agents should be discontinued. The syndrome may be masked by reinstitution of drug, increasing dosage, or switching to a different antipsychotic agent.

Other CNS Effects: Insomnia, restlessness, anxiety, euphoria, agitation, drowsiness, depression, lethargy, headache, confusion, vertigo, grand mal seizures, and exacerbation of psychotic symptoms.

Cardiovascular Effects: Tachycardia and hypotension. ***Hematologic Effects:*** Reports of mild, usually transient leukopenia and leukocytosis, minimal decreases in red blood cell counts, anemia, or a tendency toward lymphomonocytosis; agranulocytosis rarely reported and only in association with other medication. ***Liver Effects:*** Impaired liver function and/or jaundice reported. ***Dermatologic Reactions:*** Maculopapular and acneiform reactions, isolated cases of photosensitivity, loss of hair. ***Endocrine Disorders:*** Lactation, breast engorgement, mastalgia, menstrual irregularities, gynecomastia, impotence, increased libido, hyperglycemia and hypoglycemia. ***Gastrointestinal Effects:*** Anorexia, constipation, diarrhea, hypersalivation, dyspepsia, nausea and vomiting. ***Autonomic Reactions:*** Dry mouth, blurred vision, urinary retention and diaphoresis. ***Respiratory Effects:*** Laryngospasm, bronchospasm and increased depth of respiration.

The injectable form is intended only for acutely agitated psychotic patients with moderately severe to very severe symptoms.

Caution: Federal law prohibits dispensing without prescription. • 08628

IMPORTANT: Full directions for use should be read before HALDOL haloperidol is administered or prescribed.

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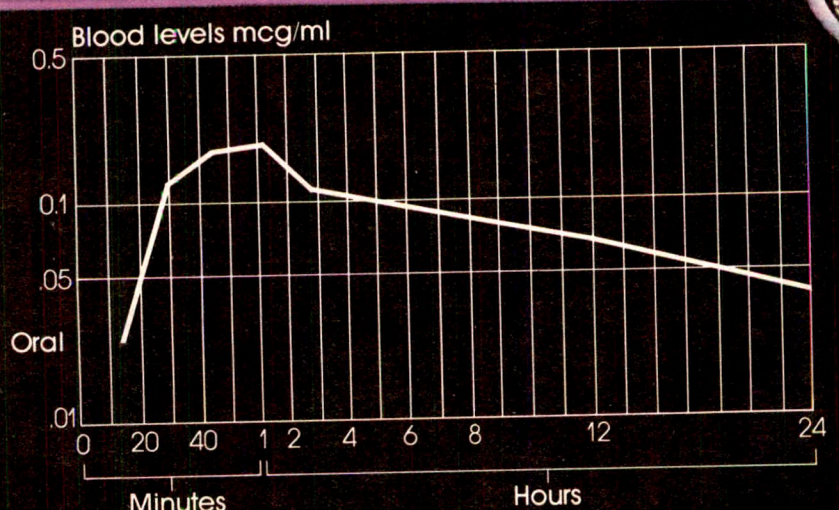
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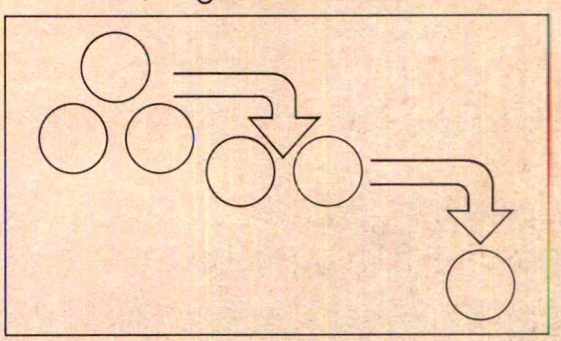
24-hour blood level curve of 10-mg oral dose of diazepam in a single healthy subject.

Smooth-out

Smooth: Avoids sudden symptom breakthrough Because of its fast and complete absorption, peak blood levels of Valium (diazepam/Roche) are achieved in 60 to 90 minutes after a single dose for a rapid initial calming effect. Typically, with an appropriate dosage regimen (e.g., 5 mg *t.i.d.*) Valium builds in a uniform and predictable way to a steady-state level within 5-7 days, without accumulating beyond. With Valium the excretion rate is similarly consistent and predictable. When pharmacologic support is no longer needed, the overlapping half-lives of diazepam and its metabolites insure a gradual elimination process. Because metabolites of short-acting benzodiazepines are inactive, blood levels of these drugs decrease more rapidly without benefit of a tapering effect.

Consistent therapeutic effect even when an occasional dose is omitted Once steady-state is achieved, sudden symptom breakthrough is highly unlikely, even if a dose is forgotten or skipped. That's because the half-life of Valium—27-37 hours—provides the advantage of self-tapering action, complementing your gradual reduction of the patient's dosage and smoothing his transition to independent coping.

To discontinue smoothly... Gradual discontinuation of medication—while rarely necessary after short-term therapy—*is* good medical practice in patients who have received high doses for extended periods. When dosage is tapered, untoward side effects can be avoided. Throughout therapy, in fact, side effects are infrequent with Valium and are seldom more serious than drowsiness, fatigue and ataxia.



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2-mg, 5-mg, 10-mg scored tablets

Valium® Smooth transition to independent coping

diazepam/Roche ℞

Valium®
diazepam/Roche

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Management of anxiety disorders, or short-term relief of symptoms of anxiety; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).
The effectiveness of Valium in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Anxiety disorders, symptoms of anxiety, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

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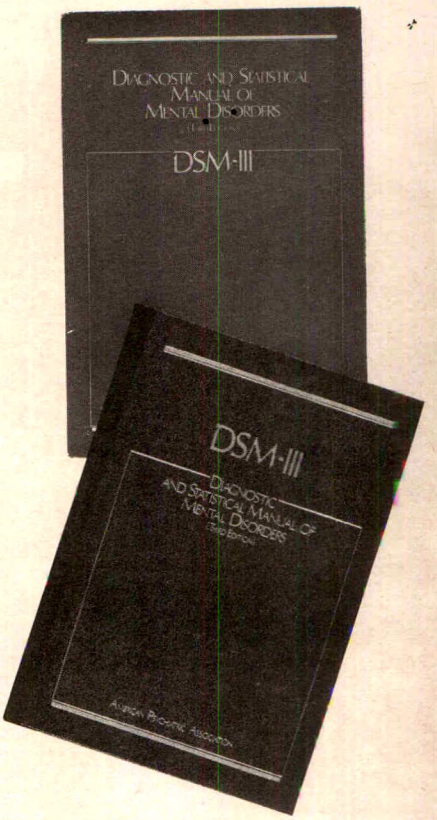
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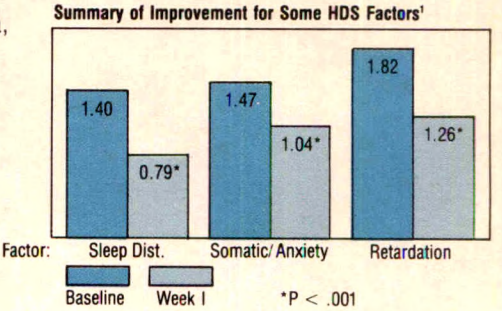


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In patients who may use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage.

Precautions–Drowsiness may occur and patients should be cautioned against driving a motor vehicle or operating hazardous machinery. Since suicide is an inherent risk in depressed patients they should be closely supervised while receiving treatment. Although Adapin has shown effective tranquilizing activity, the possibility of activating or unmasking latent psychotic symptoms should be kept in mind. This product contains FD&C Yellow No. 5 (tartrazine) which may cause allergic-type reactions (including bronchial asthma) in certain susceptible individuals. Although the overall incidence of FD&C Yellow No. 5 (tartrazine) sensitivity in the general population is low, it is frequently seen in patients who also have aspirin hypersensitivity.

Adverse Reactions–Dry mouth, blurred vision and constipation have been reported. Drowsiness has also been observed.

Adverse effects occurring infrequently include extrapyramidal symptoms, gastrointestinal reactions, secretory effects such as sweating, tachycardia and hypotension. Weakness, dizziness, fatigue, weight gain, edema, paresthesias, flushing, chills, tinnitus, photophobia, decreased libido, rash and pruritus may also occur.

Dosage and Administration–In mild to moderate anxiety and/or depression: 25 mg t.i.d. Increase or decrease the dosage according to individual response. Daily dosage, up to 150 mg may be taken at bedtime without loss of effectiveness. Usual optimum daily dosage is 75 mg to 150 mg per day not to exceed 300 mg per day.

Antianxiety effect usually precedes the antidepressant effect by two or three weeks.

How Supplied–Each capsule contains doxepin, as the hydrochloride. 10 mg, 25 mg, 50 mg, 75 mg and 100 mg capsules in bottles of 100 and 1000.

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References:

1. Barranco SF, Thrash ML, Hackett E, et al: Early onset of response to doxepin treatment. *J Clin Psychiatry* 40:265-269, 1979 (Sinequan®).
2. Karacan I, Blackburn AB, Thornby JI: The Effect of Doxepin HCl (Sinequan®) on Sleep Patterns and Clinical Symptomatology of Neurotic Depressed Patients with Sleep Disturbance, in Mendels J (ed): *Sinequan®: A monograph of recent clinical studies*, Princeton, NJ, Excerpta Medica, 1977, pp 4-22.
3. Pitts NE: The clinical evaluation of doxepin–A new psychotherapeutic agent. *Psychosomatics* 10:164-171, 1969.

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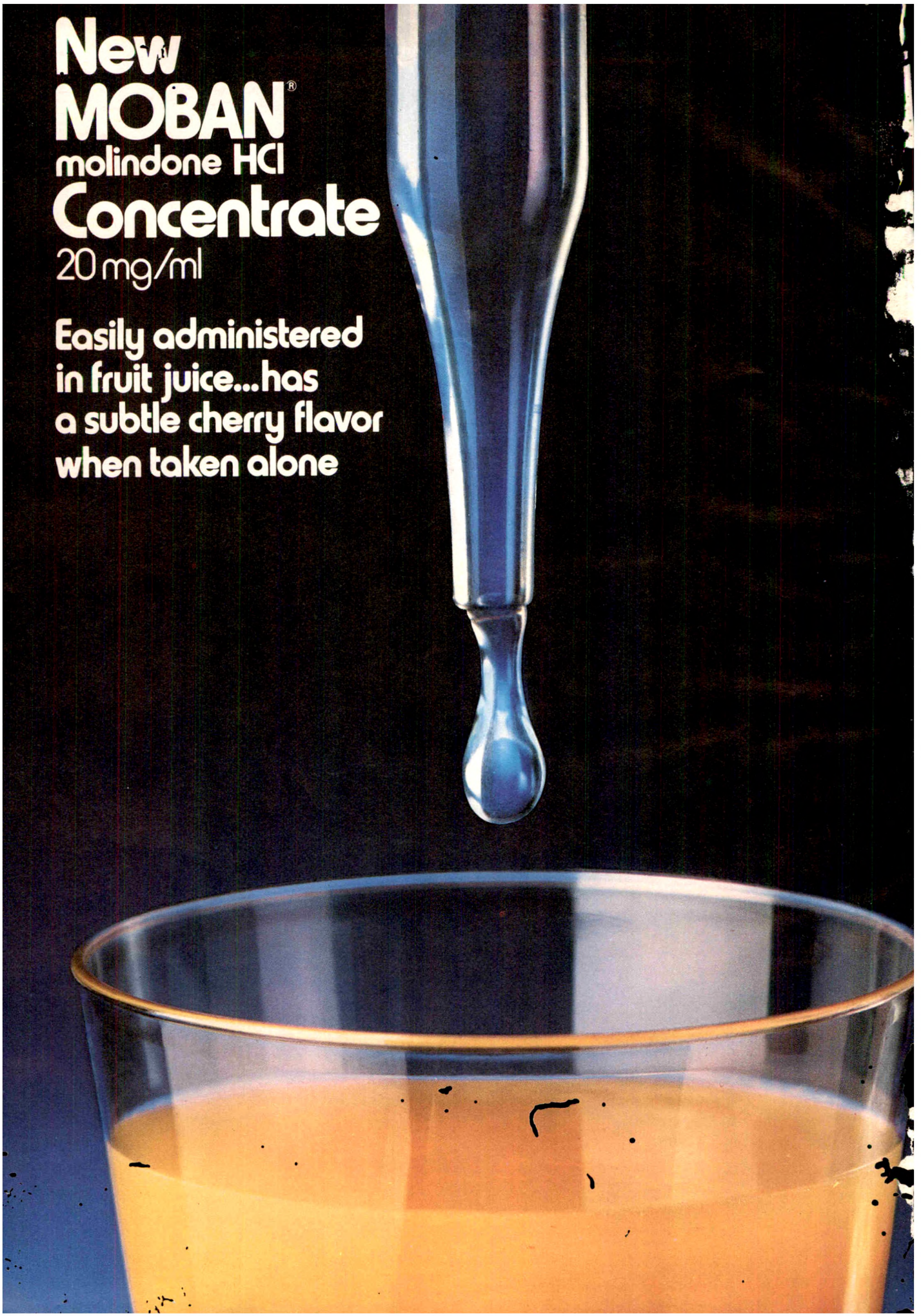
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New
MOBAN®
molindone HCl
Concentrate
20 mg/ml
Easily administered
in fruit juice...has
a subtle cherry flavor
when taken alone

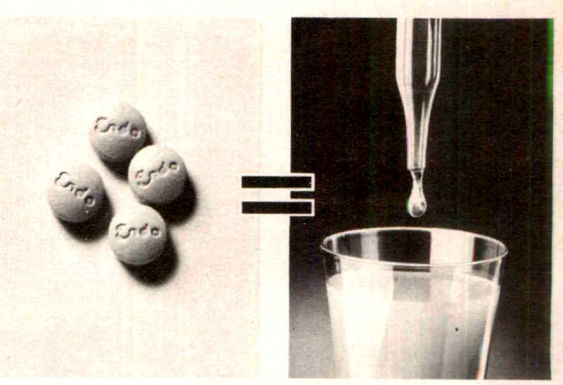
An effective antipsychotic agent with a favorable benefit/risk profile

NO incidence	excessive weight gain hepatotoxic changes ophthalmological changes persistent tardive dyskinesia skin pigmentation thyroid function changes tolerance development
LOW incidence	adverse drug interactions hematological changes hypotension nonpersistent symptoms of tardive dyskinesia sedation
MODERATE incidence	extrapyramidal reactions

This table compiled from clinical and field reports, according to the best available information.

For the schizophrenic patient

New MOBAN®
molindone HCl
Concentrate
20 mg/ml



four 25 mg tablets = 5 milliliters (100 mg)

Concentrated for maximum convenience of higher dosages...easy titration... enhanced compliance

Initial dosage with MOBAN®

- Usual starting dose is 50-75 mg/day
- May be increased to 100 mg/day in three to four days
- May be adjusted up or down according to response
- Some patients with severe symptomatology may require an increase to 225 mg/day
- Elderly and debilitated patients should be started on lower dosage

Please see next page for prescribing information.

Endo Laboratories DUPONT

An effective antipsychotic agent

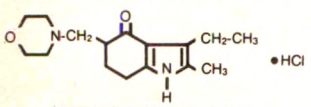
MOBAN®
molindone HCl.

New dosage form... Concentrate 20 mg/ml

continuing...Tablets 5 mg, 10 mg, 25 mg

DESCRIPTION MOBAN (molindone hydrochloride) is a dihydroindolone compound which is not structurally related to the phenothiazines, the butyrophenones or the thioxanthenes.

MOBAN is 3-ethyl-6, 7-dihydro-2-methyl-5-(morpholinomethyl) indol-4 (5H)-one hydrochloride. It is a white crystalline powder, freely soluble in water and alcohol and has a molecular weight of 312.67.



MOLINDONE HYDROCHLORIDE

ACTIONS MOBAN (molindone hydrochloride) has a pharmacological profile in laboratory animals which predominantly resembles that of major tranquilizers causing reduction of spontaneous locomotion and aggressiveness, suppression of a conditioned response and antagonism of the bizarre stereotyped behavior and hyperactivity induced by amphetamines. In addition, MOBAN antagonizes the depression caused by the tranquilizing agent tetrabenazine.

In human clinical studies tranquilization is achieved in the absence of muscle relaxing or incoordinating effects. Based on EEG studies, MOBAN exerts its effect on the ascending reticular activating system.

Human metabolic studies show MOBAN (molindone hydrochloride) to be rapidly absorbed and metabolized when given orally. Unmetabolized drug reached a peak blood level at 1.5 hours. Pharmacological effect from a single oral dose persists for 24-36 hours. There are 36 recognized metabolites with less than 2-3% unmetabolized MOBAN being excreted in urine and feces.

INDICATIONS MOBAN (molindone hydrochloride) is indicated in the management of the manifestations of schizophrenia.

CONTRAINDICATIONS MOBAN (molindone hydrochloride) is contraindicated in severe central nervous system depression (alcohol, barbiturates, narcotics, etc.) or comatose states, and in patients with known hypersensitivity to the drug.

WARNINGS Usage in Pregnancy: Studies in pregnant patients have not been carried out. Reproduction studies have been performed in the following animals:

Pregnant Rats oral dose— 20 mg/kg/day—10 days
no adverse effect
40 mg/kg/day—10 days
no adverse effect

Pregnant Mice oral dose— 20 mg/kg/day—10 days
slight increase resorptions
40 mg/kg/day—10 days
slight increase resorptions

Pregnant Rabbits oral dose— 5 mg/kg/day—12 days
no adverse effect
10 mg/kg/day—12 days
no adverse effect
20 mg/kg/day—12 days
no adverse effect

Animal reproductive studies have not demonstrated a teratogenic potential. The anticipated benefits must be weighed against the unknown risks to the fetus if used in pregnant patients.

Nursing Mothers: Data are not available on the content of MOBAN (molindone hydrochloride) in the milk of nursing mothers.

Usage in Children: Use of MOBAN (molindone hydrochloride) in children below the age of twelve years is not recommended because safe and effective conditions for its usage have not been established.

MOBAN has not been shown effective in the management of behavioral complications in patients with mental retardation.

PRECAUTIONS Some patients receiving MOBAN (molindone hydrochloride) may note drowsiness initially and they should be advised against activities requiring mental alertness until their response to the drug has been established.

Increased activity has been noted in patients receiving MOBAN. Caution should be exercised where increased activity may be harmful.

MOBAN does not lower the seizure threshold in experimental animals to the degree noted with more sedating antipsychotic drugs. However, in humans convulsive seizures have been reported in a few instances.

EDO 854M580

The physician should be aware that this tablet preparation contains calcium sulfate as an excipient and that calcium ions may interfere with the absorption of preparations containing phenytoin sodium and tetracyclines.

MOBAN (molindone hydrochloride) has an antiemetic effect in animals. A similar effect may occur in humans and may obscure signs of intestinal obstruction or brain tumor.

ADVERSE REACTIONS—CNS EFFECTS The most frequently occurring effect is initial drowsiness that generally subsides with continued usage of the drug or lowering of the dose.

Noted less frequently were depression, hyperactivity and euphoria.

Neurological—Extrapyramidal Reactions Extrapyramidal reactions noted below may occur in susceptible individuals and are usually reversible with appropriate management.

Akathisia Motor restlessness may occur early.

Parkinson Syndrome Akinesia, characterized by rigidity, immobility and reduction of voluntary movements and tremor, have been observed. Occurrence is less frequent than akathisia.

Dystonic Syndrome Prolonged abnormal contractions of muscle groups occur infrequently. These symptoms may be managed by the addition of a synthetic antiparkinson agent (other than L-dopa), small doses of sedative drugs, and/or reduction in dosage.

Autonomic Nervous System Occasionally blurring of vision, tachycardia, nausea, dry mouth and salivation have been reported. Urinary retention and constipation may occur particularly if anticholinergic drugs are used to treat extrapyramidal symptoms.

Hematological There have been rare reports of leucopenia and leucocytosis. If such reactions occur, treatment with MOBAN may continue if clinical symptoms are absent. Alterations of blood glucose, liver function tests, B.U.N., and red blood cells have not been considered clinically significant.

Metabolic and Endocrine Effects Alteration of thyroid function has not been significant. Amenorrhea has been reported infrequently. Resumption of menses in previously amenorrheic women has been reported. Initially heavy menses may occur. Galactorrhea and gynecomastia have been reported infrequently. Increase in libido has been noted in some patients. Impotence has not been reported. Although both weight gain and weight loss have been in the direction of normal or ideal weight, excessive weight gain has not occurred with MOBAN.

Cardiovascular Rare, transient, non-specific T wave changes have been reported on E.K.G. Association with a clinical syndrome has not been established. Rarely has significant hypotension been reported.

Ophthalmological Lens opacities and pigmentary retinopathy have not been reported where patients have received MOBAN (molindone hydrochloride). In some patients, phenothiazine induced lenticular opacities have resolved following discontinuation of the phenothiazine while continuing therapy with MOBAN.

Skin Early, non-specific skin rash, probably of allergic origin, has occasionally been reported. Skin pigmentation has not been seen with MOBAN usage alone.

MOBAN (molindone hydrochloride) has certain pharmacological similarities to other antipsychotic agents. Because adverse reactions are often extensions of the pharmacological activity of a drug, all of the known pharmacological effects associated with other antipsychotic drugs should be kept in mind when MOBAN is used. Upon abrupt withdrawal after prolonged high dosage an abstinence syndrome has not been noted.

Tardive Dyskinesia Although rarely reported with MOBAN (molindone hydrochloride) symptoms were reversible upon discontinuation of therapy.

Tardive dyskinesia associated with other agents has appeared in some patients on long-term therapy and has also appeared after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). There may be involuntary movements of extremities.

There is no known effective treatment of tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time the syndrome may not develop.

DOSAGE AND ADMINISTRATION Initial and maintenance doses of MOBAN (molindone hydrochloride) should be individualized.

Initial Dosage Schedule The usual starting dosage is 50-75 mg/day.
- —Increase to 100 mg/day in 3 or 4 days.
- —Based on severity of symptomatology, dosage may be titrated up or down depending on individual patient response.
- —An increase to 225 mg/day may be required in patients with severe symptomatology.

Elderly and debilitated patients should be started on lower dosage.

Maintenance Dosage Schedule
1. Mild—5 mg-15 mg three or four times a day.
2. Moderate—10 mg-25 mg three or four times a day.
3. Severe—225 mg/day may be required.

DRUG INTERACTIONS Potentiation of drugs administered concurrently with MOBAN (molindone hydrochloride) has not been reported. Additionally, animal studies have not shown increased toxicity when MOBAN is given concurrently with representative members of three classes of drugs (i.e., barbiturates, chloral hydrate and antiparkinson drugs).

MANAGEMENT OF OVERDOSAGE Symptomatic, supportive therapy should be the rule.

Gastric lavage is indicated for the reduction of absorption of MOBAN (molindone hydrochloride) which is freely soluble in water.

Since the adsorption of MOBAN (molindone hydrochloride) by activated charcoal has not been determined, the use of this antidote must be considered of theoretical value.

Emesis in a comatose patient is contraindicated. Additionally, while the emetic effect of apomorphine is blocked by MOBAN in animals, this blocking effect has not been determined in humans.

A significant increase in the rate of removal of unmetabolized MOBAN (molindone hydrochloride) from the body by forced diuresis, peritoneal or renal dialysis would not be expected. (Only 2% of a single ingested dose of MOBAN is excreted unmetabolized in the urine.)

However, poor response of the patient may justify use of these procedures. While the use of laxatives or enemas might be based on general principles, the amount of unmetabolized MOBAN in feces is less than 1%. Extrapyramidal symptoms have responded to the use of diphenhydramine (Benadryl*) and the synthetic anticholinergic antiparkinson agents, (i.e., Artane*, Cogentin*, Akineton*).

HOW SUPPLIED As tablets in bottles with potencies and colors as follows:

100's and 1000's:
5 mg orange
10 mg lavender
25 mg light green

As a concentrate containing 20 mg molindone hydrochloride per ml in 4 oz. (120ml) bottles.

*Benadryl—Trademark, Parke Davis and Co.
*Artane—Trademark, Lederle Laboratories
*Cogentin—Trademark, Merck Sharp & Dohme
*Akineton—Trademark, Knoll Pharmaceutical Co.

Rev. Nov. 1979 6102-5

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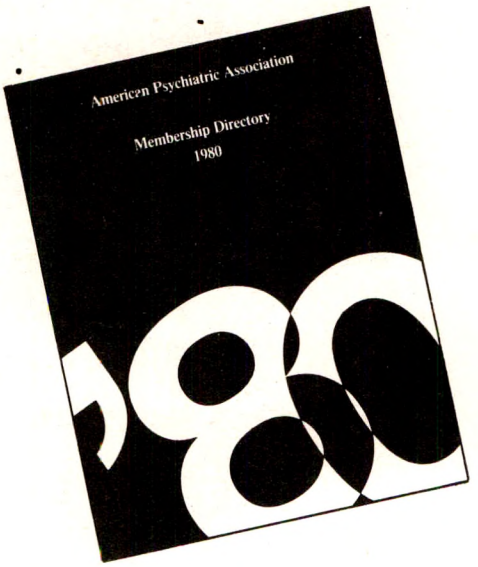
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AJP

Brief Summary

INDICATIONS — For management of anxiety disorders or short-term relief of symptoms of anxiety. Anxiety or tension associated with stress of everyday life usually does not require treatment with an anxiolytic.

Effectiveness in long-term use (over 4 months) not assessed by systematic clinical studies. Physician should periodically reassess usefulness for each patient.

For symptomatic relief of acute alcohol withdrawal.

CONTRAINDICATIONS — Known hypersensitivity to the drug. Acute narrow angle glaucoma.

WARNINGS — Not for use in depressive neuroses or psychotic reactions. Caution patients against hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles. Advise against simultaneous use of other CNS depressants, and caution patients that effects of alcohol may be increased. Not recommended for patients under 18. Nervousness, insomnia, irritability, diarrhea, muscle aches, and memory impairment have followed abrupt withdrawal from long-term high dosage. Withdrawal symptoms were reported after abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months. Use caution in patients having psychological potential for drug dependence (dependence has been observed in dogs and rabbits).

Pregnancy and Lactation: Minor tranquilizers should almost always be avoided first trimester. Consider possibility of pregnancy before initiating therapy. Patient should consult physician about discontinuation if she becomes pregnant or plans pregnancy. Do not give to nursing mothers.

PRECAUTIONS — Observe usual precautions in depression accompanying anxiety, or in patients with suicidal tendency, or those with impaired renal or hepatic function. Do periodic blood counts and liver function tests during prolonged therapy. Use small doses and gradual increments in the elderly or debilitated.

ADVERSE REACTIONS — Drowsiness, dizziness, various g.i. complaints, nervousness, blurred vision, dry mouth, headache, mental confusion, insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression, slurred speech, abnormal liver and kidney function, decreased hematocrit, decreased systolic blood pressure.

DOSAGE — ANXIETY — Usual daily dose 30 mg or less (start the elderly or debilitated at 7.5-15 mg). Adjust gradually within 15-60 mg daily range. Capsules and scored tablets: divided doses; or once daily h.s. (start patient at 15 mg). Single dose tablets, 22.5 mg (for patients stabilized on 7.5 mg t.i.d.) or 11.25 mg: once daily at any hour.

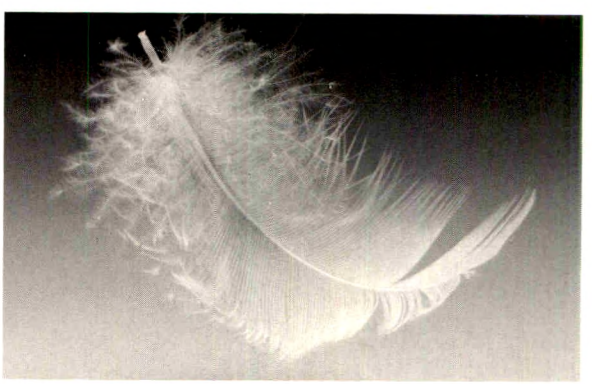
INTERACTIONS — Potentiation may occur with ethyl alcohol, hypnotics, barbiturates, narcotics, phenothiazines, MAO inhibitors, other antidepressants. In bioavailability studies with normal subjects, concurrent administration of antacids at therapeutic levels did not significantly influence bioavailability of TRANXENE.

OVERDOSAGE — Take general measures as for any CNS depressant.

SUPPLIED — Tranxene (clorazepate dipotassium) 3.75, 7.5, and 15 mg capsules and scored tablets. Tranxene-SD Half Strength 11.25 and Tranxene-SD 22.5 mg single dose tablets.

REFERENCES — 1. The elimination kinetics of an agent can be closely defined, but cannot at present be directly related to therapeutic or adverse effects. 2. Sedation reported in normal volunteers 30 minutes after dosing. TRANXENE Drug Monograph 97-0185, 1979, p. 9. 3. op cit., p 15.

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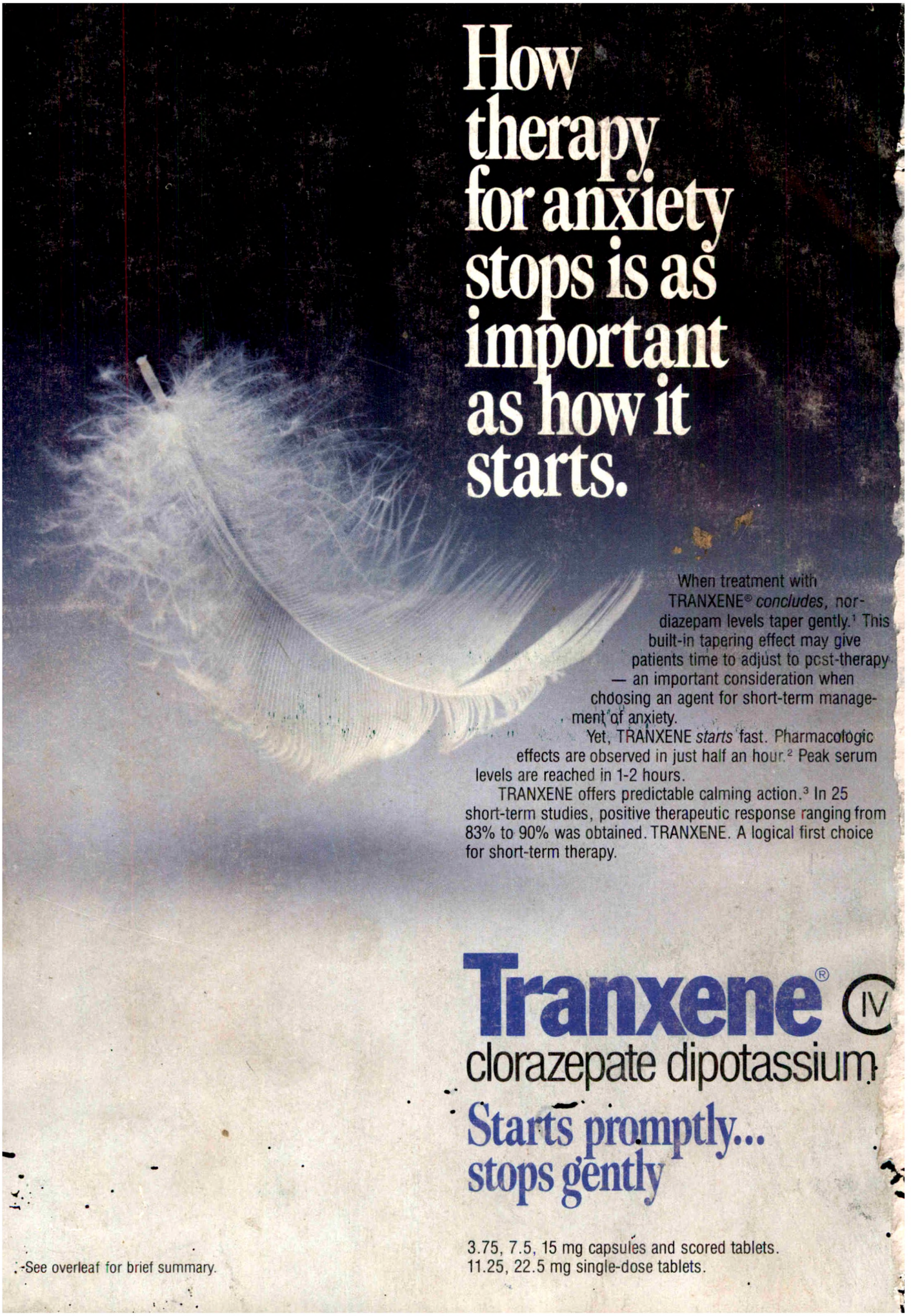
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clorazepate dipotassium

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11.25, 22.5 mg single-dose tablets.

-See overleaf for brief summary.